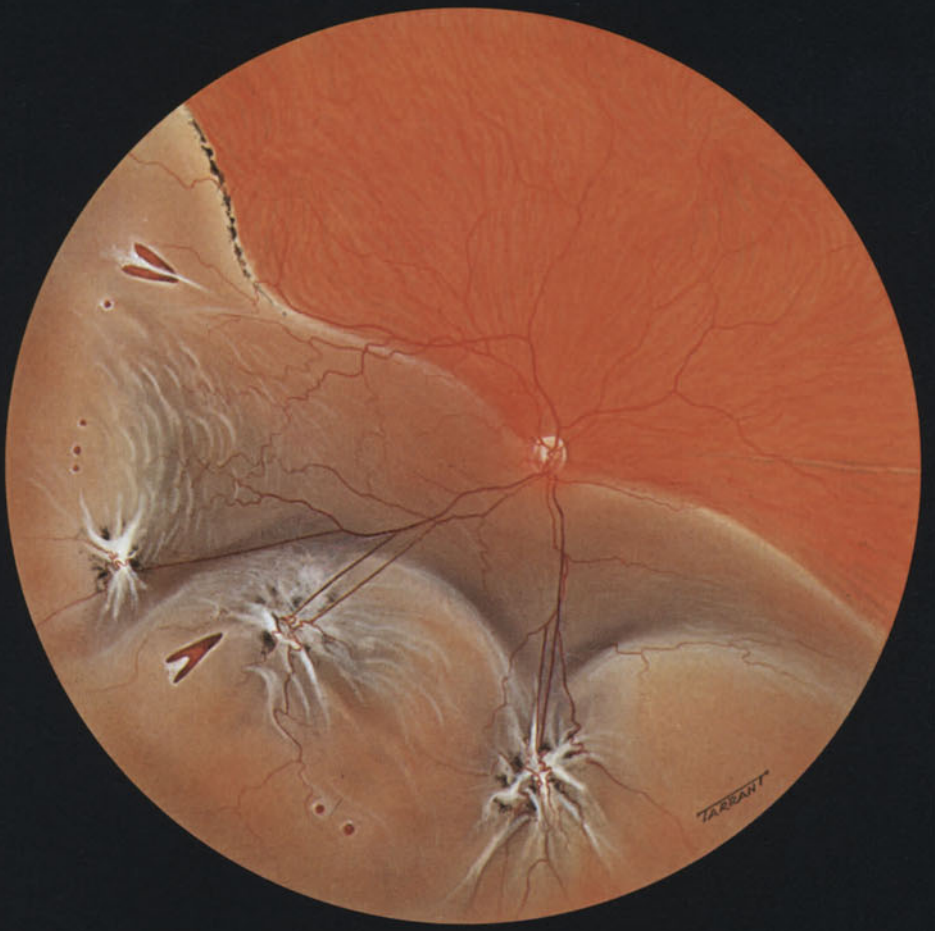
TARRANT

A. H. Chignell

Retinal Detachment Surgery

With 50 Figures

Springer-Verlag
Berlin Heidelberg New York 1980

Anthony H. Chignell F.R.C.S.
St. Thomas' Hospital
GB-London SE1 7EH

ISBN-13: 978-3-642-96532-6 e-ISBN-13: 978-3-642-96530-2
DOI: 10.1007/978-3-642-96530-2

Library of Congress Cataloging in Publication Data. Chignell, A.H. Retinal detachment surgery. Bibliography: p. Includes index. 1. Retinal detachment. 2. Retina-Surgery. I. Title. [DNLM: 1. Retinal detachment – Surgery. WW270 C534r] RE603.C46 617.7'3 79–15606

2124/3321–543210

Contents

Chapter 3. Preoperative Management and Planning of Operation

Chapter 4. Surgical Details

Chapter 5. Postoperative Management and Complications

Preface

This short book is an account of an approach to retinal detachment surgery. I hope it will be of use to all those dealing with the detached retina, although it has been written mainly for the training ophthalmologist, who, becoming familiar with the methods of examination, is faced with a bewildering array of methods of treating the detached retina. The object of surgery is to produce the desired result in the least traumatic way possible and this involves correct interpretation of how the detached retina is likely to respond to the operation that is planned. I have therefore concentrated on the aspects of clinical examination that have a direct bearing on the planning of the type of operation to be performed. I have not attempted to deal with all the different surgical techniques currently practised, but have described only those that I believe represent the simplest, safest, and most effective methods. I have drawn widely on standard techniques; thus, for examination, Schepens' indirect ophthalmoscopy with scleral depression, Lincoff's use of cryotherapy and his modification of Custodis' non-drainage techniques at operation and Rosengren's use of intraocular air. Because this book is not intended for the retinal surgical expert I have not dealt in great depth with rare cases, e.g. macular holes, or other conditions that are exceptionally difficult to treat (massive periretinal proliferation). The surgical management of these types of case is likely to be an area of continual change, and the role of the various advancing techniques (e.g. vitrectomy) one for further assessment.

In the preparation of the book I have been much helped by a number of people. The illustrations have been expertly and painstakingly prepared by Mr Terry Tarrant of the Audio Visual Department of the Institute of Ophthalmology. Additional photographic work has also been done by Mr Rolph Sennhenn and Miss Jan Shugg, of the Eye Department of St Thomas' Hospital. Mrs Margaret Grice has done the greater part of the secretarial work, a feat performed with much good humour and tolerance, and Miss Sue Patterson has given additional help in the later stages. My colleague, Mr Martin Crick, FRCS, kindly read the manuscript and made many useful suggestions. I am grateful

to Mr Jack Kanski, FRCS, for permission to use slightly modified versions of Figures 2.6 and 2.7, and also to Mr Tim ffytche, FRCS, for Figure 5.5. The colour frontispiece was made possible by kind donations from Keeler Instruments Ltd and Davis & Geck.

I am particularly indebted to the past and present junior ophthalmic staff of St Thomas' Hospital, London, with whom I work, as it is their constant enquiry and challenge of traditional practice that prevents a static approach to the clinical problems we share, problems that, if solved, offer as their reward the restoration of vision.

In conclusion, I am grateful to my Publisher for being so patient with me in the preparation of the manuscript.

London, October 1979 A.H. Chignell

Chapter 1

Production, Characteristics and Methods of Examining Retinal Detachment

Pathogenesis

Retinal detachment is the result of fluid accumulating between the photo-receptor and pigment epithelial layers of the retina, and in rhegmatogenous retinal detachment the fluid accumulates after a hole has formed in the retina.

Formation of Retinal Holes

A retinal hole results from the interplay of two factors: vitreous traction and underlying retinal weakness. Each factor plays a varying role in the production of different types of hole; for example, when horseshoe tears are produced there is a strong traction element in evidence, whereas there is little evidence that vitreous traction plays much part in the production of small round holes without opercula, when, it seems, the retina itself is primarily at fault. Often there may be a combination of factors, a tractional state being associated with an underlying retinal weakness; for example, when horseshoe-shaped tears form in areas of lattice degeneration.

The vitreous gel, which in an entirely healthy state is firmly apposed to the innermost retinal layer may, as a result of degenerative change (senility or in pathological states; for example aphakia or myopia) separate from the retina. This separation almost invariably starts posteriorly and spreads forwards. If separation continues, the vitreous will be adherent only to particularly firm points of attachment, for example at the vitreous base and along retinal blood vessels. It is clear that when the vitreous pulls on the retina, traction will be present at these points of attachment.

Genesis of Detachment

Post-mortem (Okun, 1961a) and clinical studies (Neumann and Hyams, 1972) have shown that the majority of retinal holes do not progress to retinal detachment. Nevertheless, when subretinal fluid does accumulate, posterior movement of vitreal or retrovitreal fluid will occur via the retinal hole, breaking down normal intraretinal apposition (Foulds, 1975a). However, analysis of subretinal fluid (Chignell et al., 1971; Kaufmann and Podos, 1973) has indicated that

there is also an anterior movement of fluid from choroidal to subretinal space via the pigment epithelium and Bruch's membrane. Such an anterior movement of fluid across pigment epithelium is consistent with the observation of subretinal fluid forming in non-rhegmatogenous retinal detachment; for example in central serous retinopathy and exudative detachment, where movement of fluid from the vitreous is impossible.

The following factors in the vicinity of a retinal hole are important influences in the initial formation of subretinal fluid which, once formed, will spread in a predictable way (Lincoff and Gieser, 1971).

Influence of the Vitreous

Vitreous Traction

Vitreous traction on the anterior edge of a horseshoe-shaped tear will tend to encourage the layers of the retina to separate. Its influence depends on the movement of the vitreous gel and on the position of the retinal tear. If there is a posterior vitreous detachment, when the eye moves the movement of the posterior gel, firmly fixed anteriorly at the vitreous base, will exert considerable tractional forces at its main point of anchorage, the posterior border of the vitreous base, which is a common site for producing horseshoe-shaped tears. However, tears situated within the basal vitreous are not subjected to such dynamic tractional forces and are therefore less likely to proceed to detachment.

Consistency of the Vitreous

It has been suggested that in aphakia, loss of hyaluronic acid from the vitreous gel contributes to the lowering of resistance of vitreous flow through a retinal hole (Osterlin, 1977), a feature that may be accentuated by haemorrhage.

Morphological Features of the Retinal Hole

Horseshoe-shaped tears are more likely to proceed to retinal detachment than are round holes (Lincoff, 1961; Davis, 1974), which may be a reflection of the extent of vitreous traction on the holes. It is believed that large holes are more vulnerable than small ones.

Integrity of the Pigment Epithelium Neuro-epithelial Adhesion

This natural mechanism, although demonstrated in the rabbit (Zauberman and Guillebon, 1972), has not been fully explained. The potential binding effect on the two layers of the retina by the intercellular mucopolysaccharide layer has been stressed by Foulds (1975b). It has been suggested that this normal

adhesion may be reduced, for example in aphakia, thus accounting for why these types of detachments will progress more rapidly than others (Ashrafadeh et al., 1973). A similar lowering of this adhesive force may also be a feature of local retinal degenerations; for example, lattice and snail-track degeneration, both of which may be associated with hole formation and retinal detachment.

Combination of Factors

Many eyes that develop retinal detachment have a combination of factors tending to make them more liable to this event. The predisposing factor may be present to a greater or lesser extent in the different types of detachment encountered. For example, in one aphakic patient round holes may cause detachment, whereas in another, detachment may result from a horseshoe-shaped tear (the extreme form of which is the giant tear) arising in an apparently normal area of retina. In the first case retinal weakness leading to hole formation and reduction of intraretinal adhesion is the main factor, whereas in the second case vitreous traction is the predominant feature.

Formation and Spread of Subretinal Fluid

Two aspects of the spread of subretinal fluid are of particular importance: the rate of spread and the anatomical direction of spread.

Rate of Spread

The rate of spread is related to the following:

Position of the hole in the retina. Due to the influence of gravity, subretinal fluid will accumulate quickly if the hole is in the upper half of the retina, whereas with inferior tears the accumulation is slower.

Character of the hole. Large holes lead to fluid accumulating more rapidly than in small ones.

Adhesion between pigment epithelium and neuro-epithelium. If this is deficient, as has been suggested in aphakia, subretinal fluid will accumulate more readily.

State and position of the vitreous. If the vitreous gel is of apparently normal consistency, for example in a young subject with a traumatic retinal detachment, then the rate of progression of detachment will tend to be slower.

In addition to gravity it is possible that the rate at which the fluid accumulates may be influenced by the relationship of the surrounding vitreous. Thus, in

inferior detachments the vitreous may be in close association with the detached retina, whereas in superior detachments there is usually a partial or total posterior vitreous detachment, the detaching retina thus being almost completely unsupported by vitreous gel. This lack of support may contribute to an easier descent of the detaching retina.

Anatomical Direction of Spread

Subretinal fluid accumulating around the retinal hole will first spread from the hole to the ora serrata, and its subsequent spread will be influenced by the position of the tear in the retina and by any obstructions in the natural pathway of the spread; for example, old operation sites or other choroido-retinal scars.

Knowledge of the way in which subretinal fluid spreads is of great value clinically because it enables the site of such a hole to be accurately forecast even when, on examination of the retina, the offending hole is difficult to find (Lincoff and Gieser, 1971). Subsequent treatment of the retinal detachment is thus made much easier. Similarly, when a retinal hole is found, and the distribution of subretinal fluid from it is not as expected, then further careful search is indicated to discover the whereabouts of some other hole.

Superior Temporal and Superior Nasal Detachments

Fluid accumulates around the retinal hole, and as the detachment becomes more extensive the fluid front descends, on the same side as the retinal hole, towards the disc. It will then swing below the disc and, with progressive accumulation, which often results in inferior bullae, will rise up again on the other side of the retina. It will, however, always assume an upper level lower than

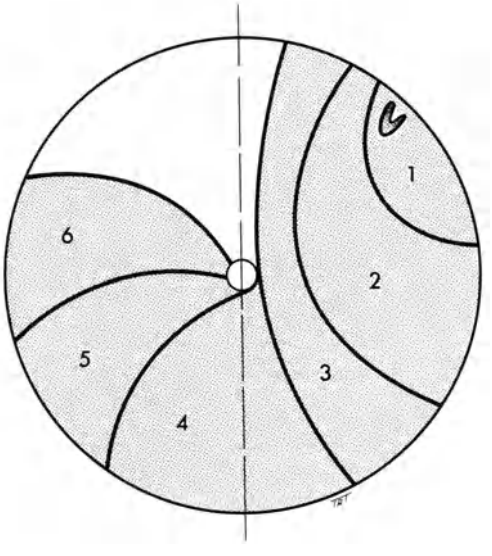

Fig. 1.1. Progressive accumulation of subretinal fluid from a hole in the upper retina but not close to the midline

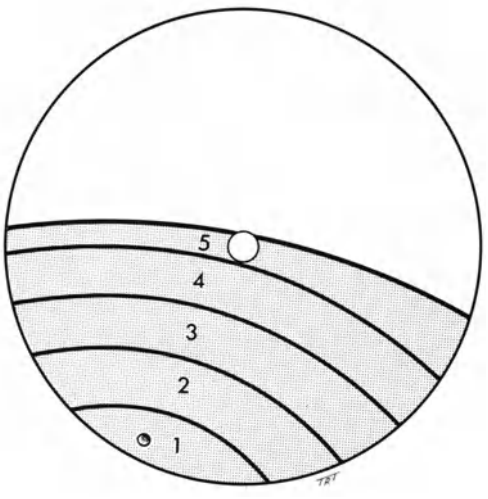

Fig. 1.2. Accumulation of subretinal fluid from an inferior hole

that on the side on which it began (Fig. 1.1). Fluid will, to a variable extent, rise up from the retinal hole to involve retina above it but it will not cross the mid-line, assuming the disc is centrally placed.

Superior Mid-line Holes

Subretinal fluid from holes situated very near the mid-line above will spread down rapidly (i.e. directly above the disc) and symmetrically on each side of the disc. If the hole is slightly to one side of the disc, the fluid front will be more prominant on the same side. If the hole is situated in a particularly posterior position above, then the tendency for fluid to cross the mid-line and descend on either side of the disc is rather greater, and the localising value of fluid edges will be of less value in these cases, although it will usually be very easy to see the position of such a hole.

Inferior Detachment

Subretinal fluid accumulates around the hole and gradual extension will produce a detachment that rises slowly from below. These detachments are seldom bullous. If the hole is situated exactly below the optic disc, the fluid will rise equally on each side of the disc. If the hole favours either the temporal or nasal half of the retina, then fluid will rise higher on the side on which the hole is located (Fig. 1.2). Inferior holes rarely result in total detachment.

Total Detachments

The great majority of fresh total detachments arise from holes situated close to the mid-line, in the superior position of the retina. This assumption is less reliable when detachments are complicated by marked retinal fibrosis.

Natural History

Untreated retinal detachment will often eventually result in massive fibrosis of the retina and vitreous cavities, and subsequently be complicated by uveitis and cataract formation. The eye may eventually become glaucomatous or phthisical. This chain of events is accelerated in cases that have been unsuccessfully treated. Untreated subtotal retinal detachments that do not become complicated by fibrosis may remain static for years, walled off by demarcation lines, although the detached retina itself becomes extremely atrophic. Spontaneous reattachment of the retina may occur but is rare.

Predisposing Factors

Retinal detachment has a strong bilateral tendency (19% of a non-traumatic group; Schepens and Marden, 1966) and is associated with a variety of predisposing factors which result in either retinal weakness, vitreous traction, or both. The most important associations are myopia, aphakia, retinal degenerations, trauma, and retinal neovascularisation.

Myopia

Myopia predisposes an eye to retinal detachment mainly because of degenerative changes in the vitreous itself, resulting in liquefaction of the vitreous gel and separation of the gel from the retina, with residual areas of strong attachment to underlying retina, particularly in the equatorial region. It has also been found (Karlin and Curtin, 1976) that the peripheral degenerative changes of lattice degeneration, pigmentary degeneration and paving stone degeneration, and white without pressure are more likely to be found in myopic eyes, possibly indicating the vulnerability of the peripheral retina in myopia. It has been estimated (Gernet, 1967) that retinal detachment occurs eight times more frequently in myopes than in the normal population. The higher the degree of myopia the greater the risk of retinal detachment (Kaufmann, 1969). When high myopia is present with retinal detachment, the male population is more frequently affected than the female population, a tendency that is not found in the lower levels of myopia (Schepens and Marden, 1966).

Aphakia

Patients who have had cataracts removed are at particular risk of developing retinal detachment (1%–2% of aphakic patients). The mechanism of aphakic detachment has not been completely explained and is probably multifactorial. One factor may be that vitreous gel tends to occupy the space previously filled

by the lens, resulting in a high incidence of posterior vitreous detachment, and increasing the risk of retinal traction and hole formation, although aphakic detachment still occurs after intraoccular lens implantation. Rupture of zonular attachments to the peripheral retina at the time of lens removal may also be a contributory factor and account for the small post-oral holes that are found particularly in aphakic detachments. In the majority of cases detachment develops within the first year of extraction, with particular risk within the first three months. In the aphakic case, if there are additional predisposing causes, e.g. underlying myopia or associated lattice degeneration, there is also increased risk of detachment.

Retinal Degenerations

Some retinal degenerations, for example lattice or snail track degeneration, have a particular predilection to retinal hole formation and subsequent retinal detachment.

Trauma

In severe trauma the association between the traumatic event and any subsequent retinal detachment is obvious (Eagling, 1974; Ruiz, 1969). The injury is either contusive or penetrative. In a study of retinal detachment associated with ocular contusion (Cox et al., 1966) retinal detachment developed soon after injury — 80% within two years. In this kind of injury the typical lesion is a retinal dialysis, but less frequently large round holes or horseshoe-shaped tears are found. In contusive injuries the retinal holes are usually produced either at the site of the impact of the blow (e.g. in the lower temporal quadrant) or indirectly of a contracoup nature as in an upper nasal dialysis. Severe contusion resulting in retinal detachment has also been reported as being part of a battered baby syndrome (Mushin and Morgan, 1971).

In association with contusive injuries, certain occupations and sports may carry some risk. Direct injury is likely in boxing and indirectly in various games such as squash racquets where the players may be struck by the ball or the racquet (Ingram and Lewkonia, 1973).

In a study of penetrative posterior segment intraocular foreign bodies (Percival, 1972) it was found that retinal detachment usually occurred after the foreign body had been removed, and appeared within eighteen months in the majority of cases. These detachments were usually the result of progressive intraocular fibrosis and, in general, the likelihood of detachment was related to the degree of intraocular damage produced, both by the injury itself and the surgical treatment in removing the foreign body. Surgical trauma may also occur as a result of inadvertent perforation of the globe during squint surgery (Gottlieb and Castro, 1970) and this may lead to retinal detachment many years later (Basmadjian et al., 1975).

The role of less obvious trauma in the production of retinal detachment is hard to assess. It was considered (Tulloh, 1968) that trauma was not a significant factor in the majority of detachments, but it is not unreasonable to suppose that comparatively trivial trauma may precipitate detachment in an eye with a strong underlying predisposition to such an event, for example a patient with established lattice degeneration and retinal hole formation; this point may be of some medico legal significance.

Retinal Neovascularisation

The natural history of neovascularisation arising from the retinal vessels is one of eventual infiltration with fibrous tissue. If the vascular process has assumed the shape of large fronds the resulting fibrovascular complex may be extensive. The complex, adherent to a retracting posterior vitreous face, may result in traction retinal detachment of the retina itself. This may occur in any disease in which new vessels form, e.g. diabetes, HbSc disease, venous occlusion, Eales disease, or retrolental fibroplasia. Retinal detachments that follow neovascularisation may be either simple traction detachments or, if the traction has in itself resulted in formation of a full thickness retinal tear, have a complicating rhegmatogenous element.

Rhegmatogenous Retinal Detachment

Classical Symptoms

The classical story of a rhegmatogenous retinal detachment is that of premonitory symptoms of flashes of light, caused by vitreous traction on the retina, a sudden shower of floaters, due to vitreous haemorrhage resulting from retinal hole formation, followed by the onset of a field defect caused by the retina separating. The timing and presence of all three symptoms is variable, and only approximately 50% of patients suffering from retinal detachment are found to have premonitory symptoms (Morse and Scheie, 1974). Flashes of light are usually experienced in the temporal field of vision and they have no localising value as to the site of the retinal hole or vitreous traction; usually they last for a few seconds and may be experienced several times a day for several days (Robertson, 1972). Floaters generally appear several days after the onset of the flashes of light. The symptoms created by the vitreous haemorrhage depend upon its severity and vary from a slight shower of black spots, the patient often noticing the blood in the vitreous cavity as a moving lace-like curtain, to extensive blacking out of vision. The vitreous haemorrhage is usually a single event, although occasionally, if a retinal vessel is avulsed with a round

operculum from the surface of the retina, there may be multiple haemorrhages, which repeat over months or years (Robertson et al., 1971). It should be noted that flashes and floaters may merely represent detachment of the vitreous itself. In a survey of patients with posterior vitreous detachment who presented with flashes and floaters it was found (Jaffe, 1968; Vena, 1972) that only 10%–15% of them had retinal holes, confirming the observations of others (Linder, 1966; Tasman, 1968). With posterior vitreous detachment if blood is present it is found in the retrovitreal or intravitreal compartments or in both. When posterior vitreous detachment occurs in association with vitreous haemorrhage it should always be assumed that there is a retinal hole, although on occasions no cause can be found (Morse et al., 1974).

Haemorrhages occurring with posterior vitreous detachment are usually small and punctate on the retinal surface, but occasionally extensive haemorrhage around the disc has been reported (Cibis et al., 1975). If a retinal hole is to progress to retinal detachment, the detachment occurs within a few days or weeks of the formation of the retinal hole. It is exceptional for a retinal hole to lead to detachment months or years after such a hole has been produced.

A field defect develops when the retinal detachment extends posterior to the equator, and therefore it may be quite extensive before such a defect is noticed. The field defect can be of localising value in positioning the starting point of the retinal detachment; for example, an inferior nasal field defect will indicate a superior temporal detachment and an inferotemporal defect will indicate a superior nasal detachment. Superior defects are of less localising value because a large inferior retinal detachment may be produced either as a result of an inferior hole with fluid ascending from below, or as a result of a superior hole resulting in subretinal fluid tracking inferiorly, leaving the superior retinal mainly attached. In superior detachments the inferior field defect extends rapidly to involve central vision when the macula detaches and, eventually, to affect all quadrants. These field defects are usually noticed early. Superior field defects, however, are characteristically often not noticed for weeks and months in spite of eventual macula detachment. The defect is itself unlikely to extend much above the macula because inferior half retinal detachments rarely progress far above the mid-line.

The field defect itself is sometimes of variable quality. Thus, a detachment that involves the upper half of the retina and produces an inferior field defect may be noticeable in the evening but during the night, if the patient lies in a horizontal position, which brings the hole into a dependent position, there may be redistribution of subretinal fluid into the vitreous cavity and progressive flattening of the retina. Thus, in the morning, the field defect may have disappeared entirely, only to reappear again as the day progresses. Such a field defect may lead to diagnostic confusion, the patient being labelled as hysterical, particularly if a careful fundal examination is not made at the appropriate time of the day. If the macula is affected in these cases there may be fluctuation in central vision or production of the macular symptoms of micropsia and metamorphosia, again with better vision and absence of symptoms in the morn-

ing, worsening as the day progresses. These symptoms highlight the need, when the macula is found to be abnormal, for examining the periphery of the retina in exactly the same way as the retinal periphery is examined for vascular anomalies when exudates are found in the macular region.

Other Symptoms

In some cases of retinal detachment the classical symptoms may not occur, the detachment being identified in other ways, such as – (a) when visual acuity is found to be reduced either subjectively by the patient or during a routine eye examination, and (b) when there are no symptoms but the patient has experienced a similar condition in his other eye or has needed an ophthalmological examination for unassociated eye trouble.

Reduced Visual Acuity

Reduction of visual acuity as the only feature of detachment arises in the following ways –

Inferior Retinal Holes. Retinal detachments associated with inferior retinal holes (e.g. a retinal dialysis) have an insidious course; premonitory symptoms rarely occur, the associated superior field defect is rarely noticed, and the detachments usually progress slowly until the macula is detached. Of detachments due to retinal dialysis (as opposed to 70% in other detachments) 84% were found with the macular detached and central vision severely depressed (Chignell, 1973).

Aphakic Detachment. If retinal detachment occurs rapidly after cataract extraction, the detachment will be detected only when ophthalmoscopy of the posterior segment has been performed after surgery because the patient will have had little chance to appreciate good vision following the extraction.

Holes Arising from the Posterior Pole. Retinal detachment associated with macular holes starts as a central defect and with reduced central vision even if the visual acuity has already been depressed by previous macular degenerative change. Similarly, traction detachments involving the posterior pole (e.g. associated with proliferative diabetic retinopathy) may result in further depression of what may already have been reduced central vision.

Vitreous Haemorrhage. Severe vitreous haemorrhage associated with retinal hole formation may depress central vision even if retinal detachment is slight.

Uveitis. The presenting feature of the detachment may be uveitis secondary to retinal detachment, and reduced central vision. A thick flare and cells, often

with fibrinous exudate, may be found in the anterior chamber with posterior synechiae, and white cells are found in the retrolental space. Rhegmatogenous retinal detachment should be excluded in all patients in whom uveitis has been diagnosed when the posterior segment of the eye is examined. Similarly, a detachment found in association with uveitis should not be assumed to be exudative.

Asymptomatic Detachments

Occasionally, peripheral retinal detachments are discovered although there have been no symptoms, and no depression of central vision. They are detected during routine ophthalmoscopy, often in patients in whom there is a high index of suspicion, as, for example, in the other eye of a patient with a retinal detachment, or of a detachment associated with inferior dialysis after trauma.

Examination Techniques

A general ocular examination of both eyes is made with the object of excluding coexisting ocular disease before centring attention specifically on the retina. The main instruments for examining the retina are the indirect ophthalmoscope and scleral depressor (Schepens, 1951), and the three-mirror gonioscope contact lens. The methods of usage and importance of these instruments has been so well documented that it is necessary here to comment on them only briefly.

Whatever surgical methods are used it is agreed by all retinal detachment surgeons that complete mastery of the techniques of examination is essential before modern retinal surgery can be successfully and consistently be performed. The examination must be thorough and unhurried, with the patient lying comfortably on a couch in a darkened room, under full mydriasis of the pupils (phenylephrine 10% and mydrilate 1% are the drops commonly used).

Indirect Ophthalmoscopy

Indirect ophthalmoscopy combined with scleral depression and three-mirror gonioscopy are complementary forms of examination, and they are not mutually exclusive. The ophthalmoscope is mainly useful for examining the retina itself, while the gonioscope is of particular value in providing a magnified view of retinal details, thus acting as a check on the indirect ophthalmoscope findings, for making a detailed examination of the angle of anterior segment, and of the mid and posterior portions of the vitreous body, with particular reference to points of attachment of the vitreous to the underlying retina and to vitreous

movement. In all cases of retinal detachment ophthalmoscopy and gonioscopy are used on both eyes.

Scleral Depression

The scleral depressor makes it possible to see areas of retina that are normally hidden from view. A further advantage is that small movements of the depressor under the retina give an additional dimension by improving stereopsis and altering light reflection which is particularly useful by showing up small holes in relief.

Application of the depressor over the anaesthetised conjunctiva, though useful on occasions, is seldom necessary; the scleral depressor should be used over the lids. As a rule, in most patients, scleral depression is performed without much difficulty, but on occasions it proves extremely troublesome, particularly in patients who have undergone recent surgery. The eye may be photophobic and lachrymation excessive. Enophthalmic eyes are also technically difficult to examine.

a b

Fig. 1.3. As well as indenting, the scleral depressor acts as a retractor of the lid over which it is applied, as does the middle finger of the hand holding the condensing lens

Ambidexterity with lens and scleral depressor must be mastered in order to examine all retinal details: thus, when examining the upper nasal quadrant of the right eye from the patient's right-hand side, or the superior temporal quadrant of the left eye from the same side (Fig. 1.3a), the examiner holds the scleral depressor in the left hand and the condensing lens in his right. The reverse arrangement of lens and depressor is used when examining the superior nasal quadrant of the left eye and the superior temporal quadrant of the right eye when examining the patient from the left-hand side (Fig. 1.3b). The inferior and superior portions of the retina are examined from above and below respectively on the same side of the patient, and the examiner's head tilted accordingly. A variety of aspheric condensing lenses are used. The 20 diopter (Nikon) lens is by far the most useful for general purpose examinations. This applies to preoperative examinations and use in the theatre. The 16 diopter lens is occasionally of value when greater magnification is needed, as also is the 30 diopter lens, particularly when the retina is being examined through a small pupil and an overall view is required.

It hardly needs to be stressed that all examinations should be performed with gentleness. Force is never necessary, and if used will only result in a protesting and uncomfortable patient. In some apprehensive patients the examination may have to be performed several times over short periods so that the patient's confidence in the examiner may be built up. After several sessions the patient, who may initially have been a most difficult subject, will often tolerate the procedure well and allow adequate examination to be made.

Examining Children

In the rare cases when retinal detachment occurs in very young children, the examination poses particular problems. The child may be unco-operative and usually is apprehensive. In these conditions the examination must not be allowed to take too long and the subject's confidence in the examiner must be built up and established gradually until with skill and patience it is possible to examine the retina in detail. Only the very gentlest scleral depression should be practised on children, indeed often it is never possible to achieve the desired result. However, in the most difficult cases only a general examination of the fundus may be made and detailed examination of the retina deferred until the patient is anaesthetised at the time of surgery. An examination should then be made with scleral depression, and a fundal chart completed in the operating room before surgery is started.

Retinal Charts

Retinal drawings are prepared of both eyes, and the time spent by the surgeon in completely familiarising himself with the retina on which he is going to operate will result in much saving of time during the operation itself, mainly

by making it easier for him to localise holes and other landmarks. When the retinal detachment has been accurately charted it should be possible to form a clear-cut idea of what needs to be done at surgery. If there is to be a period of bed-rest before operation it will be necessary to re-examine the retina before surgery because if subretinal fluid is redistributed by bed-rest the appearance of the retinal detachment may change dramatically and, thus, the surgeon will have to familiarise himself with the new conditions before operating.

Direct Ophthalmoscopy

This is of little use in examining the detached retina in most cases but may on occasion be of some help with patients who have very small pupils. It should, however, be used to examine the optic disc.

Visual Fields

The peripheral field will start to become affected only when the detachment has extended posterior to the equator. Visual fields therefore have little part to play in the diagnosis of retinal detachment, or in subsequent management, other than to confirm that the patient's objective impression of improvement, and to record such improvement. Visual field defects should, however, be charted on both central and peripheral fields if there is evidence of glaucoma. The visual field in retinal detachment usually shows areas of reduced sensitivity on the edge of a dense scotomatous area corresponding with the detached retina. This area of reduced sensitivity has occasionally been used to detect the difference between the typical abrupt dense cut-off of retinoschisis elevation.

Normal Details of the Posterior Segment

Fundus

The examiner should be familiar with the normal anatomical variations in the fundal appearances so that both harmless and potentially dangerous aspects are recognised. The normally occurring anatomical landmarks also provide a method of orientating the surgeon in relation to detachments, attendant retinal holes, and retinal degenerations, particularly with a view to establishing where the scleral buckle has to be placed at the time of surgery.

Anatomical Landmarks

The distance from the ora serrata to the limbus is approximately 6 mm, slightly more than this on the temporal side and slightly less nasally (Straatsma et al.,

1968). The pars plana is therefore situated anterior to this measurement and may be safely perforated by injections 4 to 5 mm from the limbus. The equator is approximately 6 mm further posteriorly to the ora (i.e. 12 mm from the limbus). Externally, the equator is marked as being just in front of the scleral exit to the vortex veins and internally to the horizontal part of the vortex veins before entering the ampulla of the veins.

Intraocular Vessels

Long Ciliary Arteries

The long ciliary vessels which always accompany long ciliary nerves are found in two bundles. They are remarkably constant in their position and are found in the horizontal meridian of the eye (see Fig. 2.13). Each bundle emerges at approximately the mid point between the optic disc and the equator and is usually readily seen on fundoscopy because of the increased pigmentation on the sides of the bundles as they run forwards. They are useful because they divide the retina into upper and lower parts and help surgical orientation. They must be avoided when the surgeon is working in the region of the horizontal rectus muscle as they can be damaged when buckling sutures are placed or when subretinal fluid is being drained.

Short Ciliary Arteries

These are more irregular and variable in their appearance than long ciliary arteries and quite often cannot be seen. They are usually situated around the vertical meridian, and the short ciliary nerves, which are seen as whitish marks on the fundus (see Fig. 2.13), are not often accompanied by the arteries, which are much more difficult to see. These vessels are of much less significance and less important to the surgeon.

Vortex Veins

The vortex veins vary considerably in number (usually between 5 and 8) and are readily seen on fundoscopy in the lightly pigmented subject (see Fig. 2.8). On ophthalmoscopy there is great anatomical variation in the way the veins enter their ampullae. The vortex veins are found in greatest number in the nasal quadrant, the upper half being favoured more than the lower. The superior temporal quadrant, which is the commonest quadrant for the surgeon to be operating in, has only one vortex vein. The establishment of the site of the vortex veins, usually determined at the time of surgery because retinal detachment obscures them on fundal examination, is particularly important to the surgeon if the veins are near retinal tears. If they are, there is risk of their being damaged as the result of cryotherapy, scleral sutures, or during drainage of subretinal fluid.

Peripheral Retina

This area includes the ora serrata and the retina between the ora and the equator, and is of particular importance to the retinal surgeon and one that demands painstaking observation and great experience in interpretation. The majority of retinal tears and degenerations are found in this part of the retina.

Ora Serrata

The ora serrata is most easily seen in the aphakic patient, provided there are no opacities in the media and good pupillary dilatation is achieved. In all other cases skilled scleral depression is invariably required to define the details of the area clearly. The dentate processes that make up the ora serrata are more prominent on the nasal side, and on the temporal aspect, have a rather flat, inconspicuous appearance, particularly in the lower temporal quadrant. The processes show considerable individual variation and are less obvious in children (Straatsma et al., 1968). Although a detailed drawing of the ora serrata is not necessary in retinal detachment surgery, it is important to be aware of local anatomical variations so that physical signs are not misinterpreted.

The following important variations in the ora serrata may be found (Spencer et al., 1970), (see Fig. 1.4).

1. A *giant tooth* extends almost to the ciliary process.
2. An *enclosed oral bay* may be formed by joined processes, and this may be mistaken for a peripheral retinal hole.
3. *Meridional folds* are often found and are posterior and upward projections of full thickness retina. They may occasionally be associated with small, round retinal holes at the posterior aspect of the fold. A meridional complex is defined as an alignment of a meridional fold and a ciliary process in the same meridian.
4. *Granular tissue* consisting of small whitish opacities of various shapes and sizes, probably derived from the retina itself, are often found in the postoral

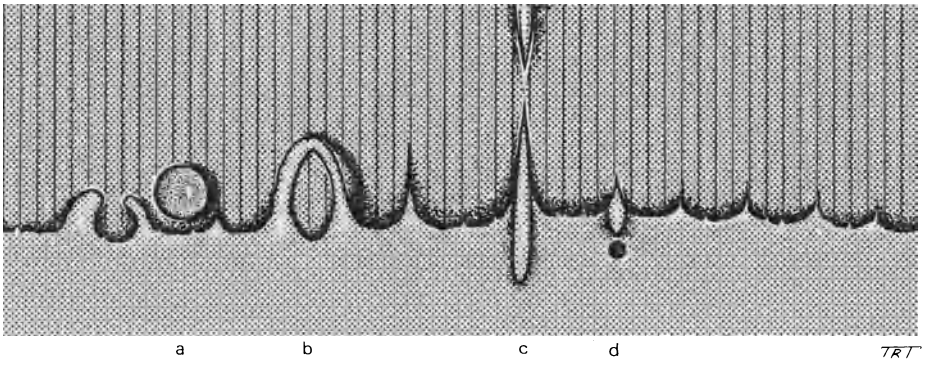

Fig. 1.4. (a) Pars plana cyst; (b) an enclosed oral bay; (c) a meridional complex; (d) a meridional fold adjacent to a small retinal hole

region, particularly on the nasal side, on the surface of the retina itself or drawn into the vitreous. These appearances may be mistaken for small peripheral opercula. However, their extremely small size, their peripheral distribution and their multiplicity are distinguishing features.

Pars Plana

The pars plana is usually flat and featureless. However, small pars plana cysts may be found and are quite common changes in the ageing eye (Okun, 1961b). The cysts are similar to the Blessig-Iwanoff cystoid spaces of the peripheral retina.

Vitreous

In the emmetropic young adult eye, the vitreous occupies a volume of approximately 4 ml. The volume varying with the size of the globe and being greatly increased in high myopia. The major constituents of vitreous are water, collagen, hyaluronic acid, and soluble proteins. It is the interaction of the double helices of hyaluronic acid with the collagen fibrils that gives the vitreous its gel structure (Roughley, 1975). Pathological states of the vitreous such as haemorrhage, infiltration of white cells or ageing, tend to eliminate this co-operative interaction and convert the vitreous gel into a more liquid state.

The normal vitreous is closely applied to the inner layers of the retina and is continuous with the internal limiting membrane. It is particularly firmly attached to the underlying retina around the disc (Gaertner and Graefes, 1962) at the macula (Grignolo, 1953; Schepens, 1954), to superficial retinal vessels (Rieger, 1943), and at the vitreous base, an area which extends for approximately 1–2 mm on each side of the ora serrata into the retina posteriorly and into the pars plana anteriorly. The posterior part of the vitreous base is of particular importance for it is at this point that traction is exerted if a detached posterior vitreous swings freely on its anchorage at this point.

Other Investigations

The following investigations are not routinely performed in the examination of retinal detachment but are of value when there is difficulty in establishing the diagnosis.

Transillumination

This may help to distinguish between a rhegmatogenous and solid non-rhegmatogenous retinal detachment. In most cases, however, the presence of a solid tumour is obvious on indirect ophthalmoscopy.

Fluorescein Angiography

This may be of value (Norton, 1967; Gass, 1972) when considering the possibility of malignant melanoma and other diseases may that cause nonrhegmatogenous detachment (e.g. Harada's disease).

Ultrasonography

Particularly in the form of a B-scan, ultrasonography may be of value in eliciting the presence and extent of retinal detachment when there is severe opacity in the media (Coleman and Jack, 1973; Jack et al., 1974).

X-rays

A plain X-ray film of the eye may be necessary to exclude the presence of an intraocular foreign body, particularly if there is a need to check that a foreign body has been completely removed, or in the assessment of a retinal detachment of unknown aetiology.

References

Ashrafadeh, M.T., Schepens, C.L., Elzeneiny, I.I., Moura, R., Morse, P., Kraushar, M.F.: Aphakic and phakic retinal detachment. Arch. Ophthalmol. *89*, 476–483 (1973)

Basmadjian, G., Labelle, P., Dumas, J.: Retinal detachment after strabismus surgery. Am. J. Ophthalmol. *79*, 305–309 (1975)

Chignell, A.H.: Retinal dialysis. Br. J. Ophthalmol. *57*, 572–577, 1973

Chignell, A.H., Carruthers, M., Rahi, A.H.S.: Clinical, biochemical and immuno-electrophoretic study of subretinal fluid. Br. J. Ophthalmol. *55*, 525–532 (1971)

Cibis, G.W., Watzke, R.C., Chua, J.: Retinal haemorrhages in posterior vitreous detachment. Am. J. Ophthalmol. *80*, 1043–1046 (1975)

Coleman, D.J., Jack, R.L.: B-scan ultrasonography in diagnosis and management of retinal detachment. Arch. Ophthalmol. *90*, 29–34 (1973)

Cox, M.S., Schepens, C.L., Freeman, H.M.: Retinal detachment due to ocular contusion. Arch. Ophthalmol. *76*, 678–685 (1966)

Davis, M.D.: Natural history of retinal breaks without detachment. Arch. Ophthalmol. *92*, 183–194 (1974)

Eagling, E.M.: Ocular damage after blunt trauma. Br. J. Ophthalmol. *58*, 126–140 (1974)

Foulds, W.S.: Aetiology of retinal detachment. Trans. Ophthalmol. Soc. U.K. *95*, 118–128 (1975a)

Foulds, W.S.: The vitreous in retinal detachment. Trans. Ophthalmol. Soc. U.K. *95*, 412–416 (1975b)

Gaertner, J., Graefes, I.: Electronenmikroskopische Untersuchungen über die Feinstrukturen der normalen und pathologisch vitreoretinalen Grenzschicht. Arch. Ophthalmol. *165*, 71–102 (1962)

Gass, J.D.M.: Fluorescein angiography – an aid to the retinal surgeon. In: Retinal congress. Pruett, R.C., Regan, C.D.J. (eds.), pp. 181–200. New York: Appleton Century Crofts 1972

Gernet, H.: About relative and absolute frequency of retinal detachments in myopes. Ophthalmologica *154*, 373–377 (1967)

Gottlieb, F., Castro, J.L.: Perforation of the globe during strabismus surgery. Arch. Ophthalmol. *84*, 151–157 (1970)

Grignolo, A.: Fibrous components of the vitreous body. Arch. Ophthalmol. *47*, 760–774 (1952)

Grignolo, A.: Les connaissances actuelles sur la structure du corps vitré. In: Advances in Ophthalmology, Vol. 2. Streiff, E.B. (ed.), pp. 1–35. Fortschr. Augenheilkd. Basel, New York: Karger 1953

Ingram, D.V., Lewkonia, I.: Ocular hazards of playing squash racquets. Br. J. Ophthalmol. *57*, 434–438 (1973)
Jack, R.L., Hutton, W.L., Machemer, R.: Ultrasonography and vitrectomy. Am. J. Ophthalmol. *78*, 265–274 (1974)
Jaffe, N.A.: Complications of acute posterior vitreous detachment. Arch. Ophthalmol. *79*, 568–571 (1968)
Karlin, D.B., Curtin, B.J.: Peripheral chorio-retinal lesions and axial length of the myopic eye. Am. J. Ophthalmol. *81*, 625–635 (1976)
Kaufmann, T.: Myopia and retinal detachment, a statistical analysis of 800 cases. Ophthalmologica *157*, 249–262 (1969)
Kaufman, P.L., Podos, S.M.: Subretinal fluid — butyrylcholinesterase. Am. J. Ophthalmol. *75*, 627–636 (1973)
Lincoff, H.A.: The prophylactic treatment of retinal detachment. Arch. Ophthalmol. *66*, 48–60 (1961)
Lincoff, H.A., Gieser, R.: Finding the retinal hole. Arch. Ophthalmol. *85*, 565–569 (1971)
Linder, B.: Acute posterior vitreous detachment and its retinal complications. Acta. Ophthalmol. [Suppl.] (kbh) *87*, 1–108 (1966)
Morse, P.H., Scheie, H.G.: Prophylactic cryoretinopexy of retinal breaks. Arch. Ophthalmol. *92*, 204–210 (1974)
Morse, P.H., Aminlari, A., Scheie, H.G.: Spontaneous vitreous haemorrhage. Arch. Ophthalmol. *92*, 297–298 (1974)
Mushin, A., Morgan, G.: Ocular injury in the battered baby syndrome. Br. J. Ophthalmol. *56*, 343–347 (1971)
Neumann, E., Hyams, S.: Conservative management of retinal breaks. Br. J. Ophthalmol. *56*, 482–486 (1972)
Norton, E.W.D.: The use of fluorescein in the differential diagnosis of secondary retinal detachments. Mod. Probl. Ophthalmol. *5*, 202–217 (1967)
Okun, E.: Gross and microscopic pathology in autopsy eyes. Part III. Retinal breaks without detachment. Am. J. Ophthalmol. *51*, 369–391 (1961a)
Okun, E.: Gross and microscopic pathology in autopsy eyes. Part IV. Pars plana cysts. Am. J. Ophthalmol. *51*, 1221–1228 (1961b)
Osterlin, S.: Preludes to retinal detachment in the aphakic eye. Mod. Probl. Ophthalmol. *18*, 464–467 (1977)
Percival, S.P.B.: Late complications from posterior segment intra-ocular foreign bodies. Br. J. Ophthalmol. *56*, 462–468 (1972)
Rieger, H.: Zur Histologie der Glaskörperabhebung. Albrecht von Graefes Arch. Klin. Ophthalmol. *146*, 305–335 and 452–462 (1943)
Robertson, D.M.: Vitreous floaters and flashing lights: vitreous collapse and sequelae. Minn. Med. *55*, 314–317 (1972)
Robertson, D.M., Curtin, V.T., Norton, E.W.D.: Avulsed retinal vessels with retinal breaks. Arch. Ophthalmol. *85*, 669–672 (1971)
Roughley, P.J.: Helix formation — its involvement in the formation of gel structures. Trans. Ophthalmol. Soc. U.K. *95*, 369–372 (1975)
Ruiz, R.S.: Traumatic retinal detachments. Br. J. Ophthalmol. *53*, 59–61 (1969)
Schepens, C.L.: Progress in detachment surgery. Trans. Am. Acad. Ophthalmol. Otolaryngol. *55*, 606–615 (1951)
Schepens, C.L.: Clinical aspects of pathological changes in the vitreous body. Am. J. Ophthalmol. *38*, 8–21 (1954)
Schepens, C.L., Marden, S.: Data on the natural history of retinal detachment. Am. J. Ophthalmol. *61*, 213–226 (1966)
Spencer, L.M., Foos, R.Y., Straatsma, B.R.: Meridional folds, meridional complexes and associated abnormalities of the peripheral retina. Am. J. Ophthalmol. *70*, 697–714 (1970)
Straatsma, B.R., Landers, M.B., Kreiger, A.E.: The ora serrata in the adult human eye. Arch. Ophthalmol. *80*, 3–20 (1968)
Tasman, W.S.: Posterior vitreous detachment and peripheral retinal breaks. Trans. Am. Acad. Ophthalmol. Otolaryngol. *72*, 217–224 (1968)
Tulloh, G.G.: Trauma in retinal detachment. Br. J. Ophthalmol. *52*, 317–321 (1968)
Vena, A.: La rôle joué par le corps vitre dans la pathogenie des déchirures de la rétine. Ann. Ocul. *205*, 295–298 (1972)
Zauberman, H., Guillebon, H., de: Retinal traction in Vitro. Invest. Ophthalmol. *11*, 46–55 (1972)

Chapter 2

Preoperative Examination

The patient should first be examined by slit lamp, followed by an examination of the posterior segment by indirect ophthalmoscopy and scleral depression, and then returned to the slit lamp for an examination with the three-mirror gonioscope.

Initial Slit-lamp Examination

The initial examination of the cornea, anterior chamber, lens iris diaphragm, retrolental space and the anterior vitreous is made with the slit-lamp.

Cornea

Corneal opacities may interfere with the view of the fundus, but it is unusual for opacities to be of such severity that they interfere seriously with fundal examination. The view through the periphery of the cornea is usually adequate even if there are central corneal opacities, nevertheless a dense peripheral opacity may make observation of peripheral retina difficult; also corneal suturing following a recent intraocular foreign body repair may produce an annoying distortion of the retinal image. It is extremely rare for any preliminary corneal graft procedure to be necessary prior to retinal surgery. After phakic grafts, the incidence of retinal detachments does not increase but does so after aphakic grafts in which there has been vitreous manipulation (Forstot et al., 1975).

Anterior Chamber

Anterior uveitis may be found in association with retinal detachment and is of two main types.

1. Uveitis secondary to retinal detachment is the more commonly encountered type, and although usually associated with a long-standing retinal detachment it may occur after detachment has been present for only a few days. It is not known why some cases of detachment excite such a marked inflammatory response, which may be brisk, with the formation of posterior synechiae and secondary glaucoma. In addition to flare and cells in the anterior chamber, there may be cellular infiltration of the anterior vitreous, and if this is severe, it may mask the underlying detachment.

2. Retinal detachment may be secondary to an underlying uveitis or pigment epitheliitis and is of two main types:

a) bullous detachments found in the Harada-like syndromes, mainly detected in the inferior fundus, and

b) small and relatively insignificant, inferior detachments found in the extreme periphery of the fundus in some cases of generalised uveitis of a non-specific type (Brockhurst and Schepens, 1968).

The depth of the anterior chamber is noted and, if considered to be at all shallow, can be measured on a pacometer. The angle will be assessed during the three-mirror examination.

The intraocular pressure is routinely recorded and in the majority of cases the pressure is lower in the eye with a retinal detachment due to a reduction of aqueous secretion (Tulloh, 1972). Occasionally, raised intraocular pressure may be found in an eye with retinal detachment (Schwartz, 1973). Raised intraocular pressure indicates glaucoma of an underlying nature (chronic simple glaucoma or aphakic glaucoma, usually) or resulting from the detachment itself, in which case it is usually associated with secondary uveitis. In some series, the incidence of glaucoma was found to be high, with an incidence of 9.5% (Phelps and Burton, 1977), of which over 7% was found to be glaucomatous before detachment occurred. Reduced pressure may be found in eyes that have underlying chronic glaucoma and it is therefore important to watch the intraocular pressure after re-attachment has been achieved (Smith, 1963). Cases of preoperative rise of intraocular pressure secondary to uveitis resolve spontaneously after the retina has become re-attached.

Iris

If pupillary dilatation is not achieved to the level of approximately 3 to 4 mm, then great difficulty will be found in accurately examining details of the fundus peripheral to the equator. The cause of the pupil failing to dilate may be physiological, e.g. in heavily melanotic irides, or due to one or other of a variety of pathological states such as diabetes, aphakia, (particularly if the cataract surgery has been complicated), Marfan's syndrome, or glaucoma. Iris atrophy may be discovered but its significance is difficult to interpret, particularly if the eye has been subjected to previous retinal surgery or surgery in the anterior segment. However, if segmental iris atrophy is found following previous retinal detachment surgery, ischaemia of the anterior segment should be suspected. These changes may be confirmed by iris fluorescein angiography, the examiner thereby being warned against the risk of further anterior segment ischaemia after further buckling procedures.

Lens

Position of Lens

If the lens is subluxated or even dislocated, as may be found in Marfan's syndrome or following trauma, difficulties may arise from accurate observation

of the fundus periphery. The presence of intraocular lenses inserted during cataract surgery may make examination of the retina more difficult not only because of poor pupillary dilatation but also because these lenses cause distortion of the retinal view, a distortion that is further augmented by the annoying reflexes arising from the surface of the lens (Norton, 1976).

Lens Opacities

Peripheral cortical opacities are frequent causes of difficulty in examining the fundus periphery. On some occasions it may be possible to obtain only a hazy view. However, in most cases of lens opacity, the observer will find that with good pupillary dilatation and by varying his position and line of observation, most fundal details will be revealed through chinks in the lens striae. Only rarely is it necessary to precede retinal detachment surgery by cataract extraction.

Retrolental Space

Pigment

The retrolental space will usually be found to contain pigment granules (tobacco dusting, Fig.2.1) when there is a retinal detachment (Shafer, 1965; Hamilton and Taylor, 1972), although exceptions to this may be a long-standing retinal detachment such as is found in cases of retinal dialysis or in detachments that have complicated retinoschisis. This type of pigmentation in the retrolental space is of no particular significance, although how the pigment achieves this position is not clearly understood. Presumably it is derived from pigment epithelium in the retina, and traverses the vitreous cavity via the retinal holes.

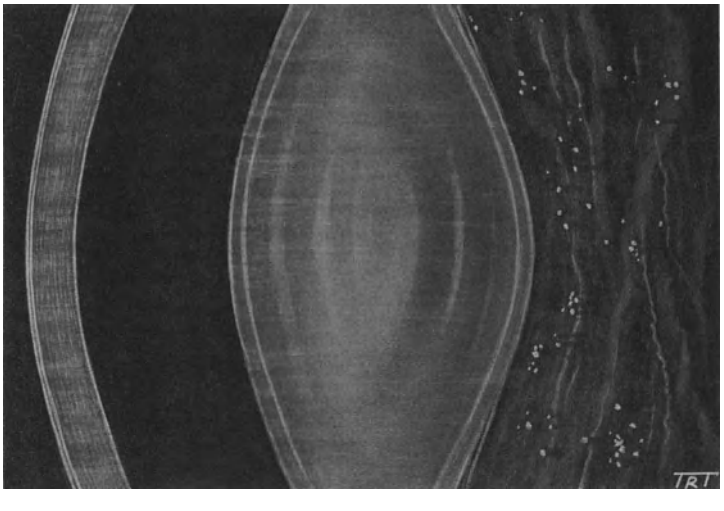

Fig. 2.1. Pigment granules in the retrolental space and anterior vitreous

Red Cells

Red cells in the retrolental space and anterior vitreous are found when there has been extensive vitreous haemorrhage. In particularly severe cases, pooling of blood in a hyphaema-like arrangement is found at the lower border of the lens on its posterior surface, a configuration produced by the attachment of the vitreous gel to the back of the lens.

White Cells

White cells may be found in the retrolental space or anterior vitreous. The accumulation of inflammatory cells in the vitreous may contribute both to vitreous degeneration and collapse (Hogan, 1975).

Anterior Vitreous

Although examination of most of the vitreous cavity is best performed with a three-mirror contact lens, the anterior third of the vitreous gel can also be examined at this stage and much useful information obtained about the state of the vitreous as a whole. Normal vitreous gel appears to have a delicate ribbon-like structure, a characteristic feature being its vigorous mobility, which can easily be seen in the anterior vitreous. Detachment of the vitreous from the lens itself, an unusual finding, is of no particular significance to the retinal surgeon.

When fibrous-like membranes start to form in relationship to a retinal detachment, the vitreous also eventually becomes involved in the process. When this happens, its normal gel structure collapses normal mobility is lost, and the vitreous may become extensively infiltrated by fibrous strands. These semitranslucent strands are usually extensively infiltrated with pigment (pigment epithelial macrophages), often arranged in large clumps in the vitreous cavity and are also closely related to the detached retina. Fibrous strands, pigment infiltration, and gross immobility of gel may easily be seen in the anterior vitreous.

Indirect Ophthalmoscopy of the Posterior Segment

Following the initial slit-lamp examination, the posterior segment is examined by indirect ophthalmoscopy and scleral depression. In the observation and charting of the findings in the posterior segment it is stressed that all the observations are to enable a correct choice of operation to be made. This involves

1. Choosing the type of buckle to be used;
2. Deciding whether or not subretinal fluid needs to be drained;
3. Considering the need for any other special procedures, e.g. vitreous injections.

Details of Retinal Detachment

Macula Involvement

Marked reduction in central vision gives the most important clue as to the likelihood of detachment of the macula, although on occasions reduction of central vision may be due to an overhanging bullous retinal detachment, the macula itself being spared. It is essential to know whether or not the macula is detached for two main reasons:

1. To determine the urgency of the operative procedure. If the macula has not detached, then surgery should be performed as soon as is reasonably possible to prevent extension of the detachment into the macular region.

2. If the macula has been detached, the patient should be warned of the uncertainty of the return of central vision. This latter point may have medico-legal significance, particularly if detachments have occurred after trauma.

The Contour

It has already been seen that the spread of subretinal fluid from a retinal hole occurs in a predictable way and this, in turn, means that the contour of the retinal detachment – the line representing the junction between the attached and detached retina – will indicate the way subretinal fluid has accumulated and thus point to the site of the primary retinal hole. As subretinal fluid accumulates, it pushes the detaching retina progressively ahead of it, producing a contour that is concave towards the advancing accumulation of fluid and to the retinal hole. If on completion of a retinal drawing, the retinal contours and distribution of subretinal fluid cannot be explained on the basis of the retinal hole found, then it is almost certain that a further hole has been missed. In cases in which it is not possible to find a retinal hole, an operation may be planned on the presumed site of the hole and obviate the need for unnecessarily extensive buckling surgery.

Retinal Holes

The description of a retinal hole can be varied either to describe its shape e.g. round or U-shaped, the position of the retinal hole (macula) or even the size of the retinal hole (giant). The site, size and shape of all retinal holes are carefully indicated on the retinal chart.

The observations concerning retinal holes are essential to ensure a rapid and accurate localisation of the holes at surgery and, where possible, for the correct preoperative selection of type and size of buckle to be used.

Round and U-shaped tears are by far the most commonly encountered retinal holes. In one series (Tulloh, 1965) round holes were found most frequently on the temporal side, and in myopic patients the upper temporal quadrant

was preferred, as opposed to the non-myopic, where the lower temporal was the commoner site. In U-shaped tears, in both the myopic and non-myopic patients, the upper temporal quadrant was the commoner site. It can be seen, therefore, that the upper temporal quadrant is of the greatest importance to the retinal surgeon and the one in which he will be performing his buckling procedure most often.

In 60% of cases (Chignell and Markham, 1978) more than one hole is found in the detached retina. Of the cases in which multiple holes are found, the majority (64%) have the retinal holes in fairly close proximity to each other and are not separated by more than one quadrant of detached retina. This again may be of value in deciding where the buckle should be placed if opacities in the media are obscuring part of the view of the retinal detachment.

Retinal dialysis accounts for approximately 10% of detachment cases and giant tears and macular holes 1 to 2% each.

The charting of the position of the retinal holes is made with references to convenient anatomical landmarks; thus, the relationship to the long ciliary bundles will indicate whether the holes are in the upper or lower of the retina, and further orientation is achieved by dividing the retina with an imaginary line drawn vertically through the disc into temporal and nasal halves. The posterior extremity of the holes is estimated by noting the relationship of the holes to neighbouring vortex veins, if they can be seen. The relationship of the retinal holes to local retinal landmarks in the detached retina is assessed, e.g. retinal blood vessels traced as they run peripherally until the tear is reached, or to the proximity of local retinal degenerations, e.g. lattice degeneration. It should be noted that the position of the posterior limit of the retinal hole is always difficult to estimate when there is bullous retinal detachment.

The size of the retinal hole must be estimated so that the correct choice of buckle can be made. The tip of the scleral depressor is approximately 6 mm in width, thus the size of the hole can be estimated provided the retinal hole can be approximated to the scleral depressor. Difficulty in estimating size is encountered when subretinal fluid is deep because it will not be possible to oppose pigment epithelium to the detached part of the retina with a scleral depressor. The actual extent of the retinal hole will be checked at the time of surgery when its extremities are accurately localised.

The shape of the retinal hole is important not only because of the various problems in buckling that the different shapes of retinal hole pose, but also because the shape may be altered by the presence of local pre-retinal membrane (for example, grossly elongating the long axis of a U-shaped tear) or by the folding of the detached retina when the holes are related to the edges of such folds.

Pigmentation

Not infrequently pigmentation may be found in relationship to a retinal hole, particularly at the posterior margin of a hole. Pigmentation of a hole, however,

does not necessarily indicate that it has been there for a long time, for pigmentation may be found in cases that have had symptoms indicating recent onset (Morse and Eagle, 1975). Pigmentation around a hole therefore does not necessarily indicate the length of time that the retinal hole has been there, nor does it mean that the pigmentation has rendered the hole secure and that there is little risk of subsequent detachment.

Although round holes and U-shaped tears show considerable morphological difference, they do not, in general, influence the choice or method of operation to be performed, but they do pose different questions as to how best to seal the defect. However, round holes are smaller and often multiple and may be found with no evidence of active vitreous traction upon them unless associated with areas of retinal degeneration, e.g. lattice degeneration. Consequently, detachments characterised by round holes are less likely to have had a preoperative vitreous haemorrhage or premonitory symptoms than those cases in which horseshoe-shaped tears are found. For this reason retinal detachments arising below and associated with round holes and with slow accumulation of subretinal fluid may be particularly insidious and not noticed by the patient until the macula has detached. When a round hole is formed, there may or may not be an operculum. If there is one, it is usually found detached from the retina and lying on the posterior hyaloid face at a variable distance from the hole itelf, sometimes so close as to make detection of the underlying hole difficult, and sometimes separated by quite a wide space. On many occasions no operculum is present and it is as if the retina has just melted at the site of the retinal hole. When a horseshoe-shaped tear is present, vitreous traction is active on the anterior aspect of the tear.

Subretinal Fluid

Depth

The depth of subretinal fluid between the retinal hole and the underlying pigment epithelium is of particular importance to the surgeon. Although the depth in other sites, for example in bullous retinal detachment, the retina may overhang the disc, or may make observation of retinal holes more difficult, it is the immediate relationship of subretinal fluid to the retinal hole that is important. The depth of the fluid can easily be estimated when scleral depression is performed during the preoperative examination: the hole may be closed easily be gentle indentation, or it may be closed only by firm indentation, or it may not be possible to close it at all. This examination gives the surgeon an estimate of how deep the subretinal fluid will be at the end of operation after the buckle has been raised underneath the hole, if a non-drainage procedure is to be performed. On the basis of this information a decision will have to be made on whether or not subretinal fluid needs to be drained. The depth of fluid underneath the hole is also an important contributory factor in solving

the difficulty of localising the hole at the time of surgery; large tears on high balloons will obviously make localisation more difficult than small holes with little subretinal fluid. In highly elevated holes difficulty can also be expected in visualising the cryotherapy reaction. If deep subretinal fluid is found underneath the hole at the preoperative examination, then a period of bed-rest may be advised in order to encourage approximation of the hole to the pigment epithelium, thus enabling easier localisation and cryotherapy at the time of surgery.

Shift of Fluid

Rapidly shifting bullous subretinal fluid in the inferior retina is a characteristic feature of non-rhegmatogenous retinal detachment. Shift of fluid is, however, sometimes seen in rhegmatogenous detachment and, occasionally, this movement of fluid may be of value in localising the site of the retinal hole if situated above horizontal meridian. When the contour and distribution of fluid indicates the presence of a superior retinal hole, but where in fact one cannot be demonstrated, tilting the patient's head towards the side of the suspected retinal hole may persuade inferior fluid to track upwards and push the hidden hole into view.

Mobility of the Detached Retina

In fresh retinal detachment a characteristic undulating movement of the detached retina is easily seen in the normal course of examination. Two main factors effect the mobility of the detached retina.

1. Depth of Subretinal Fluid

If subretinal fluid is very shallow there is little scope for actual movement of the detached retina which may in fact seem to be immobile. This situation, however, is rarely significant as in the presence of minimal fluid the retinal holes can easily be closed at the time of surgery and there is no need to drain off the subretinal fluid.

2. Presence of Periretinal Membranes

These membranes (described later in detail during the three-mirror examination) convert the detached retina into a more rigid structure which creates the problem, of particular importance both preoperatively and at the time of surgery, of having to decide whether or not the non-drainage operation can be expected to succeed, (Chignell, 1977) since the capacity of the retina to settle back against a buckle may be impaired. It is the retina in the immediate vicinity of the retinal hole that is of particular importance as it is at this point that the buckle will be raised. The mobility of the detached retina is best estimated when it is examined by indirect ophthalmoscopy both preoperatively or in

doubtful cases at the time of operation. Free undulating movements of the retina are easy to see as, also, is complete immobility of retina when heavily infiltrated with membrane. Sometimes the degree of retinal mobility may be difficult to estimate, particularly when early membrane has formed, mobility having been reduced but not completely abolished.

Haemorrhage

Retinal

Small, round haemorrhages are often found in the vicinity of retinal holes in fresh cases of retinal detachment. These are more often associated with U-shaped tears than with round holes, and may be found on the operculum itself, or on the posterior aspect of the break. Their presence often aids the detection of a particularly small tear. Similar haemorrhages are also found in long-standing retinal detachment in which case they are often widely, though thinly, scattered throughout the periphery of the detached retina.

Vitreous

Some degree of vitreous haemorrhage is a common finding in cases of retinal detachment and is associated with a rupture of a retinal blood vessel during the development of the retinal hole. In most cases the haemorrhage is not severe enough to interfere seriously with the observation of the retina, nor does it usually influence the operative technique or choice of operation. Occasionally, however, a haemorrhage will be severe enough to prevent any examination of the fundus at all. Haemorrhage occurs most frequently from an upper half horseshoe-shaped tear and either descends by gravity down the back of the detached posterior vitreous or passes into the vitreous gel itself. The behaviour and spread of vitreous haemorrhage depends mainly on the extent of the haemorrhage, the state of the overlying vitreous, and of its attachments. Retrovitreal haemorrhage can be found in the inferior retina piled up on the back of the posterior vitreous detachment at whatever level this has reached, (Fig. 2.2). In the degenerate myopic vitreous, haemorrhage usually settles rapidly. Conversely, in traumatic detachments that may arise in an eye that has normal gel, large intravitreal and preretinal collections of blood may be found and may take weeks to settle and absorb. In aphakia, this blood may pass forward into the anterior chamber, and there may be hyphaema. Blood introduced into the vitreous cavity will clot due to contact with collagen, and evidence of old haemorrhage is provided by the presence of a whitish coagulum that is found in the inferior vitreous and which often obscures details of the inferior retina. Preretinal haemorrhage, however, will not clot (Constable, 1975) and will absorb without inducing membrane formation. The presence of vitreous haemorrhage is of practical importance for several reasons.

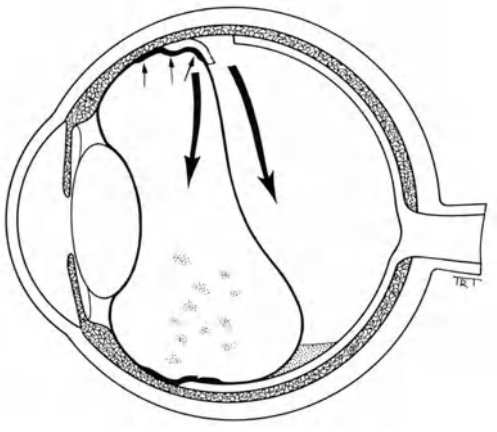

Fig. 2.2. Haemorrhage from a retinal tear passes either into the vitreous cavity or into the retrogel space (*large arrows*). The firm attachment of the vitreous base to ora serrata, peripheral retina, and to the anterior aspect of a horseshoe-shaped tear (*small arrows*) is shown

1. An inadequate view of the fundus may be produced. In these cases a period of bed-rest in the upright position and double padding is advised in order to encourage the vitreous to clear and enable detection of the underlying retinal hole.

2. Recent and extensive vitreous haemorrhage makes it unwise for anything but the most gentle scleral depression in the vicinity of the tear to be performed for fear of precipitating further haemorrhage. This applies to be preoperative examination and particularly to the manipulation of the cryoprobe in the vicinity of the tear at the time of operation.

3. Extensive vitreous haemorrhage may result in a secondary uveitis or glaucoma.

4. The effect of vitreous haemorrhage on subsequent retinal reattachment. Although it has been suggested (Havener, 1973) that eyes with vitreous haemorrhage do not apparently have a predisposition to subsequent intraocular fibrosis and massive preretinal retraction, other workers (Percival, 1973; Chignell et al.; 1973) have found that severe preoperative vitreous haemorrhage indicates that an eye is more likely to pass subsequently into massive vitreous retraction than one that did not have such a haemorrhage. In these cases the haemorrhage, by promoting collapse of gel, encouraging an inflammatory response, and obscuring the view of the retina, thus making surgery more difficult and likely to fail, is probably only one of several factors contributing to the fibrotic reaction.

Choroidal Detachment

Choroidal detachments are occasionally found preoperatively in eyes with retinal detachment (4.5%, Gottlieb, 1972). They are recognised by their characteristic dark rounded shapes seen through the retinal detachment (Fig. 2.3), and occur more commonly in eyes that have previously been operated on (Seelenfreund et al., 1974). They may be localised or annular in extent and have an identical

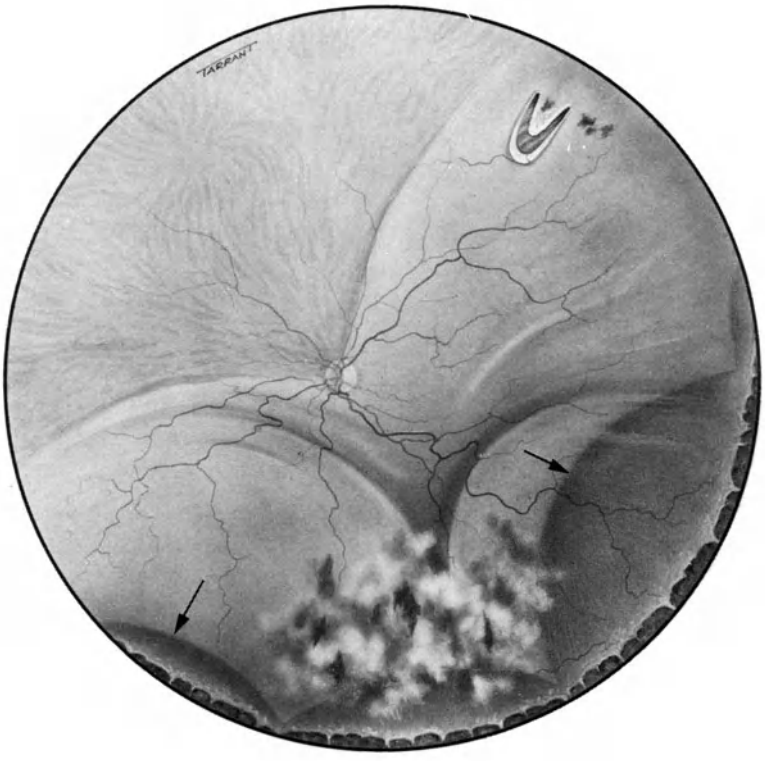

Fig. 2.3. A subtotal retinal detachment. There is associated choroidal detachment in the lower half of the retina (*arrowed*) which has pushed the ora serrata into view. Vitreous haemorrhage with fresh and altered blood can be seen in the inferior vitreous

appearance to the choroidal detachments seen following cataract or glaucoma surgery. The development of preoperative choroidal detachments is probably due to the hypotony resulting from the sudden onset of retinal detachment and reduced aqueous production, the latter being further encouraged by ciliary body detachment, which is often present (Dobree, 1962). Their importance when found in the vicinity of the retinal hole is that it will be impossible to apply cryotherapy through the sclera at the affected site, as the choroid and pigment epithelium are separated and no reaction will appear. In these cases it may be necessary to drain suprachoroidal fluid if the choroidal detachment still exists at the time of operation. Choroidal detachments do not usually disappear spontaneously before retinal surgery, and systemic steroids have, on occasions, been advocated to prompt reabsorption. Some reabsorption may occur spontaneously preoperatively and if the choroidal detachment is in the close vicinity of the retinal hole it is worth postponing surgery for a few days to see if it will. If the choroidal detachment is situated somewhere other than in close proximity to the hole, there is no point in delaying surgery because the choice and method of operation will not be influenced by the choroidal detachment, which will spontaneously disappear in the postoperative period as the retina becomes reattached. It has been suggested (Seelenfreund et al.,

1974) that in eyes that show choroidal detachment pre-operatively there is a higher risk of eventual massive periretinal proliferation.

Signs of Long-standing Detachment

It is useful to be able to give an approximate estimation of how long the retinal detachment has existed. It may be of value in medico-legal cases when injury may incorrectly be considered to have caused a retinal detachment that may in fact have preceded the traumatic incident. Also, if the macula has become detached and central vision reduced, a cautious prognosis regarding the likelihood of recovery of central vision should be given since one of the most important factors governing recovery is the length of time that the macula has been detached.

In long-standing retinal detachment the retina becomes increasingly transparent and develops a cystic appearance. These cystic spaces, which develop in the outer plexiform layer of the retina, may become enormously enlarged and give the appearance of secondary intraretinal cysts (Hagler and North, 1967). From the time of onset of detachment, these cysts usually take at least a year

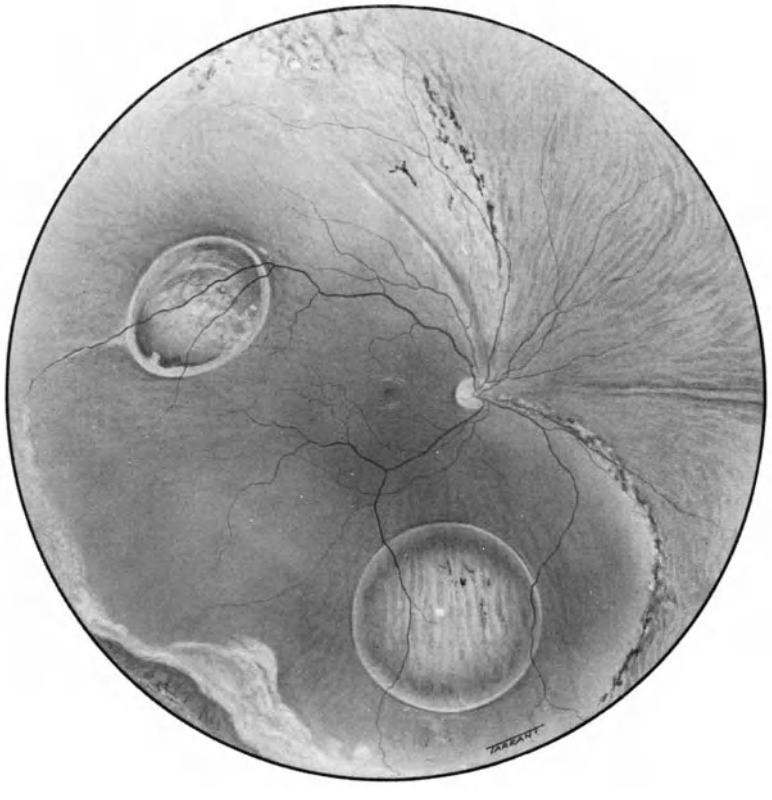

Fig. 2.4. A long-standing retinal detachment due to an inferior temporal dialysis has resulted in high watermark formation. Two large intraretinal cysts can also be seen

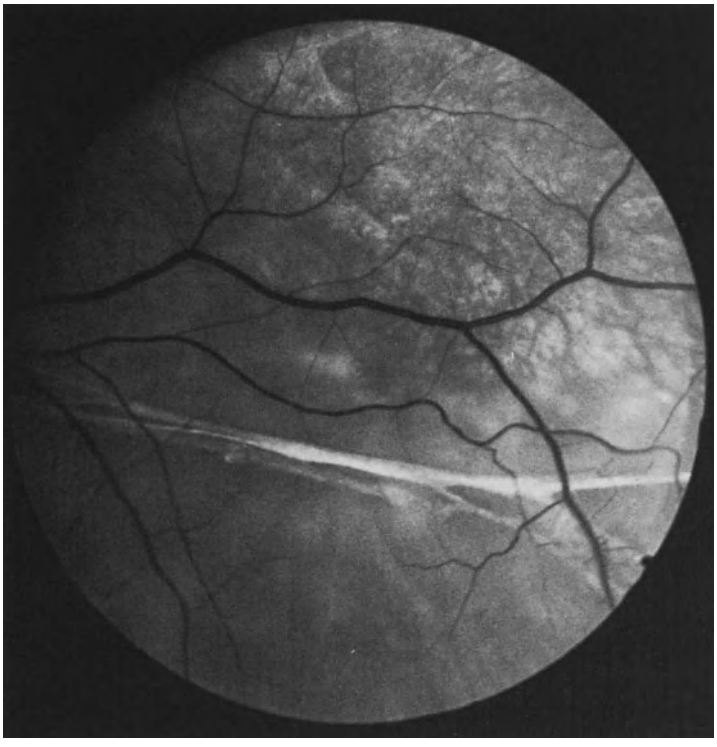

Fig. 2.5. White lines on the back of a detached retina in a case of long-standing detachment

to develop and although of striking ophthalmological appearance (Fig. 2.4), they do not pose any particular problems to the surgeon, disappearing spontaneously in the postoperative period when the retinal hole has been closed.

The other characteristic feature of long-standing retinal detachment is the appearance of black pigmented demarcating lines (high-water marks) which form in flat retina just in front of the advancing detachment. Several separate levels of water marks may be found if there has been a slow advance of the detachment edge. This finding shows that the demarcation lines themselves offer no complete protection against further separation of the retina, but they may contribute somewhat to slower progression. The degree of pigmentation of the lines varies and often there is an associated whitish fibrotic appearance.

In long-standing cases, an interlacing network of retroretinal white lines can often be seen (Fig. 2.5). These formations, while not progressive like preretinal membranes, can lead to considerable splinting and immobility of the detached retina in the vicinity of the retinal hole.

Previous Surgery

These cases are usually more difficult to examine and interpret. The vitreous may be hazier and scleral depression itself will often be less well tolerated,

particularly if the eye is still tender and photophobic from recent surgery. The previous surgery will have resulted in choroido-retinal scarring, making retinal holes more difficult to detect against the white background. The following points that relate to previous choroido-retinal scarring should be noted.

The Site

These areas should be carefully noted, particularly when diathermy has been used, which may have resulted in scleral weakness, warning the surgeon that the appropriate area must be approached with caution at operation.

Colour of Scar

If the choroido-retinal scar is completely white and atrophic, pigment epithelium and choroid will show no further reaction to cryotherapy at reoperation, and any reaction obtained will be seen only in any overlying detached retina.

Relationship to Detachment

The retina may be attached up to the edges of the choroido-retinal scar while the scar itself remains flat (occasionally the scar itself is the only flat part of a totally detached retina) or the retina may be detached overlying the choroido-retinal scar. The presence of choroido-retinal scarring with adhesion of overlying neuroepithelium may cause some obstruction to the natural spread of subretinal fluid, resulting in distortion of the retinal contours and estimation of the site of an undetected retinal hole less reliable.

Previous Buckles

Indentation from previous buckles will usually be obvious. If it is excessive, or the underlying sclera unusually weak, then there may have been erosion of the implant, particularly if the previous surgery took place several years before. The implant may be seen to have come to lie under the retina but occasionally it may have gained the vitreous cavity itself. If there is erosion, great care is necessary at the time of operation in approaching such an implant as the globe may be perforated.

Evidence of Previous Complications

Haemorrhages either vitreous or subretinal, will be obvious. In the subretinal space they have a somewhat blackish appearance because they are seen through the detached retina. Other complications arising from the drainage of subretinal fluid may also be seen. Incarceration of the retina results in a characteristic stellate puckering effect at the site of incarceration. If vitreous has also been incarcerated, traction lines are produced in the vitreous in a stellate-like manner.

Peripheral Retinal Degenerations

The presence of peripheral retinal degenerative change is important when flat retina is being examined, either in eye that contains a detachment (if flat retina is present) or in the other eye. The object of noting these degenerative changes is to detect those that will predispose to subsequent retinal detachment. Following these observations it then has to be decided whether such lesions require prophylactic treatment.

These lesions may belong to one of two main groups. The degeneration may be either benign, and thus, although requiring diagnosis, does not predispose the affected retina to retinal hole formation or detachment, or one that predisposes the retina to hole formation and the risk of detachment.

Benign Peripheral Lesions

All these lesions are found in the equatorial and pre-equatorial regions of the retina, extending forward in some cases to the ora serrata. In these conditions there is no associated overlying vitreous abnormality, which probably contributes to the benign way in which these lesions behave.

Choroido-retinal Degeneration

This type of degeneration is found in the retina immediately adjacent to the ora serrata and varies in severity according to its appearance. In its mild form, the only detectable change is a whitish grey appearance on the surface of the retina with some degree of mottling and accentuation of pigmentation. If slightly more marked, the peripheral retina assumes a more opalescent appearance with increase in the pigmentary disturbance. When the degenerative changes are even more pronounced, peripheral terminal arterioles are sometimes seen to be whitened in colour and there is quite marked hyperpigmentation of peripheral retina. If there is choroido-retinal degeneration in the equatorial region it takes the form either of a series of drusen-like bodies with hyperpigmented borders (cobblestone degeneration, Fig. 2.6c) or of a honeycombed area of pigmentation with the pigment tending to collect along the retinal blood vessels in a fine lace-like manner (Fig. 2.6a). The various types of choroido-retinal degeneration are harmless, widespread, and nonprogressive conditions found in large numbers of eyes (Okun, 1960).

Choroido-retinal Atrophy (Pavingstone Degeneration)

This type of degeneration (Fig. 2.6d) is found in the equatorial and pre-equatorial regions (O'Malley et al., 1965). It is characterised by the appearance of

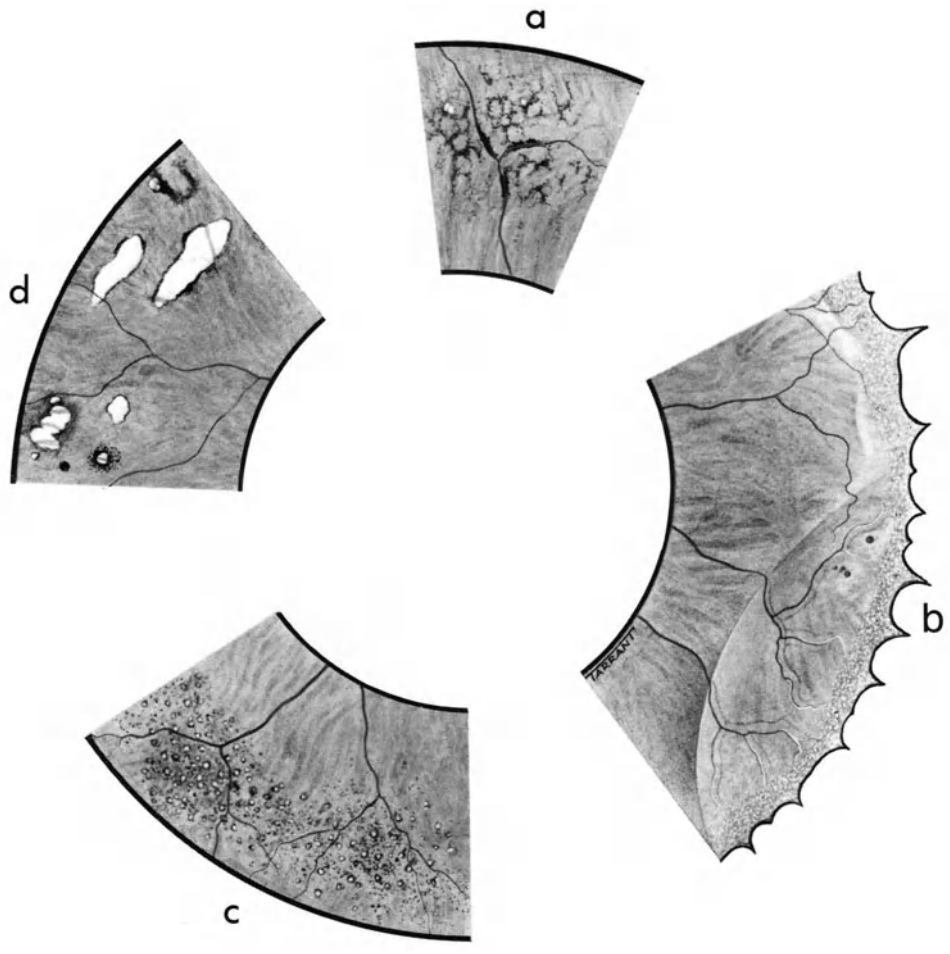

Fig. 2.6a–d. Peripheral retinal lesions; (a) non-specific pigmentary degeneration; (b) cystoid degeneration and retinoschisis; (c) cobblestone degeneration; (d) pavingstone degeneration

punched areas of retina with hyperpigmented borders. The lesions are usually linear in configuration, and the centre of the affected area varies in colour according to the degree of atrophy present. In extreme cases the whole choroid appears to be deficient, leaving only bare sclera visible at the bottom of the lesion, but in most cases larger choroidal blood vessels can be seen traversing the white area. The overlying retina, however, with the exception of the pigment epithelium, which is absent, is found to be normal. It is not unusual to find extensive confluent lesions which may, on occasions, extend through 360° of the peripheral retina, and which are usually bilateral; the lower half of the retina is somewhat favoured. There is usually normal retina between the lesions and the ora serrata, but the lesions, although in themslves harmless and not leading to retinal hole formation, may, if extensive, make detection of small holes more difficult if retinal detachment is present. Detached retina will always be adherent to the edge of the pavingstone areas.

Cystoid Degeneration

Cystoid degeneration begins in the outer molecular layer of the retina and results in cavities forming in the neuroepithelium. When these cavities coalesce and enlarge so that actual elevation of the retina can be observed, the term 'retinoschisis' is used (see Fig. 2.6b). In its mildest form, cystoid degeneration is found immediately posterior to the ora serrata, the lower temporal being the most favoured quadrant. Pink-red vesicles on a whitish grey background, give the retina an opalescent appearance, and on scleral depression the affected tissue assumes the appearance of fine frog-spawn. Occasionally the cystic cavities may rupture to produce small excavations similar to retinal holes. Cystoid degeneration is commonly found in eyes that show other forms of degenerative change; for example choroido-retinal degeneration or atrophy. If extensive, cystoid degeneration may extend posteriorly towards the equator, but not posterior to the equator itself.

Pigment Clumping

A localised area of pigment clumping in the peripheral fundus is frequently seen, the clumps usually being distributed between the ora serrata and equator. The clumps are of no particular significance and are more often found in myopia.

White With and Without Pressure

These appearances (Fig. 2.7a) are seen in flat retina with or without scleral depression respectively and it is likely that white without pressure is simply an exaggeration of white with pressure. The condition is to be distinguished from the normal blanching of the choroid that comes about with scleral depression and which, accordingly, moves as the scleral depressor alters its position. In both white with and without pressure, geographical areas which as a rule have a linear configuration are found on the extreme periphery of the fundus. On occasions, however, these areas may, remarkably, extend posteriorly as far as the equator and even beyond. The more linear edge of the area is on the ora serrata side of the lesion and the geographical shapes of the more central area vary considerably.

In most cases, when there is white with or without pressure the changes are extensive and the superior temporal quadrant is favoured. The exact significance of the findings have not been clearly established. Certainly, the appearance is rarely seen in normal eyes but it is often found in association with changes such as cystoid degeneration and lattice degeneration (Rutnin and Schepens, 1967). In both white with and without pressure, vitreo-retinal adhesions have been described (Watzke, 1961; Karlin and Curtin, 1973). Occasionally, the configuration of these lesions has been seen to change (Nagpal et al., 1976). These

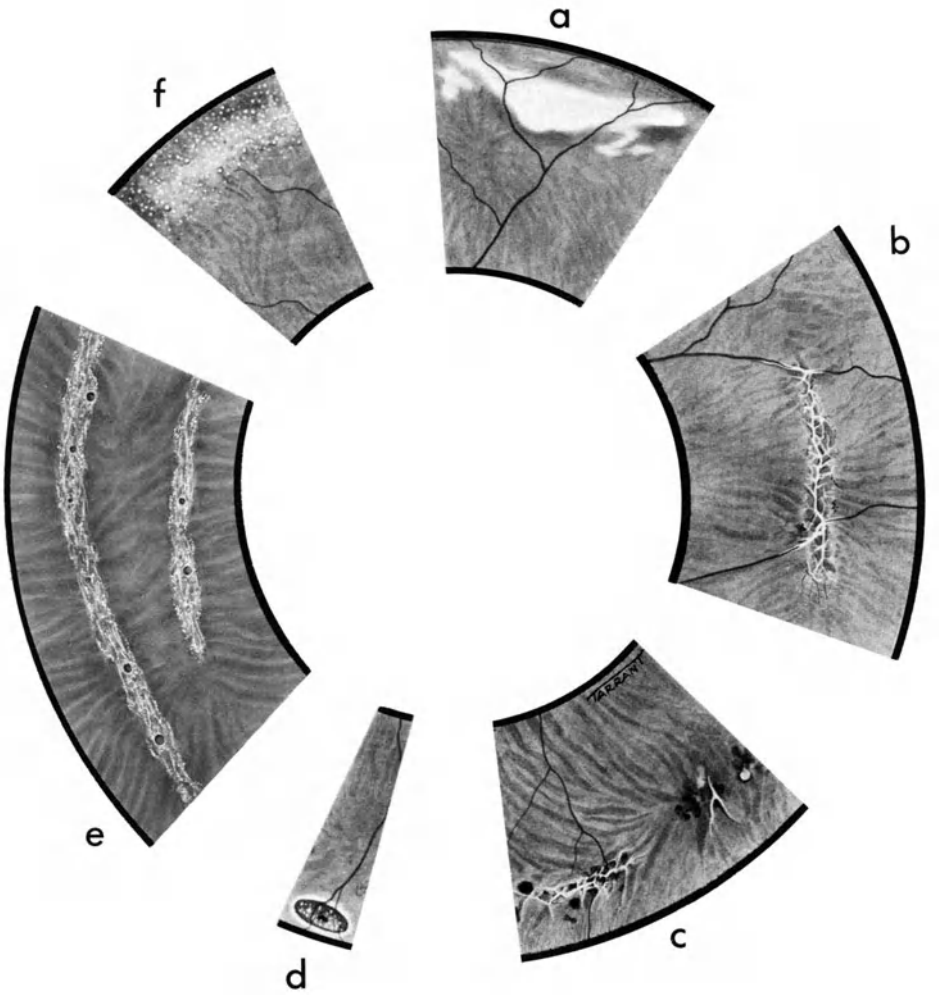

Fig. 2.7a–f. Peripheral retinal lesions; (a) white without pressure; (b, c) non-pigmented and pigmented lattice degeneration; (d) retinal erosion; (e) snail-track degeneration; (f) snowflake degeneration

areas, however, have not themselves been shown to progress to vitreo-retinal degeneration of a more serious nature or to hole formation and may therefore be regarded as ophthalmoscopic curiosities rather than areas of much significance.

Retinal Erosions

These oval-shaped excavated areas are situated with the vitreous base (Fig. 2.7d). The margins of such areas may appear to be slightly elevated and the base of the erosion cratered in appearance. Small whitish tags of vitreous, often exactly similar to those overlying areas of classical lattice degeneration, are

seen in most cases. There is no evidence that these areas are associated with retinal hole formation and retinal detachment, but they are often seen in eyes that contain lattice degeneration.

Lesions that may Predispose to Retinal Detachment

Lattice Degeneration

This degeneration has been described clinically and histopathologically since the earliest days of successful retinal detachment surgery (Gonin, 1935; Vogt, 1936; Meyer-Schwickerath, 1960; Okun, 1966; Schepens; 1952a, b; Allen and Straatsma, 1966; Straatsma et al., 1974). In its typical form, lattice degeneration consists of a sharply demarcated circumferentially orientated lesion located, usually, at the equator or in a pre-equatorial region (Fig. 2.7b, c)

The fine white lines of a lattice degenerative lesion are continuous with the retinal blood, vessels and, indeed, the blood vessels may on occasions be found emerging apparently quite normal in appearance on the anterior side of the lesion. Pathologically the fine white lines consist of hyalinisation of the retinal blood vessels; there is thinning of the underlying neuroepithelium although the choriocapillaris is itself relatively unaffected in this condition.

Vitreous abnormalities overlying the lesion are frequent. In parts the vitreous adheres firmly in a strand-like manner to the area of degeneration interspersed with areas of vitreous liquefaction.

A high incidence of lattice degeneration in autopsy eyes (6%; Straatsma and Allen, 1962) has been reported in otherwise normal fundi, indicating that most cases of lattice degeneration do not progress to retinal detachment, a risk that is increased if there is an appreciable amount of myopia (Morse, 1974b). It has been noted (Byer, 1974) that the progression of lattice-like lesions is extremely slow. The areas may be multiple and at different levels and the upper temporal quadrant is the most favoured. Bilateral lesions are found in about 50% of cases. There is a higher incidence of lattice degeneration in myopic eyes. Typically, the lesions may be found in the second decade. They evolve slowly, although the usual time of presentation is not until the fourth or fifth decade during an ophthalmic examination. Rapid progression and appearance of new lesions is rare but is occasionally seen (Lemcke and Pischel, 1966). Associated clinical appearances may be a retina with a thinned and shaggy appearance, yellow dots that may be found in the vicinity of the lattice lesions and a pigment epithelium that varies in response from complete absence of pigment to extensive clumping. This pigmentation, however, does not confer protection against subsequent retinal detachment (Morse and Eagle, 1975). Retinal holes with risk of detachment are the most significant additional clinical features of lattice degeneration. They may be

a) round, in which case they tend to favour the ends of the lattice lesion if it is a long lesion, and are found in among the fine white lines;

b) horseshoe-shaped tears which are the outcome of vitreous traction and the retina splitting on the posterior edge of the lattice lesion so that when the operculum is formed the lattice lesions are detected in the operculum itself;

c) giant tears which, exceptionally, result from the posterior limit of a long section of lattice degenerative change.

Snail-track Degeneration

This degeneration is so named because the lesions consist of bands of sharply demarcated areas resembling the trail made by a snail and has a glistening frost-like appearance (Fig. 2.7e). The degenerative areas are found in the equatorial region of the retina, and the superior temporal quadrant is preferred. There are overlying vitreous abnormalities consisting of liquefaction but marked vitreous traction is seldom if ever in evidence. These lesions have a pronounced predisposition to form retinal holes which are characteristic, rather large, round holes; horseshoe-shaped tears are never seen. Myopia is often associated with these lesions and there is a strong tendency to retinal detachment (Aaberg and Stevens, 1972). This condition bears some similarity to lattice degeneration and, indeed, it may be that it is just a variation of it; nevertheless, there are some striking differences. White lines are not seen in association with snail-track degeneration and the two types of degeneration are not found together. Although snail-track degeneration is considerably rarer than the lattice type, and most of the reported cases are small, eyes in which snail track degeneration features appear to have a considerably greater risk of retinal detachment than those with lattice degeneration.

Retinoschisis

Retinoschisis may be classified as senile, juvenile, or secondary.

Senile

This condition originates from peripheral cystoid degeneration of the retina (Fig. 2.7b). The cystoid cavities become confluent in the outer plexiform layer; the retinoschisis thus formed progresses by extending in a posterior direction through the layers of the neuroepithelium. The condition is usually bilateral, occurs more often in hypermetropic eyes, and the lower temporal quadrant of the eye is the most favoured (Straatsma and Foos, 1973). In its most obvious form the retinoschisis reveals itself as a smooth domelike elevation with an absence of retinal folding, but this cystic type appearance can be much less obvious. In some cases only one or two cyst-like elevations are seen and these are usually continuous with a less elevated zone extending around the periphery of the fundus and continuous with obvious peripheral cystoid degeneration.

Other clinical features of retinoschisis (Byer, 1968) include

1. White dots scattered on the inner surface of the schisis cavity, apparently lying at the level of the internal limiting membrane. These yellow-white spots are not in themselves specific and may be found in other conditions, for example lattice degeneration, or when the rest of the retina is apparently normal. They have also been described as part of a condition known as snowflake degeneration (Hirose et al., 1974), which consists of a vitreo-retinal dystrophy with an autosomal dominant inheritance pattern. In this condition, as well as snowflake yellow-white spots, there is extensive white with pressure, sheathing of terminal retinal vessels, with or without pigmentation, and eventual disappearance of peripheral vessels altogether. There is usually a nonspecific fibrillary vitreous degenerative change and a marked tendency towards rhegmatogenous retinal detachment.
2. The inner leaf of the schisis has a beaten metal appearance which is best seen on slit-lamp examination and retro-illumination.
3. There may be sheathing or obliteration of peripheral retinal vessels. The white lines found correspond to the retinal blood vessels and they are mainly on the venous side of the circulation.
4. Retinal holes are infrequent but may be found on the outer or inner leaf of the schisis and occasionally both. Holes in the inner leaf are always round, whereas those in the outer leaf, which are difficult to detect, particularly at their posterior borders, often have a scalloped appearance, the edges of which are rolled.
5. Pigment demarcation lines are rarely seen in the simple form of schisis unless retinal detachment intervenes.
6. Scleral depression over the outer leaf of the schisis will make the depressed area appear white, as it does in normal retina: a whiteness that is not seen in cases of retinal detachment.
7. If retinoschisis extends posterior to the equator, an absolute field defect corresponding to the area of the schisis is found.
8. Vitreous or retinal haemorrhage is rare.

Senile retinoschisis is either static or progresses very slowly (Byer, 1976) and cases in which the schisis has extended into the posterior pole are extremely rare.

The great majority of patients with retinoschisis have no symptoms and suffer no visual loss. The condition requires no treatment other than periodic observation. Retinoschisis may occasionally be complicated by retinal detachment. This problem will be considered later, under the differential diagnosis of retinal detachment.

Juvenile

In this condition the retina splits at the nerve fibre layer rather than at the outer plexiform layer as occurs in senile retinoschisis. The condition has been described by various authors (Sabates, 1962; Broderick and Wyatt, 1973)

and is rarely seen. It is inherited as a sex-linked recessive disease affecting males or, less commonly, as an autosomal recessive disorder. It is found in infants and children and together with Favre's disease (retinoschisis and tapeto-retinal dystrophy inherited as an autosomal recessive disorder) and Wagner's diseases (pigmentary retinopathy, cataract and vitreous degeneration inherited as an autosomal dominant) constitutes one of the vitreo-retinal dystrophies. In juvenile retinoschisis degenerative changes are found in both vitreous and retina and, as in Wagner's diseases, the condition may be complicated by retinal detachment.

The age of onset of this condition is usually less than 20 years and the retina has something of the appearance of a Swiss cheese. Early in the disease changes of retinoschisis are present at the macula as well, but they disappear in later life. Treatment of the congenital form of retinoschisis is conservative; the dangers of photocoagulation have been stressed (Brockhurst, 1970), as treatment may in fact precipitate retinal detachment. If retinal detachment supervenes in cases of congenital schisis the prognosis for surgery is poor.

Secondary

This condition is difficult to distinguish from traction retinal detachment and is found in conditions where there is, in any case, marked traction on the retina from vitreous strands, e.g. where retinitis proliferans exists. The features of this kind of schisis are:

1. Spread is slow with little evidence of progression.
2. Watermarks do not occur (unlike traction retinal detachments).
3. Tears may form along retinal blood vessels but the tears do not result in rhegmatogenous detachment. This again is a distinguishing feature between secondary schisis and traction retinal detachment.
4. The contour at the edge of the schisis, where it meets normal retina is convex towards the centre of the schisis.

No treatment is required for secondary retinoschisis.

Other Fundal Details

Disc

To be able actually to see the disc is important because, if it is hidden, due for example to opacities in the media or overhanging retinal detachment itself, the non-drainage operation may be impossible since it is essential to observe the patency of the arterial supply to the retina during this operation. The presence of glaucomatous cupping is also noted.

Pars Plana

Details of the pars plana can be detected with scleral indentation and clear media if good mydriasis has been achieved. This is very simple in cases of

aphakic detachment when the periphery of the crystalline lens does not interfere with the view. Harmless local anomalies such as small pars plana cysts are often found and occasionally these cysts may be very large (Ruiz, 1971). A thin white line running concentric to the ora serrata and indicating the anterior part of the vitreous base can usually be seen. Retinal detachment itself can extend into the pars plana, particularly in cases of aphakic detachment (Dobbie and Phillips, 1962). Occasionally, also, small slit-like holes may be detected in the pars plana in traumatic retinal detachment, but pars plana holes are exceptionally uncommon (Tasman, 1968) and are rarely a cause of failure in retinal detachment surgery.

Following the preliminary charting of the fundi of both eyes, the three-mirror examination (using 1% methyl cellulose) in the contact lens is now performed. This examination should not be carried out on the day of operation as prolonged examination may result in some loss of transparency of the cornea.

Three-Mirror Examination

Angle of Anterior Chamber

The width of the angle should be estimated. If the angle is shallow then there is a risk of angle closure in the postoperative period. Angle recession following trauma may indicate that the outflow from such an eye is impaired and softening, following the tightening of the buckle sutures as a result of aqueous outflow, may take somewhat longer than usual if a non-drainage operation is to be used. Multiple peripheral anterior synechiae, which may be found in aphakic detachment, may also warn against poor outflow. In the aphakic eye there may be adhesion of vitreous strands to the cataract section.

The Vitreous

Having assessed the angle, the next step is to examine the vitreous cavity. When a clinical examination of the vitreous is conducted it must be borne in mind that the observations of particular importance are those that will affect the choice of operation and to enable the observer to anticipate the response of the eye to the surgery that is to be performed. For this reason, examination of the vitreous may not greatly help in the choice of operation in uncomplicated cases, and drawings of the state of liquefaction of the vitreous and of detailed assessment of vitreous attachments will not be a profitable preoperative maneouvre. In complicated cases, however, and this almost invariably means the presence of intraocular fibrosis, the examination of the vitreous and its relationship to the retina, to appreciate the tractional forces at play, is an essential prerequisite for successful surgery (Hruby, 1960).

Degeneration

Degenerate changes in the vitreous body may be found to a lesser or greater degree. In its simplest form there may be liquefaction and cavitation of the vitreous body followed by posterior vitreous detachment, which usually results in a condensation of the collagenous frame-work so that the normal ribbon-like structure of the vitreous body is lost. In the early stages of degeneration, the vitreous body not only tends to detach from the underlying retina but usually assumes a more fibrillary structure and artificial liquid spaces within the gel are found (Lacunae). Occasionally, these spaces become very large, when it is then difficult to tell whether in fact the posterior hyaloid is detached from the retina itself or whether a thin layer of cortical gel remains to lie in contact with the surface of the retina.

These changes, although they may be induced by pathological states such as haemorrhage or infiltration with white cells, are often found in old age and myopia, and to a much greater extent in the aphakic than the phakic eye (Heller et al., 1972). However, a much more sinister degenerative change is the complete loss and collapse of normal vitreous structure with secondary immobility of the vitreous gel, which may be accompanied by the formation of fibrous-like membranes within the vitreous cavity, and by greatly increased pigment infiltration.

Vitreous Detachment

A total posterior vitreous detachment, when the posterior hyaloid face lies in the anterior third of the vitreous cavity, can be detected by simple slit-lamp examination of the anterior gel, whereas a partial posterior vitreous detachment, in which vitreous is still attached to the posterior pole of the eye and inferior half of the retina, and the posterior hyaloid lies in the posterior vitreous cavity, is best seen with the three-mirror contact lens in place. By studying a point on the posterior aspect of the vitreous gel (provided the latter has in fact become detached from the retina), the patient is asked to look first up then down and then back to the central straight ahead point. Upon such movement the gel will be set in motion and will flow up and down in a curtain like movement (the ascension phenomenon). When this movement is initiated, the separated posterior hyaloid membrane can be seen as the most posterior part of the moving gel. Behind the posterior hyaloid membrane is the optically empty retrohyaloid space. Holes in the posterior hyaloid can usually be detected where the hyaloid membrane has become detached from the optic disc. The detection of lacunae and the distinction between partial and total vitreous detachment is of no great importance and does not influence the type of procedure to be used.

An anterior detachment of the vitreous from the lens is rare and clinically is of no significance. Similarly, detachment of the vitreous base is also extremely unusual but occasionally, in traumatic cases, there is avulsion of the vitreous

base which, with adherent non-pigmented epithelium of the pars plana ciliaris, can be seen festooned in the vitreous cavity in a characteristic frill-like fashion (Cox et al., 1966).

Retinal Details and Vitreo-retinal Relationships

Retinal holes seen on indirect ophthalmoscopy and scleral depression will be confirmed with the three-mirror examination. Occasionally, previously undetected small holes will be found, the detection of which in cases for reoperation, or areas posterior to the reach of the scleral depressor is particularly difficult with indirect ophthalmoscopy. For such cases the three-mirror examination will prove of special value. To the inexperienced ophthalmoscopist the magnified view that the three-mirror examination allows will be particularly helpful, but as greater experience and expertise is attained with the indirect ophthalmoscope and scleral depressor, less dependence on the three-mirror examination will be needed to confirm the fundal findings.

After confirming the presence and site of the retinal holes the vitreous examination will proceed to consider the problem of retinal traction.

Traction on the Retina

Consideration of the type of traction present in any case of retinal detachment is important since it influences the type of buckling procedure to be used. The pull of vitreous on the retina (vitreo-retinal traction) may be exerted over a wide area, particularly at the posterior border of the vitreous base, where the separating posterior hyaloid remains firmly adherent (Fig. 2.2), or from narrow points of attachment. These, too, are found either at the posterior vitreous base or in isolated vitreous strands attached to the retina in more posterior positions. When a horseshoe tear is examined it will be seen that as the posterior hyaloid, which is attached to the operculum of the tear, is put in motion on ordinary movement of the eye, the operculum of the tear moves up and down considerably as the posterior hyaloid swings about its anchorage at the posterior vitreous base. This type of vitreo-retinal traction has been described as dynamic (Scott, 1971).

Vitreous traction is described as static when the pull on the retina by the vitreous is continuous. A simple example of this is when the posterior border of the vitreous base is seen to be tenting up peripheral detached retina in a linear fashion, a sharp white line marking its point of attachment. More advanced static traction may be seen, for example, following an intraocular foreign body, when fibrous strands traverse the vitreous cavity from its point of entry to the point of impaction on the contralateral retina.

The extreme example of static traction is when there is contraction of a thickened posterior hyaloid membrane dragging the retina into the centre of the eye to produce a picture of massive periretinal proliferation. In many cases

of advanced vitreo-retinal traction, the vitreous is tending to pull the retina towards the centre of the vitreous cavity. In other situations, however, fibrotic membranes growing on the front or the back of the detached retina produce a tractional force not directed towards the vitreous cavity, but one that is tangential to the surface of the retina. Thus, the directional pull of traction forces varies with the different clinical pictures encountered.

In advanced cases, static and dynamic traction will be present together. However, if there is static traction it is of much greater clinical significance than dynamic traction. When purely dynamic traction is present, permanent and longlasting relief from traction is not necessary (indeed, it has already been partially relieved when the hole is actually produced in the retina). Such cases are cured by local buckling and closure of the hole. Even if the buckle subsequently becomes reduced in height or, indeed, has to be removed, there is little risk of detachment from the original site if choroido-retinal adhesion is present. When there is advanced static retinal traction, however, higher and more permanent buckles have to be produced and, in extreme cases, conventional surgery may be hopeless.

The overall problems of traction lead to a consideration of the types of membranes within the eye.

Retinal and Vitreous Membranes

These membranes can be seen on indirect ophthalmoscopy and three-mirror examination. On examining a case of retinal detachment cellular membranes may sometimes be found on the inner and outer surfaces of detached retina and also within the vitreous cavity itself. Some degree of membrane formation has been found to occur in approximately 30% of a modern retinal detachment series (Chignell, 1977). This membrane formation is of great importance because the progressive fibrosis of retina and vitreous is the commonest cause of failure in retinal detachment surgery (Chignell et al., 1973). Membranes produce their most serious effects by progressive contraction once they are formed. The most potent cell responsible for the formation of these membranes is likely to be the pigment epithelial cell, the metaplastic activity of which can produce periretinal and vitreal membranes (Machemer and Laqua, 1975; Laqua and Machemer, 1976; Mueller-Jensen et al., 1975). The glial retinal cell is also capable of fibroblastic activity and is thus capable of contributing particularly to the formation of preretinal membranes (Gloor, 1969; Gloor and Daicker, 1975; Laqua and Machemer, 1975a). The actual stimulus to the production of these membranes is unknown but the detached retina itself is certainly one, possibly aided by the presence of blood and inflammatory cells in the vitreous cavity. Whether the invading cells are in fact glial or metaplastic pigment epithelium (as in the ordinary rhegmatogenous detachment), or adventitial cells (in, for example, detachments associated with retinal neovascularisation), or even choroidal and episcleral connective tissue cells (in detachments following perforating injuries) varies from one clinical situation to another (Constable, 1975).

Certain clinical features concerning the onset of membranes are well known to the retinal surgeon; for example, the longer the retinal detachment has existed the greater is the risk of membrane formation, a risk that is increased in certain types of retinal detachment, such as giant tears and aphakic detachments. Likewise, failed retinal detachment surgery will tend to produce new membranes or accelerate the progression of preoperative ones and this is more likely to occur if the retinal surgery has been complicated by haemorrhage or, say, retinal incarceration and vitreous loss. Excessive cryotherapy may be a contributory factor in stimulating membrane growth (Laqua and Machemer, 1976).

Types of Membrane

Preretinal

Preretinal membranes growing on the surface of the retina may be found in the vicinity of retinal tears or entirely separate from them. Once preretinal membranes have started to form the general tendency is for progression, although the progression occurs at a very variable rate. Early evidence of contracting preretinal membrane formation is the distortion of neighbouring retinal

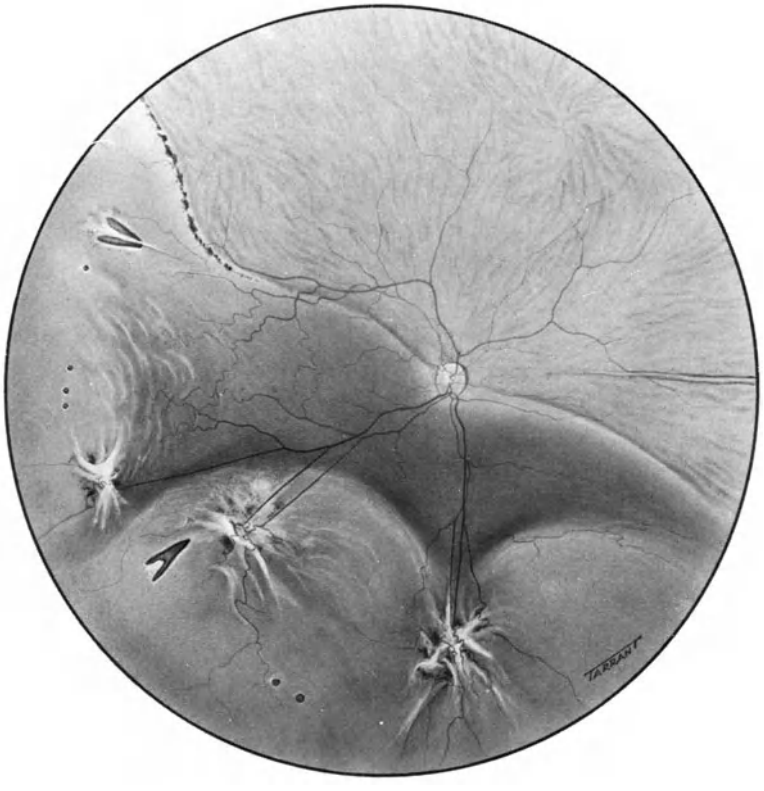

Fig. 2.8. A subtotal retinal detachment complicated by preretinal membrane formation. The membrane has resulted in star-shaped folds with secondary distortion of retinal blood vessels. Clumps of pigment are found near the folds. Vortex veins are seen in the upper retina

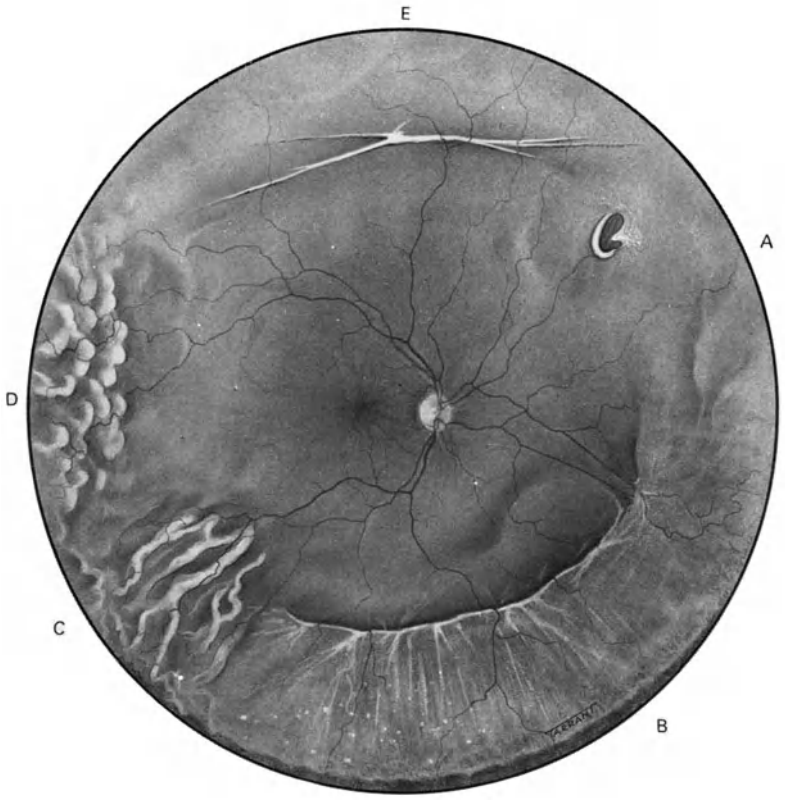

Fig. 2.9A–E. The effect on the retina of various types of periretinal membranes; (A) rolling of the posterior edge of a V-shaped tear due to preretinal membrane formation; (B) a circumferential equatorial fold due to vitreo-retinal traction. The retina is highly elevated, the retinal periphery is stretched, and the ora serrata dragged into view; (C, D) irregular folds produced by preretinal or subretinal membrane formation; (E) retroretinal strands found in long-standing detachment

holes, or blood vessels. This is best seen in the macular area where it is accompanied by loss of the normal reflex and wrinkling of the internal limiting membrane (cellophaning). A more peripheral example of early preretinal membrane formation is the backward rolling of the posterior edge of a horseshoe-shaped tear that may occur before the membrane can actually be seen (see Fig. 2.9A). The membranes progress and produce a whitish effect on the surface of the retina. Progressive contraction throws the underlying retina to which the membrane is attached into folds and these may be star-shaped or irregular in configuration (Fig. 2.8 and Fig. 2.9C, D). The membrane is initially semi-transparent and weak, involving only a superficial part of the detached retina, and the folds themselves are only of partial thickness; and there is very little evidence of increased pigmentation in the vicinity of these folds. However, as the preretinal membrane increases in density, full thickness folds which are, again, either star-shaped or irregular, may be produced, and these folds are usually associated with some degree of pigmentation. Clumps of pigment are found lying in close relationship to the folds within the retina itself, or imme-

diately overlying them in the vitreous cavity. With progressive fibrosis the retinal blood vessels become buried into the fold, the whole fibrovascular complex having a knotted appearance. Star-shaped folds arise as a result of changes occurring at a focus on the retina whereas irregular membranes arise as a result of the process being somewhat more diffuse. It is not known why star-shaped folds tend to be produced in one type of detachment while irregular folds are found in another.

Subretinal

a) Yellowish white retinal strands may be found, particularly in association with long-standing retinal detachments. They are often widely distributed in a strut-like configuration on the posterior retinal surface and have numerous interconnecting branches (Fig. 2.9E). These structures, thought to be of glial cell origin, may cause immobility of the retina but are otherwise notably benign in their behaviour, are not associated with full thickness retinal fibrosis, and have little tendency to progress.

Yellow-white dots representing depigmented pigment epithelial macrophages can be found in clusters on the posterior surface of a long-standing detached retina. These, too, are of little clinical significance.

b) Subretinal membranes growing on the back of the detached retina may contribute to fixed folding of the retina, either in the form of star-shaped or irregular folds. The folds themselves then tend to obscure the subretinal proliferation, which becomes difficult to detect, particularly as there are almost always associated preretinal membranes.

Intravitreal Membrane Formation

Membranes within the vitreous cavity are found before retinal detachment and contribute to it, or appear after detachment has occurred, when they are then part of the periretinal proliferative response to the detachment. In the latter case, while helping to perpetuate the detachment and frustrate surgical attempts at reattachment, they are not responsible for its production.

Sometimes found are isolated strands of fibrous tissue. These bands may be vitreo-retinal, e.g. in retinitis proliferans, a contracting posterior hyaloid pulling on the fibrous bands which results in traction detachment at the point of attachment of the band to the retina (Fig. 2.10B), or they may be transvitreal, e.g. resulting from the entry or the removal of an intraocular foreign body (Fig. 2.10A). Perforation of the globe may, in addition to transvitreal strands, have produced incarceration of pars plana or retina with basal gel at the site of entry. This incarceration with the retina, and vitreous dragged into the perforation site may result in fibrosis radiating into the vitreous cavity and along the surface of adjacent retina which may or may not be detached at this site. Traction bands may not only result in traction detachment of the retina but

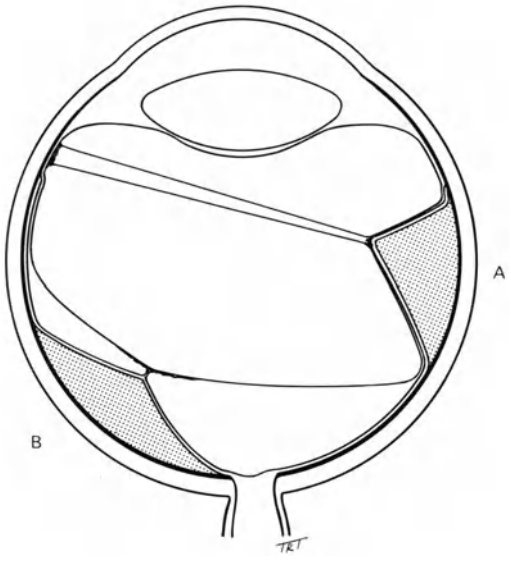

Fig. 2.10. (A) transvitreal bands from an intraocular foreign body; (B) vitreoretinal traction from retinitis proliferans. In both conditions traction detachment may occur

may, in turn, also cause retinal hole formation, resulting in an ordinary rhegmatogenous element to the detachment.

In ordinary primary rhegmatogenous retinal detachment, membranes within the vitreous cavity are usually found in association with membrane formation on the surfaces of the retina. In this case, pigment clumps are often on both sides of the retina and also scattered in clumps or strands throughout the vitreous body, which itself becomes progressively more collapsed and immobile. Membranes within the vitreous cavity often have no exact configuration, particularly when fibrosis is extensive. However, fibrosis within the vitreous cavity will contribute to folding of the detached retina. In general, two main types of retinal fold have been described as a consequence of these membranes.

The circumferential fold is produced at the level of the pre-equatorial region of the retina and is caused by the contraction of a membrane traversing the anterior part of the vitreous cavity, approximately at the level where a posterior hyaloid face is found when there is total posterior vitreous detachment. This membrane is usually fairly transparent and may have associated clumps of pigment in it. When this fold is found, the retina is usually highly elevated and drawn towards the centre of the vitreous cavity, a change representing part of the process of massive periretinal proliferation (Fig. 2.11). The circumferential fold, however, is not necessarily complete when seen earlier in its evolution (see Fig. 2.9 B). In these cases an equatorial retinal fold corresponding to only part of the total circumference is found. The traction on the fold drags the peripheral retina in a central direction, giving it a stretched appearance, and often the ora serrata is pulled into view. The retina is usually totally detached, but not necessarily bullous, and there is usually associated retinal fibrosis, with the eye on the verge of passing into the end stages of massive periretinal proliferation.

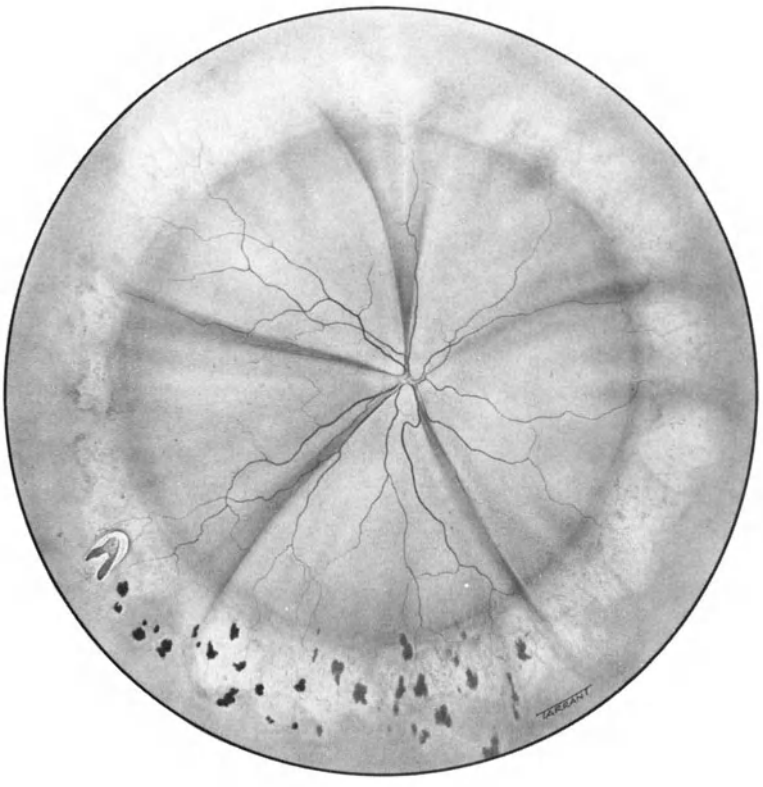

Fig. 2.11. Massive periretinal proliferation. The totally detached retina is drawn forward into the centre of the eye although the disc is still visible. A previous encircling procedure has failed; there is marked pigment clumping

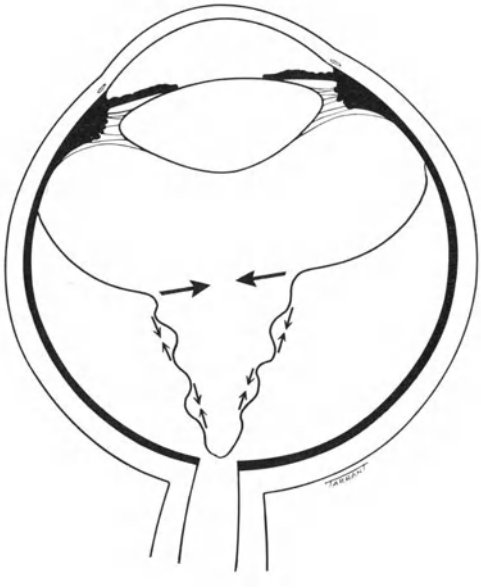

Fig. 2.12. Advanced periretinal membrane formation. Contraction of transvitreal membrane initially causes circumferential folding of the retina and eventually draws the retina into the centre of the eye. Irregular folds are found in the posterior retina

Irregular folds located in the posterior vitreous cavity take the form of multiple folds of detached retina as a result of interconnecting bridges of semi-opaque vitreous tissue and are often associated with pigmentary clumping (Fig. 2.12).

Massive Periretinal Proliferation (MPP)

This term (synonymous with the older terms of massive vitreous retraction or massive preretinal retraction) has been given to the clinical appearance of the extreme form of retinal and vitreous fibrous proliferation (see Fig. 2.11). Progressive contraction of transvitreal membrane results in accentuation of the circumferential equatorial retinal fold, the retina being pulled into the centre of the eye (see Fig. 2.11). Contraction of dense periretinal fibrotic tissue may result in shortening of the detached retina, an event that further reduces the chance of reattachment. It may be possible to see the disc through the narrow channel formed by the close apposition of the bullae, but in extreme cases, the disc is obscured by fibrous tissue. The vitreous body is collapsed and immobile, although total posterior vitreous detachment may not have occurred and the gel occupies the anterior vitreous cavity. The gel is diffusely infiltrated by pigment strands and membranes.

The clinical importance of fibrous membranes in relationship to the retina and vitreous obviously varies according to the extent and severity of the process but the practical clinical importance of these membranes may be summarised as follows.

Local Retinal Effects

Distortion of retinal features. Retinal blood vessels may be considerably distorted by the fibrotic process. Tortuosity of vessels is found in the immediate vicinity of the fibrotic area and traction on the vessel will result in straightening of the vessel central to the membrane. Distortion of retinal holes also results in elongation of such holes which makes for more difficult localisation, and closure by the buckle at the time of surgery.

Infiltration of the detached retina with membranes results in immobility of detached retina. Of particular interest is the degree of mobility in the vicinity of the holes as this will influence the decision concerning the drainage of subretinal fluid.

Retinal folds are produced, often making detection and closure of holes more difficult.

In very severe cases *retinal shortening* occurs.

Disturbance of vision. If membranes occur at the macula, then, although this will have little obvious affect on central vision, which will be depressed if the macula is detached, little return of central vision can be expected even if there is successful replacement of the retina.

Progression of Membranes

The rate of progression of membranes is extremely variable. In some cases, for example the retroretinal strands found in long-standing detachments, progression appears to be extremely slow or even static. These cases appear to stay the same for many years and do not usually progress to end-stage massive periretinal proliferation. In other cases, particularly when there is preretinal membrane formation, remorseless progression over weeks and months is the general rule, partial and then full thickness star and irregular shaped folds are formed, and the eye progresses to total detachment and massive periretinal proliferation. It has been pointed out (Morse, 1974a) that when fixed star folds are present in a retinal detachment a high proportion of these cases (23.8%) will progress to MPP, a figure that is not apparently influenced by the kind of surgery used. Occasionally, progression is extremely rapid, for example in cases of giant tear. The response to surgery of these membranes (see chapter 5) is also variable. Operation has an excellent chance of halting the progression of the less severe membrane formations by closing the holes and flattening the retina. This will not be possible if the membrane formation is too extensive, indeed surgery, particularly if complications occur, may accelerate membrane progression.

Characteristics of Types of Retinal Detachment

Aphakic Detachment

Retinal detachments are estimated to occur in 2% of patients who are aphakic (Scheie et al., 1973). The majority occur within the first year of cataract surgery, and within that year itself the first three months is the most vulnerable time. The risk of detachment is greatly increased in eyes that were previously myopic (Hyams et al., 1975) and this risk appears to increase with severity of the pre-existing myopia (Ruben and Rajpurohit, 1976). Other groups that appear to be particularly at risk are those that have had pre-existing lattice degeneration (Morse, 1974b) and also those in which the other eye has suffered aphakic retinal detachment. The incidence of aphakic retinal detachments in different retinal detachment series has varied from approximately 10 to 30%. This wide difference probably reflects differences in needs and attitudes as to when cataract surgery should be performed. The incidence of retinal detachment after phako emulsification appears slightly lower than that found by conventional intracapsular methods (Shafer, 1974). Aphakic detachments have a variety of features that tend to distinguish them from their phakic counterparts.

U-shaped tears are much less commonly found in aphakic detachment than in their phakic counterparts. The commonly encountered hole is the small, round hole situated in the immediate post-oral vicinity of the retina (Fig. 2.13)

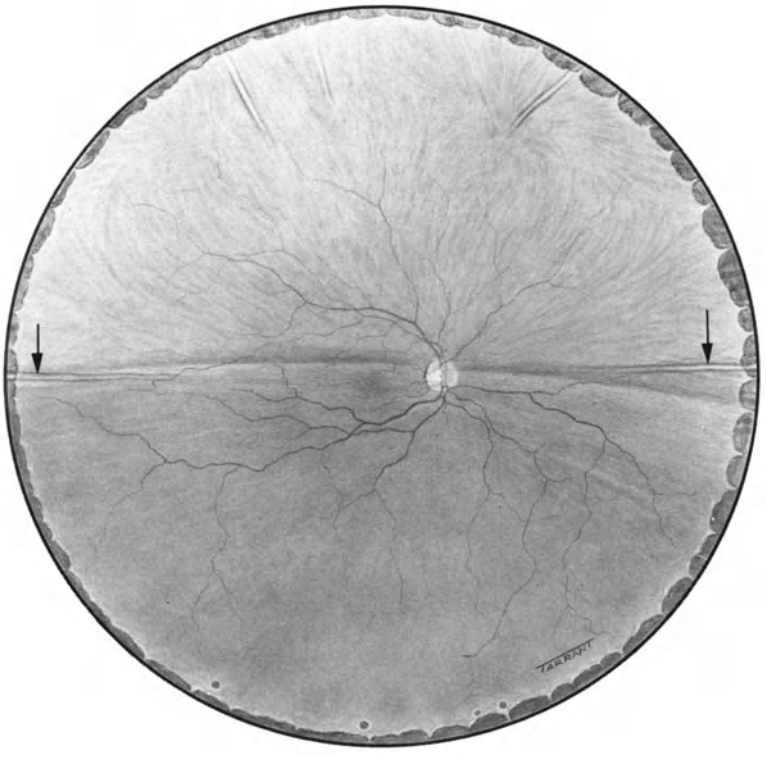

Fig. 2.13. Aphakic retinal detachment associated with multiple small post-oral holes. The long ciliary bundles are seen in the horizontal meridians (*arrows*)

and such holes may be multiple (Phillips, 1963). They are difficult to find, a difficulty that may be compounded by poor pupillary dilatation (exacerbated by the reluctance of cataract surgeons to perform broad iridectomies in high-risk retinal detachment situations), by capsular lenticular remnants, and by opacities of the vitreous body. If cataract surgery has been recent and complicated the eye will be red and irritable, increasing the difficulty of the preoperative examination. It was found (Norton, 1964) that there was no particular difference of hole distribution in the various quadrants in aphakic cases and, no doubt due to the small size of the holes involved, it was felt that failure to find a hole in the aphakic group was of much less significance than in the phakic counterpart.

There is a greater tendency in the aphakic group for fibrous proliferation resulting in progression to fixed retinal folds and end-stage massive periretinal proliferation (Ashrafzadeh et al., 1973). This picture is likely to occur where the cataract surgery has been complicated by poorly managed vitreous loss with incarceration of vitreous into the cataract section, or extensive haemorrhage.

There is a higher incidence of inferior detachment in aphakic cases and there is also a greater tendency for the detachment to become total (Norton, 1964).

In long-standing aphakic detachments there is a reduced tendency to high-watermark formation even though the incidence of intraretinal cyst formation is unaltered. The reduction of high-watermark formation indicates a reluctance of the pigment epithelium to respond to the presence of a subretinal fluid.

The ability of the retina to detach when these are small round holes, the absence of high watermarks, and the tendency to rapid progression to total detachment has been interpreted as being indicative of poor adhesion between the receptor layer of the retina and pigment epithelium in aphakic cases, allowing an easy stripping off of the receptor layer when detachment begins (Ashrafzadeh et al., 1973). This weakness may have been induced by the actual removal of the cataract itself, possibly by disruption of the zonular fibres and their attachment to peripheral retina.

In some series (Chignell et al., 1973) aphakic retinal detachments have fared less well than their phakic counterparts in the response to retinal surgery. This has mainly been due to difficulty in detection of the retinal holes and, also, by the tendency to fibrous proliferation. If surgery is successful, however, the final visual acuity obtained is not significantly different from that obtained from the surgery of phakic retinal detachments (Norton, 1964).

Retinal Dialysis

Retinal dialysis is the term applied to retinal holes at the ora serrata. The commonest site for the presence of a retinal dialysis is the lower temporal quadrant. In some series (Hagler and North, 1968) the dialysis was found in the upper nasal quadrant in a surprisingly high proportion of cases, but this was not confirmed in other series (Chignell, 1973). The reason why the lower temporal quadrant of the retina is the most vulnerable to this form of detachment has never been fully explained; trauma may play a part since the temporal quadrant of the globe is less protected. A stretching force on the vitreous base as a result of injury has been shown experimentally (Cox et al., 1976). However, the relationship of trauma and dialysis is not fully understood (Scott, 1977) and in many cases it is not possible to elicit the history of trauma. Indeed, in approximately 5% of cases the condition is found to be bilateral, i.e. the non-traumatised eye may contain a dialysis. Even in cases where dialysis is not in fact bilateral the other eye often has cystoid degenerative changes in the post-oral region of the retina. It is probable that while trauma may play a part in the production of some cases of retinal dialysis, there is an underlying weakness in the region of the ora serrata. In most cases arising inferiorly, progression is insidious so that in the majority (85%; Chignell, 1973) the macula is detached at presentation. A field defect is occasionally noticed, but other than that the detachments are usually asymptomatic, presenting only when reduced visual acuity is noticed. There is seldom any refractive error and, unlike other types of detachments, myopia is not a striking feature, occurring in only about 6% of cases. Men heavily outnumber women, and a much

younger age group is encountered in this type of detachment, being far the commonest cause of detachment in children and young adults. Approximately half of the cases arise in patients under the age of 20.

In the majority, the vitreous base remains intact and its posterior limit is firmly adherent to the edge of the dialysis, an attachment that results in splinting and some degree of immobility of the dialysis. Posterior vitreous detachment is rare in these cases. The presence of dialysis is often signposted by characteristic whitish opacities in the vitreous overlying its attachment to the dialysis edge, and pigment particles are also found at the point of attachment of the vitreous base to the dialysis. When the retinal dialysis is fresh, the actual dehiscence may be slit-like and it is difficult to detect in the peripheral retina, a difficulty that underlines the need for careful scleral depression which itself must be gentle in these cases because firm pressure will close the dialysis and render it invisible to the casual observer. Multiple dialyses are sometimes found and these are generally situated in the same quadrant of the retina separated by small bridges of intact retinal tissue; it is rare for separate dialyses to be widely apart. Retinal dialyses extending for more than a quadrant or so are classified as giant dialyses and are exceptionally rare. With the passage of time, particularly if the dialysis is large, the posterior edge becomes retracted and gives the dialysis its characteristic semilunar appearance. Demarcation lines appear and occasionally secondary intraretinal cysts may be found (Hagler and North, 1967).

It is most unusual for the detachment associated with inferior retinal dialysis to become total unless complicated by massive intraocular fibrosis.

The usually emmetropic eye with its more normal vitreous, and only a slight tendency to marked intraocular fibrosis, contributes to an exceptionally good prognosis for anatomical reattachment of the retina (Blach and Davies, 1967). Although such cases do extremely well anatomically, with at least return of peripheral vision, recovery of central vision is usually poor due to the long-standing detachment of the macula.

Children

Trauma, either penetrative or contusive is a more important factor in the production of retinal detachment in children than in adult cases (Daniel et al., 1974), the commonest individual type of case being the retinal dialysis. The retinopathy of prematurity that produces temporal traction may also lead to detachment (Tasman, 1967). Rare congenital anomalies such as posterior hyperplastic primary vitreous (Pruett and Schepens, 1970) and optic disc abnormalities (Hamada and Ellsworth, 1971) may be associated with retinal detachment in very young children.

Giant Retinal Tears

Giant retinal tears are defined as those that are 90° or more of the retinal circumference of the eye. These cases usually pose difficult management prob-

lems to the retinal surgeon. The average age in this group of patients is younger than that usually encountered in the detachment population and males occur much more frequently. Although some authors (Schepens and Freeman, 1967) have shown that the incidence of myopia in this group is no higher than in the normal detachment patient, others (e.g. Norton et al., 1969) have found there was a shift towards myopia. The retinal tears themselves arise from the pre-equatorial region of the retina and in most cases there is no detectable underlying weakness of the retina from which they arise (Schepens and Freeman, 1967). However, lattice degeneration is occasionally found, as also are areas of white without pressure. Such areas are frequently found in the other eye as well. Giant tears have sometimes been seen in association with colobomata of the lens (Hovland et al., 1968). Trauma may be a precipitating factor. The incidence of bilaterality in the non-traumatic case is high (about 40%) but an exceptionally high incidence of 75% was found in one series (Scott, 1976). The incidence is important when prophylaxis is concerned. A complete posterior vitreous detachment is unusual but there is a strong attachment of the vitreous to the rigid anterior edge of the tear which is often considerably elevated into the vitreous cavity. The posterior flap of the giant tear tends to hinge on

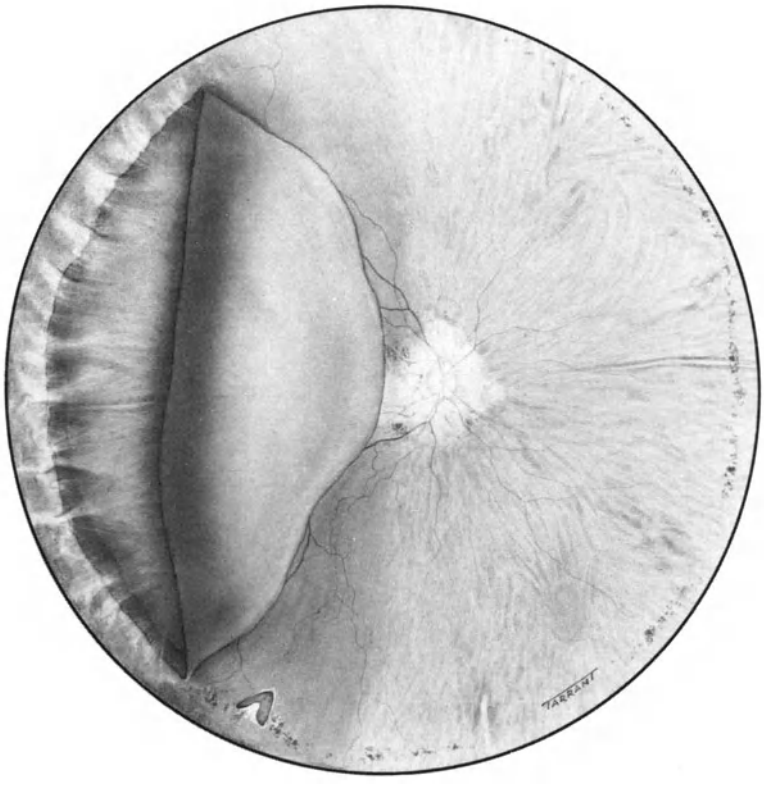

Fig. 2.14. A giant tear of the temporal retina arising in a highly myopic eye. The retina has flapped over but has not become fixed. The anterior part of the tear is elevated and immobilised by vitreous traction. There is a small associated V-shaped tear

a line joining the two extremities of the tear and the amount of movement of this flap is variable. Although sometimes the flap will stay almost in situ when the tear is approximately 90° in extent, in larger tears there is a tendency for it to hinge right over so that the back of the neuroepithelial layer is viewed on ophthalmoscopy (Fig. 2.14). The movement of the posterior flap may produce the unusual symptoms of a dense, moving curtain passing in front of the eye as it flaps over the macula. As time progresses the posterior movement of the flap will be reduced as it becomes immobilised due to the formation and contraction of surface preretinal membrane. The flap will start to roll up and, also, become fixed in a retroverted position where it will eventually be incorporated into dense fibrous preretinal tissue. Horseshoe-shaped tears are often found in association with giant tears and are invariably situated at the same circumferential level of the retina. The tendency of giant tears to progress rapidly to massive periretinal proliferation is one of the reasons why these types of cases fare so badly.

Macular Holes

Macular holes resulting in retinal detachment are rare and were estimated to occur in approximately 1% of retinal detachment cases in one series (Margherio

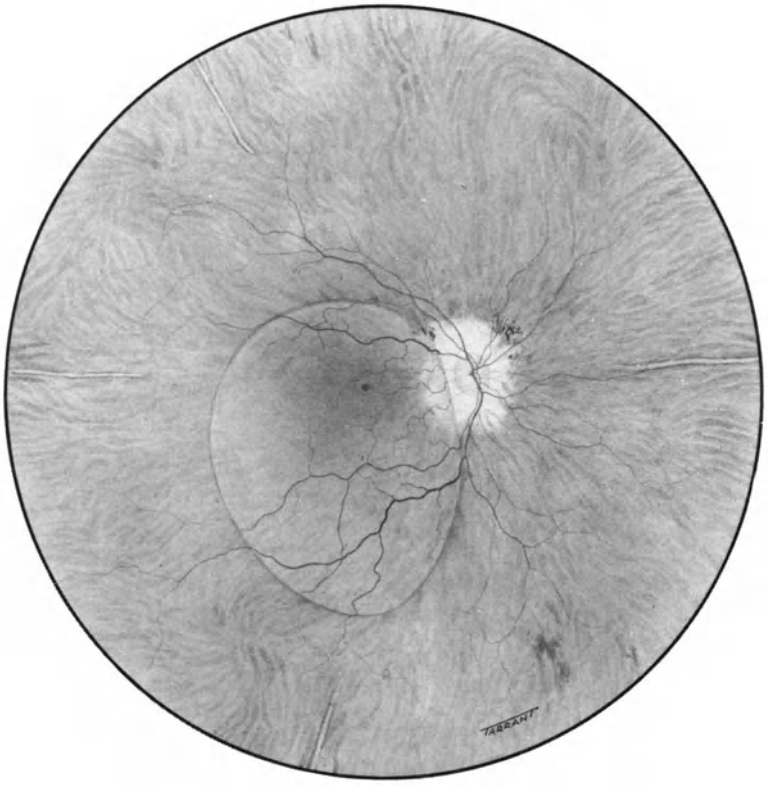

Fig. 2.15. Detachment arising from a macular hole in a highly myopic eye

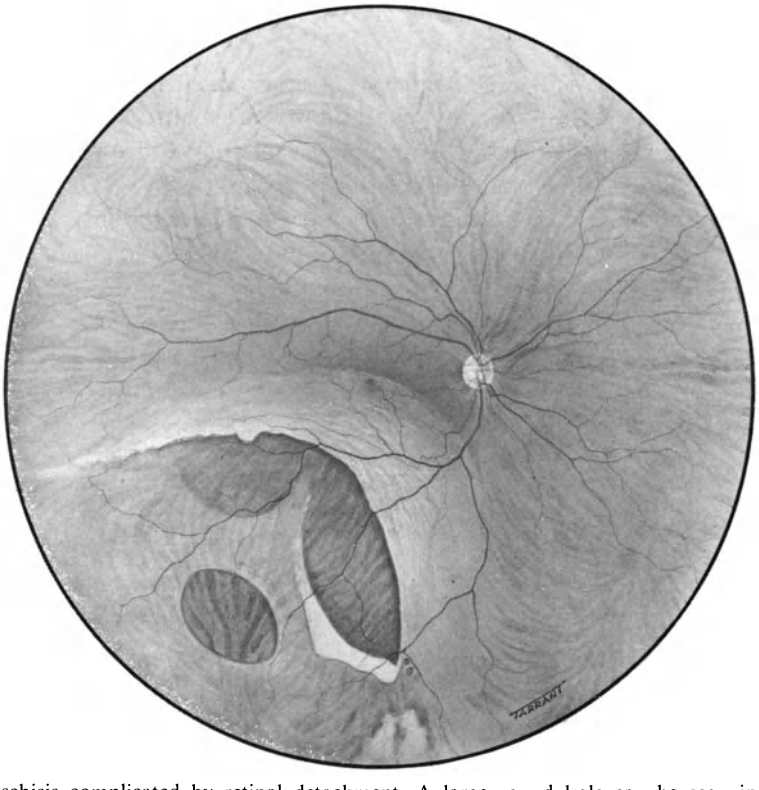

Fig. 2.16. Retinoschisis complicated by retinal detachment. A large round hole can be seen in the inner leaf of the schisis and an even bigger kidney-shaped hole with a rolled anterior edge in the outer leaf

and Schepens, 1972). These cases represent particular problems in management and treatment as do other holes that are extremely posteriorly situated in the fundus (Regenbogen and Stein, 1968). Macular holes associated with retinal detachment usually arise in high myopia with associated posterior staphyloma (Fig. 2.15). Rather more rarely they are associated with emmetropia or following trauma, but senile macular holes arising as a degenerative condition rarely progress to retinal detachment (Aaberg et al., 1970). Macular holes are round or somewhat oval in shape and vitreous traction is seldom found to be an associated feature although it has been considered by some to be important (Margherio and Schepens, 1972). Horseshoe-shaped tears are exceptionally rare in the macular region but have been found occasionally (Jungschaffer, 1971). Detachments with a macular hole may present in the following ways:

1. A localised retinal detachment confined to the posterior pole;
2. A retinal detachment with a macular hole that has spread to the periphery but in which no peripheral hole can be found;
3. A retinal detachment with detectable peripheral holes and also a macular hole.

The management of patients with macular holes associated with retinal detachment is discussed in chapter 3.

Retinoschisis Complicated by Detachment

These cases are rare (an incidence of approximately 1% of detachment cases) and it may be difficult to tell the difference between a long-standing retinal detachment and that of a detachment superimposed on a pre-existing schisis, the diagnosis being established with certainty only when holes in the outer leaf of the schisis are detected. These holes are usually large and have an oval or kidney-shaped appearance with a rolled ill-defined posterior border (Fig. 2.16). Inner leaf holes are found only in approximately 40% of cases (Hagler and Woldoff, 1973). The combination of retinal detachment and retinoschisis makes it difficult to determine the exact extent of the pre-existing retinoschisis, an extent that may be apparent only in the postoperative period, either by the visual field recovery or by residual retinoschisis elevation. In freshly detached cases the appearance of the retina varies from place to place. Thus, in one area the typical change of retinoschisis predominates, and arising from one border of the schisis – usually its posterior aspect – the usual appearance of retinal detachment is seen. Signs of retinoschisis are usually found in the other eye.

Differential Diagnosis of a Rhegmatogenous Retinal Detachment

There is usually no difficulty in diagnosing rhegmatogenous retinal detachment with its typical appearance of undulating folds of the retina, loss of the normal underlying choroidal reflex, and increase in darkening of the retinal vessels. The detection of full thickness retinal holes, although very rarely found in malignant melanoma (Bedford and Chignell, 1970) virtually eliminates non-rhegmatogenous detachments of one sort or another. The main difficulty in diagnosis arises when it is not possible to find retinal holes, in which event difficulty may be experienced in distinguishing retinoschisis from long-standing retinal detachment, and rhegmatogenous from non-rhegmatogenous detachments.

Retinoschisis and Retinal Detachment

The importance of making this distinction, which can be difficult when detachment is long standing and the retina is thinner and straighter, lies in the difference of approach to the management of the problem. In retinoschisis the management is conservative and operation rarely indicated. The main practical points of distinction are: (a) in retinoschisis, demarcation lines and secondary intraretinal

cysts are not found; (b) scleral depression over the outer layer of the retinoschisis will produce a blanching effect, which is not seen in retinal detachment; (c) retinoschisis tends to be a bilateral condition and is usually asymptomatic with no premonitory signs or loss of vision.

Retinoschisis complicated by retinal detachment as opposed to long-standing retinal detachment may be hard to distinguish, but the decision is less important as in either case operative treatment will be advised.

Exudative Retinal Detachment

In most cases (Kanter and Goldberg, 1974) very little difficulty exists in distinguishing exudative from rhegmatogenous detachments. In some cases it is associated with extraocular disease (e.g. hypertension) in which event a general examination of the patient will reveal the underlying problem and the ocular examination will itself show retinopathy associated with symmetrical, shifting, bilateral inferior retinal detachments, in which retinal holes cannot be found. If the exudative detachment is secondary to an intraocular disease, then indirect ophthalmoscopy aided by transillumination will nearly always reveal the true nature of the detachment, e.g. the exudative detachment associated with choroidal malignant melanoma, Leber's miliary aneurysms, or Von Hippel's retinal angiomatosis. Occasionally, however, considerable diagnostic difficulty may be encountered, particularly in the following group of diseases.

Harada's Disease and Similar Conditions

Vogt-Koyanagi-Harada's disease, originally described as two separate conditions (Bruno and McPherson, 1949; Cowper, 1951), has gradually become recognised as a single rather ill-defined entity of inflammation of retina and choroid, producing exudative retinal detachment, with three recognisable phases.

1. A meningo-encephalitic phase characterised by severe headache, meningeal symptoms and pleocytosis of the cerebrospinal fluid.

2. An ocular phase manifested by bilateral uveitis and non-rhegmatogenous retinal detachment.

3. The appearance of cutaneous and auditory symptoms, the cutaneous symptoms consisting of vitiligo, alopecia and poliosis and the auditory ones of dysacousia and deafness.

Considerable diversity in the clinical appearance of these diseases and of their intensities exists from one patient to another and while the onset is usually dramatic, the subsequent course is extremely slow with a tendency to spontaneous remission within about a year. The disease has a tendency to affect middle-aged people of dark pigmentation, and while the ocular signs are usually bilateral, one eye is often affected at a different time from the other. In this condition, fluorescein angiography may be of diagnostic help showing early masking and late leakage from pigment epithelium.

In general, any inferior retinal detachment, in which a retinal hole has not been found and in which the subretinal fluid is found to shift markedly, and in which there is no other obvious intraocular cause of such a detachment (e.g. tumour) should arouse a high index of suspicion that a Harada-like condition is present. Signs of uveitis in the same or the other eye must be sought, and fluorescein angiography performed to help establish the diagnosis.

Uveal Effusion

The term 'uveal effusion' was based on a description of cases (Schepens and Brockhurst, 1963) the cause of which is unknown and which have the following clinical features:

1. A tendency to affect middle-aged men;
2. The clinical appearance of annular choroidal detachment, usually bilateral and involving the whole peripheral fundus;
3. Non-rhegmatogenous retinal detachment, usually bilateral with shifting subretinal fluid;
4. Minimal or absence of signs of uveitis.

In some cases there was an associated elevation of cerebrospinal fluid pressure with an increase in protein content. The course of the disease was usually progressive and, again, one eye was affected at a different time from the other. These cases responded badly to any attempted retinal surgery.

References

Aaberg, T.M., Stevens, T.R.: Snail track degeneration of the retina. Am. J. Ophthalmol. *73*, 370–376 (1972)

Aaberg, T.M., Blair, C.J., Gass, J.D.M.: Macular holes. Am. J. Ophthalmol. *69*, 555–562 (1970)

Allen, R.A., Straatsma, B.R.: The pathology of lattice degeneration of the retina. Mod. Probl. Ophthalmol. *4*, 49–66 (1966)

Ashrafzadeh, M.T., Schepens, C.L., Elzeneiny, I.I., Moura, R., Morse, P.H., Kraushar, M.F.: Aphakic and phakic retinal detachment. Arch. Ophthalmol. *89*, 476–483 (1973)

Bedford, M.A., Chignell, A.H.: U-shaped retinal tear associated with a presumed malignant melanoma of the choroid. Br. J. Ophthalmol. *54*, 200–202 (1970)

Blach, R.K., Davies, E.W.G.: The management of retinal dialysis. Trans. Ophthalmol. Soc. U.K. *87*, 317–333 (1967)

Brockhurst, R.J.: Photocoagulation in congenital retinoschisis. Arch. Ophthalmol. *84*, 158–165 (1970)

Brockhurst, R.J., Schepens, C.L.: Uveitis: peripheral uveitis the complication of retinal detachment. Arch. Ophthalmol. *80*, 747–753 (1968)

Broderick, J.D., Wyatt, H.T.: Hereditary sex linked retinoschisis. Br. J. Ophthalmol. *57*, 551–559 (1973)

Bruno, M.A., McPherson, S.D.: Harada's disease. Am. J. Ophthalmol. *32*, 513–522 (1949)

Byer, N.E.: Clinical study of senile retinoschisis. Arch. Ophthalmol. *79*, 36–44 (1968)

Byer, N.E.: Changes in and progress of lattice degeneration of the retina. Trans. Am. Acad. Ophthalmol. Otolaryngol. *78*, 114–125 (1974)

Byer, N.E.: The natural history of senile retinoschisis. Trans. Am. Acad. Ophthalmol. Otolaryngol. *81*, 458–471 (1976)
Chignell, A.H.: Retinal dialysis. Br. J. Ophthalmol. *57*, 572–577 (1973)
Chignell, A.H.: Retinal mobility and retinal detachment surgery. Br. J. Ophthalmol. *61*, 446–450 (1977)
Chignell, A.H., Markham, R.H.C.: Buckling procedures and drainage of subretinal fluid. Trans. Ophthalmol. Soc. U.K. *91*, 474–477 (1978)
Chignell, A.H., Fison, L.G., Davies, E.W.G., Hartley, R.E., Gundry, M.F.: Failure in retinal detachment surgery. Br. J. Ophthalmol. *57*, 525–530 (1973)
Constable, I.: Pathology of vitreous membranes and the effect of haemorrhages and new vessels on the vitreous. Trans. Ophthalmol. Soc. U.K. *95*, 382–386 (1975)
Cowper, A.R.: Harada's disease and Vogt-Koyanagi syndrome: Uveo-encephalitis. Arch. Ophthalmol. *58*, 764–766 (1951)
Cox, M.S., Schepens, C.L., Freeman, H.M.: Retinal detachment due to ocular contusion. Arch. Ophthalmol. *76*, 678–685 (1966)
Daniel, R., Kanski, J.J., Glasspool, M.G.: Retinal detachment in children. Trans. Ophthalmol. Soc. U.K. *94*, 325–335 (1974)
Dobbie, J.G., Phillips, C.I.: Detachment of ora serrata and pans ciliaris retinae in idiopathic retinal detachment. Arch. Ophthalmol. *68*, 610–614 (1962)
Dobree, J.H.: The nature of choroidal detachment fluid. Trans. Ophthalmol. Soc. U.K. *81*, 113–123 (1962)
Forstot, S.L., Binder, P.S., Fitzgerald, C., Kaufman, H.E.: The incidence of retinal detachment after penetrating keratoplasty. Am. J. Ophthalmol. *80*, 102–105 (1975)
Gloor, B.P.: Zellproliferation, Narbenbildung und Pigmentation nach Lichtkoagulation (Kaninchen-Versuche). Klin. Monatsbl. Augenheilkd. *154*, 633–648 (1969)
Gloor, B.P., Daicker, B.C.: Pathology of the vitreo-retinal border structures. Trans. Ophthalmol. Soc. U.K. *95*, 387–391 (1975)
Gonin, J.: Le décollement de la rétine: pathogénie: traitement. Lausanne: Librairie Payot et Cie. 1934
Gottlieb, F.: Combined choroidal and retinal detachment. Arch. Ophthalmol. *88*, 112–125 (1972)
Hagler, W.S., North, A.W.: Intra-retinal macrocysts and retinal detachment. Trans. Am. Acad. Ophthalmol. Otolaryngol. *71*, 442–454 (1967)
Hagler, W.S., North, A.W.: Retinal dialysis and retinal detachment. Arch. Ophthalmol. *79*, 376–388 (1968)
Hagler, W.S., Woldoff, H.S.: Retinal detachment in relation to senile retionoschisis. Trans. Am. Acad. Ophthalmol. Otolaryngol. *77*, 99–113 (1973)
Hamada, S., Ellsworth, R.M.: Congenital retinal detachment and the optic disc anomoly. Am. J. Ophthalmol. *71*, 460–464 (1971)
Hamilton, A.M., Taylor, W.: Significance of pigment granules in the vitreous. Br. J. Ophthalmol. *56*, 700–702 (1972)
Havener, W.H.: Massive Vitreous Retraction. Ophthalmic Surg. *4*, 22–67 (1973)
Heller, M.D., Straatsma, B.R., Foos, R.Y.: Detachment of the posterior vitreous in phakic and aphakic eyes. Mod. Probl. Ophthalmol. *102*, 23–36 (1972)
Hirose, T., Lee, K.Y., Schepens, C.L.: Snowflake degeneration in hereditary vitreo retinal degeneration. Am. J. Ophthalmol. *77*, 143–153 (1974)
Hogan, M.J.: Inflammation and its effect on the vitreous. Trans. Ophthalmol. Soc. U.K. *95*, 378–381 (1975)
Hovland, K.R., Schepens, C.L., Freeman, H.M.: Developmental giant retinal tears associated with lens coloboma. Arch. Ophthalmol. *80*, 325–331 (1968)
Hruby, K.: Clinical Observations of vitreous changes. In: Importance of the vitreous body in retinal surgery with special emphasis on reoperations. Schepens, C.L. (ed.), pp. 94–111. St. Louis: C.V. Mosby 1960
Hyams, S.W., Bialik, M., Neumann, E.: Myopia-aphakia-prevalence of retinal detachment. Br. J. Ophthalmol. *59*, 480–482 (1975)
Jungschaffer, O.H.: Arrow head tear in the macula. Arch. Ophthalmol. *86*, 19–20 (1971)
Kanter, P.J., Goldberg, M.F.: Bilateral uveitis with exudative retinal detachment. Arch. Ophthalmol. *91*, 13–19 (1974)
Karlin, B., Curtin, B.J.: Axial length measurements and peripheral fundus changes in the myopic eye. In: Retinal congress. Pruett, R.C., Regan, C.D.J. (eds.), pp. 629–642. New York: Appleton Century Crofts 1973

Laqua, H., Machemer, R.: Glial cell proliferation in retinal detachment. (Massive peri-retinal proliferation). Am. J. Ophthalmol. *80*, 602–618 (1975a)
Laqua, H., Machemer, R.: Clinico-pathological correlation in massive peri-retinal proliferation. Am. J. Ophthalmol. *80*, 913–929 (1975b)
Laqua, H., Machemer, R.: Repair and adhesive mechanisms of the cryotherapy lesion in experimental retinal detachment. Am. J. Ophthalmol. *81*, 833–846 (1976)
Lemcke, H.H., Pischel, D.K.: The time interval in the development of lattice degeneration of the retina. Am. J. Ophthalmol. *61*, 1216–1221 (1966)
Machemer, R., Laqua, H.: Pigment epithelial proliferation in retinal detachment. (Massive peri-retinal proliferation). Am. J. Ophthalmol. *80*, 1–23 (1975)
Margherio, R.R., Schepens, C.L.: Macula breaks. Am. J. Ophthalmol. *74*, 219–232 (1972)
Meyer-Schwickerath, L.: Light coagulation. St. Louis: Mosby 1960
Morse, P.H.: Fixed retinal star folds in retinal detachment. Am. J. Ophthalmol. *77*, 760–761 (1974a)
Morse, P.H.: Lattice degeneration of the retina and retinal detachment. Am. J. Ophthalmol. *78*, 930–934 (1974b)
Morse, P.H., Eagle, R.C.: Pigmentation and retinal breaks. Am. J. Ophthalmol. *79*, 190–193 (1975)
Mueller-Jensen, K., Machemer, R., Azarnia, R.: Autotransplantation of retinal pigment epithelium in intravitreal diffusion chamber. Am. J. Ophthalmol. *80*, 530–537 (1975)
Nagpal, K.C., Huamonte, F., Constantaras, A., Asdourian, G., Goldberg, M.F.: Migratory white without pressure retinal lesion. Arch. Ophthalmol. *94*, 576–579 (1976)
Norton, E.W.D.: Retinal detachment in aphakia. Am. J. Ophthalmol. *58*, 111–124 (1964)
Norton, E.W.D.: Management of retinal detachment in patients with intra-ocular lenses. Trans. Am. Acad. Ophthalmol. Otolaryngol. *80*, 135–136 (1976)
Norton, E.W.D., Aaberg, T.M., Fung, W., Curtin, V.T.: Giant retinal tears. Am. J. Ophthalmol. *68*, 1011–1021 (1969)
Okun, E.: Gross and microscopic pathology in autopsy eyes. Part II. Peripheral chorio-retinal atrophy. Am. J. Ophthalmol. *50*, 574–583 (1960)
Okun, E.: Histopathology of changes which precede retinal detachment. Mod. Probl. Ophthalmol. *4*, 76–97 (1966)
O'Malley, P., Allen, R.A., Straatsma, B.R., O'Malley, C.C.: Pavingstone degeneration of the retina. Arch. Ophthalmol. *73*, 169–182 (1965)
Percival, S.P.B.: Late Complications from Posterior Segment. Intraocular foreign bodies. Br. J. Ophthalmol. *56*, 462–468 (1972)
Phelps, C.D., Burton, T.L.: Glaucoma and retinal detachment. Arch. Ophthalmol. *95*, 418–422 (1977)
Phillips, C.I.: Distribution of breaks in aphakic and senile eyes with retinal detachments. Am. J. Ophthalmol. *47*, 744–752 (1963)
Pruett, R.C., Schepens, C.L.: Posterior hyperplastic primary vitreous. Am. J. Ophthalmol. *69*, 535–543 (1970)
Regenbogen, L., Stein, R.: Retinal detachments due to juxta papillary microholes. Arch. Ophthalmol. *80*, 155–160 (1968)
Ruben, M., Rajpurohit, P.: Distribution of myopia in aphakic retinal detachment. Br. J. Ophthalmol. *60*, 517–521 (1976)
Ruiz, R.S.: Giant cyst of the pars plana. Am. J. Ophthalmol. *72*, 481–482 (1971)
Rutnin, U., Schepens, C.L.: Fundus appearances in normal eyes. IV. Retinal breaks and other findings. Am. J. Ophthalmol. *64*, 1063–1078 (1967)
Sabates, F.N.: Juvenile retinoschisis. Am. J. Ophthalmol. *61*, 683–688 (1962)
Scheie, H.A., Morse, P.H., Aminlari, A.: Incidence of retinal detachment following cataract extraction. Arch. Ophthalmol. *89*, 293–295 (1973)
Schepens, C.L.: Sub-clinical retinal detachments. Arch. Ophthalmol. *47*, 593–606 (1952a)
Schepens, C.L.: Symposium: Retinal detachment; diagnostic and prognostic factors as found in pre-operative examination. Trans. Am. Acad. Ophthalmol. Otolaryngol. *56*, 398–418 (1952b)
Schepens, C.L., Brockhurst, R.M.: Uveal effusion 1. Clinical picture. Arch. Ophthalmol. *70*, 189–201 (1963)
Schepens, C.L., Freeman, H.M.: Current management of giant retinal breaks. Trans. Am. Acad. Ophthalmol. Otolaryngol. *71*, 474–487 (1967)
Schwartz, A.: Chronic open angle glaucoma secondary to rhegmatogenous retinal detachment. Am. J. Ophthalmol. *75*, 205–211 (1973)

Scott, J.D.: Static and dynamic vitreous traction. Trans. Ophthalmol. Soc. U.K. *91*, 175–188 (1971)
Scott, J.D.: Equatorial giant tears affected by massive vitreous traction. Trans. Ophthalmol. Soc. U.K. *96*, 309–313 (1976)
Scott, J.D.: Retinal dialysis. Trans. Ophthalmol. Soc. U.K. *97*, 33–35 (1977)
Seelenfreund, M.H., Kraushar, M.F., Schepens, C.L., Freilich, D.B.: Choroidal detachment associated with primary retinal detachment. Arch. Ophthalmol. *91*, 254–258 (1974)
Shafer, D.M.: Controversial aspects of the management of retinal detachment. Schepens, C.L., Regan, C.D.J. (eds.), p. 51. London: Churchill 1965
Shafer, D.M.: Retinal detachment after phacoemulsification. Trans. Am. Acad. Ophthalmol. Otolaryngol. *79*, 28–30 (1974)
Smith, J.L.: Retinal detachment and glaucoma. Trans. Am. Acad. Ophthalmol. Otolaryngol. *67*, 726–732 (1963)
Straatsma, B.R., Allen, R.A.: Lattice degeneration of the retina. Trans. Am. Acad. Ophthalmol. Otolaryngol. *66*, 600–613 (1962)
Straatsma, B.R., Foos, R.Y.: Typical and reticular degenerative retinoschisis. Am. J. Ophthalmol. *75*, 551–575 (1973)
Straatsma, B.R., Seeren, P.D., Foos, R.Y., Feman, S.S., Shabo, A.L.: Lattice degeneration of the retina. Am. J. Ophthalmol. *77*, 619–649 (1974)
Tasman, W.: Retinal detachment in children. Trans. Am. Acad. Ophthalmol. Otolaryngol. *71*, 455–460 (1967)
Tasman, W.: Retinal detachment with breaks in the pars plana. Am. J. Ophthalmol. *52*, 181–183 (1968)
Tulloh, C.G.: Distribution of holes and tears in primary retinal detachment. Br. J. Ophthalmol. *49*, 413–431 (1965)
Tulloh, C.G.: The aqueous flow and permeability of the blood aqueous barrier in retinal detachment. Trans. Ophthalmol. Soc. U.K. *92*, 585–611 (1972)
Vogt, A.: Die operative Therapie und die Pathogenese der Netzhautablösung. pp. 199–207, 223 Stuttgart: Enke 1936
Watzke, R.L.: The ophthalmoscopic sign "white with pressure" – a clinico-pathologic correlation. Arch. Ophthalmol. *66*, 812–923 (1961)

Chapter 3

Preoperative Management and Planning of Operation

Preoperative Management

In the preoperative discussion with the patient, the following points relating to retinal detachment should be stressed. Most patients prefer optimism in their surgeon, but although restoration of lost peripheral vision can be guaranteed with successful reattachment of the retina, a cautious prognosis should be given to the patient regarding the likelihood of return of central vision if the macula has been detached. The patient should be warned that the other eye will also be subjected to a careful examination at the time of detachment surgery and prophylactic treatment carried out if necessary. This procedure may result in lid swelling, and although the eye will not be patched, the discomfort and the swelling may cause patients some alarm in the postoperative period. The knowledge that both eyes are not to be covered after operation will allay anxiety in most patients, many of whom will fear waking after anaesthesia and being unable to see out of either eye.

The nature of any preoperative or postoperative posturing manoeuvres should be explained carefully to the patient since his active cooperation is usually an intrinsic part of such manoeuvres. If the case being dealt with is of particular difficulty, e.g. a giant tear, and it is felt that reoperation is a possibility, then this also should be discussed with the patient so that no loss of confidence is experienced if reoperation becomes necessary.

Local antibiotics are given routinely for an eye that is to be operated on, although conjunctival cultures are not taken.

Bed Rest

Bed rest for a period of 24–48 hours, combined with double padding, has been shown to reduce eye movements (Adams et al., 1973), and the reduction of eye movement and appropriate posturing may sometimes be a useful preoperative adjunct to retinal detachment surgery (Hofman and Hanselmayer, 1973; Chignell and Lean, 1979). It is contra-indicated in patients with a history of thromboembolism in whom deep vein thrombosis in the legs is a risk, and is avoided for the same reason in patients who are very old, or in patients likely to be emotionally disturbed. In all cases in which bed rest is to be employed the patient should be permitted to visit the bathroom and allowed

to sit up in bed without bandages for a short time to eat meals. Bed rest is instituted for the following reasons.

To allow dense vitreous haemorrhage to clear (Lincoff and Kreissig, 1975b). It is usually better to nurse the patient sitting upright to encourage vitreous haemorrhage to settle below on the retina and thus allow at least a view of the upper part of the retina which is the most likely site of a retinal hole and of detachment in such cases. The period of bed rest, particularly in younger patients, may be usefully extended up to four days. On rare occasions vitreous haemorrhage fails to clear sufficiently, to allow a limited view of the detached retina, which may then be confirmed by ultrasonography. When haemorrhages fail to clear, the hidden retinal detachment if untreated may proceed to massive periretinal proliferation and a vitrectomy, to wash out the vitreous haemorrhage, may then be indicated.

To aid reduction of subretinal fluid. The mechanism by which subretinal fluid may be reduced in quantity by bed rest is probably a simple descent of the retina due to gravity and the passage of subretinal fluid from the subretinal to the retrovitreal space via the retinal hole (Foulds, 1969). This reduction of subretinal fluid may be useful: a) to prevent detachment of the macula in early cases of superior detachment prior to surgery; and b) by encouraging approximate of the hole to the underlying pigment epithelium it will facilitate cryotherapy and localisation of the buckle at surgery.

The object of bed rest is to place the patient so that the retinal hole is in the most dependent position; thus, in superior detachment, the patient lies as flat as can be tolerated, sometimes with the foot of the bed raised. The head is inclined to one side or the other, depending on whether the hole is on the nasal or temporal side of the retina. The maximum effect of posturing to reduce subretinal fluid is achieved over a period of 24 hours if the patient is cooperative. In a consecutive series treated by bed rest (Chignell and Lean, 1979), the procedure was found to be of no value in long-standing retinal detachment, or of inferior detachment or when there was evidence of intraocular membrane formation resulting in immobile retina. It was found to be beneficial, however, in patients who had recent upper-half detachments: bed rest in this group resulted in appreciable reduction of subretinal fluid in 25% of cases.

Posturing

Much information may be gained of the behaviour of a giant tear from the preoperative posturing of the patient. When the posterior edge of the tear has flapped over, by posturing the patient for 24 hours before surgery to encourage unfolding, it will establish if there is any mobility of the flap. The patient is postured so that the posterior flap of the tear is dependent (Fig. 3.1). If the posterior flap of the tear is immobile the prognosis is poor, and a different surgical technique must be employed to when there is free mobility.

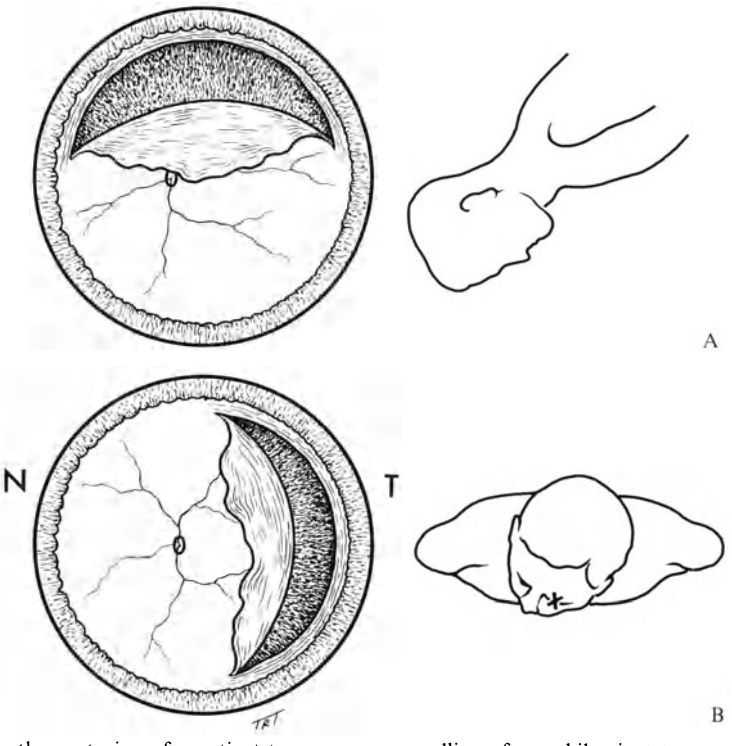

Fig. 3.1A–B. To show the posturing of a patient to encourage unrolling of a mobile giant tear. – The patient is arranged so that the posterior edge of the tear is encouraged to fall forwards into the vitreous cavity and to settle towards the anterior edge; (A) a patient with an upper half tear, postured prone and tipped slightly downward; (B) patient with temporal half tear of the left eye, prone, horizontal and lying on the left side of face

Medical Treatment

Uveitis

Treatment of preoperative uveitis consists of vigorous pupillary dilatation to prevent posterior synechiae and administration of local steroids. Systemic steroids are occasionally necessary, particularly if there is marked infiltration of the vitreous. The moderate degree of uveitis usually encountered is seldom enough to cause postponement of the operation as the view of the underlying retinal detachment will not be significantly reduced.

In cases of uveitis secondary to retinal detachment, surgical reattachment of the retina will result in rapid resolution of the uveitis.

Glaucoma

The administration of Diamox (250 mg four times a day) is usually adequate to control any preoperative rise in intraocular pressure.

Preparative Surgical Treatment

Removal of Infected Buckles

If it is apparent that an implant inserted at a previous operation has become infected, the implant should be removed as a preliminary surgical step. If it is protruding through a dehiscence in the conjunctiva, then it may be removed under local anaesthesia. If, however, the implant has led to considerable granuloma formation, or is not visible, then a general anaesthetic will usually be necessary, since the removal of the infected objects may be accompanied by much haemorrhage and the need for a difficult dissection. If an infected implant has to be removed, it is advisable if possible to wait for about one month before retinal detachment surgery can be performed.

Preparatory Anterior Segment Surgery

It is common for the examiner to have difficulty in visualising the fundus, mainly due to obstruction at the lens iris diaphragm which may be caused by cataract formation (senile or traumatic) or by iridocapsular remanants. The latter can arise from a variety of clinical situations, e.g. congenital cataract surgery following perforating injuries which have damaged the lens, or from complicated senile cataract surgery in which an intracapsular extraction was attempted but the lens capsule was ruptured, resulting in an extracapsular extraction. Such cases may greatly tax the expertise and patience of the observer, but in most cases at least some gap in the opacities will be found to allow a limited view of the posterior segment. This will usually suffice for a fundal examination and subsequent retinal detachment surgery. On rare occasions cataract surgery has to be performed in order to allow a better view of the detached retina. In senile cataracts a routine intracapsular extraction and broad iridectomy may be performed, whereas a traumatic cataract in a young person may respond best to lens aspiration or lensectomy. Excision of iris and capsular remnants by ab externo irido capsulectomy is to be avoided and generally results in a worsening of the retinal detachment situation. This latter condition will probably best be dealt with by removing the remnants with vitrectomy instruments, using an anterior segment approach since the pars plana approach is more dangerous in the presence of poorly seen rhegmatogenous detachment.

Selection of Operative Procedure

Method of Adhesion

Cryotherapy has become the accepted method of achieving, if not true choroidoretinal adhesion then intraretinal adhesion (Lincoff and McLean, 1960), convert-

ing the treated retina into firm scars from which redetachment will not occur (Bietti, 1933; Lincoff et al., 1964; Lincoff and McLean, 1969). The actual strength of the adhesion varies with the intensity of application of the cryotherapy and subsequent damage to the retina (Bloch et al., 1971). Recent experimental evidence, however, has suggested that it may be necessary to include the detached retina itself to achieve effective subsequent adhesion (Laqua and Machemer, 1976). Cryotherapy has now replaced diathermy as the method of choice of achieving adhesion (Norton, 1969) and over which it has several outstanding advantages. They are:

1. It allows full thickness scleral buckling procedures to be used because it can be applied through full thickness sclera and does not damage the underlying sclera. This lack of damage to sclera also contributes to a reduction in intraocular infection following surgery;
2. Heat damage is avoided to both sclera and vitreous (Banks, 1969; Kirkconnell and Rubin, 1965);
3. It can be applied transconjunctivally or directly through the sclera;
4. It does not damage large vessels, e.g. vortex veins and long ciliary vessels, and when applied to the long ciliary vessels there is no risk of anterior segment ischaemia (Freeman et al., 1966);
5. It is less likely to produce postoperative macular pucker than does diathermy (Hagler and Ataraliya, 1971).

The main practical disadvantage of cryotherapy is that it is sometimes dificult to see where treatment has been applied, particularly so when there is deep subretinal fluid. In general, most retinal surgeons now employ cryotherapy and clinical experience regarding its adhesive strength has amply confirmed experimental results (Lincoff et al., 1968).

Having accepted that cryotherapy is the method of achieving adhesion, the basic decisions to be made concerning the choice of operation are:

1. The type of buckle to be used;
2. Whether or not subretinal fluid has to be drained;
3. Whether or not any additional procedure is likely to be necessary (e.g. vitreous injection); and
4. The need for prophylaxis.

In arriving at a decision regarding the method of surgery to be used, an attempt must be made to select the least traumatic procedure that is consistent with a successful result, a principle stressed by Colyear, 1968; and Pischel, 1968. In most cases these decisions can be made prior to surgery, and only occasionally has the surgeon to alter the preoperative plan in the light of operative findings.

Scleral Buckle

Full thickness scleral buckles are almost invariably preferred today as the advent of cryotherapy has obviated the need for the partial thickness scleral beds

required in diathermy (Rubinstein and Hayes, 1969). This avoids the need for scleral dissection which can be difficult and hazardous when sclera is thin or when reoperations are performed.

Materials

For *local buckles,* silastic sponge implants are available in various diameters (either 3, 4 or 5 mm as round sponges, or an oval sponge 5.5 mm × 7.5 mm is used). Surgeons still preferring scleral beds have used a variety of other materials including gelatin and solid implants (Ray et al., 1975). Silastic sponges, first introduced by Lincoff, appear to be satisfactory for a variety of reasons.

1. The soft consistency of the sponge does not result in underlying scleral necrosis and only very slight thinning of sclera is produced even when the deepest buckles are raised.

2. The sponges are relatively inert and are well tolerated by the eye.

3. The inherent elasticity of the sponges allows some expansion of the sponge to occur in the postoperative period of a nondrainage operation as the intraocular pressure returns to normal and this will result in some additional heightening of the buckle.

The main disadvantages of silastic sponges are:

a) infection;

b) extrusion of the implants may occur at any time following surgery if the scleral sutures give way; and

c) the height of the buckle decreases in the weeks and months following detachment surgery. This, however, seems in most cases to be of very little disadvantage.

Silastic sponge cylinders may be used for *encirclement procedures* (Chawla, 1971) but the silicone rubber band is the most favoured method due to its simplicity in use. These flat rubber bands are available in a variety of sizes, of which the 2 mm (size 40 band) and less often the 5 mm bands are the most useful. They are inert and have superseded the Arruga suture and solid silicone rods because there is much less tendency for these flat bands to erode through the underlying sclera and, indeed, will not do so in normal sclera unless they have been tightened excessively.

It is often necessary when an encircling procedure is used to increase the height of the buckle at any one point of the encircling ridge. This can be done by using sponges or, if a particularly long segment needs additional support, an underlying silicone gutter may be used which is appropriately grooved for the rubber band in which it is to sit.

Principles of Scleral Buckling

The object of producing a scleral buckle is to close the retinal hole by approximating the pigment epithelium to the detached part of the retina containing

the retinal hole. The best method of achieving this, however, has been a source of continual disagreement among surgeons since scleral buckling was first practised. The encirclement procedure became popular as it was considered desirable not only to seal the tear with a buckle, but also because

1. The production of a 360° buckle achieved permanent relief of vitreous traction in the plane of the encirclement by reducing the diameter of the globe;

2. By creating 'a false ora serrata' there is a theoretical protection against further hole formation and redetachment anterior to the buckle;

3. Retinal holes not detected on examination will be inadvertently sealed if an encirclement procedure is used.

Increasing success with local buckling operations has shown that these procedures often only need to be confined to the region of the retinal hole, and the buckles produced do not necessarily have to be permanent. Progressive shallowing of the buckle raised by silastic sponge implants is usual, and even complete removal because of infection or extrusion has seldom resulted in redetachment. Thus, ordinary dynamic vitreous traction (Scott, 1971) needs only local relief and this does not necessarily have to be permanent. The permanent relief of traction is only useful if there is static vitreo-retinal traction which will produce a permanent and sometimes increasing pulling force on the retina.

The production of false ora serrata by an encircling element theoretically guards against detachment at a later date but there is very little evidence that the long-term redetachment rate with local procedures is significantly higher than that following encirclements.

Encirclement, with resulting buckling of what may be undetected retinal holes, is particularly favoured by the occasional retinal surgeon who may not have the time or expertise to elicit the preoperative physical signs or the confidence to place a local buckle accurately at the time of surgery. The encirclement procedure will apparently provide him with a greater margin for error whereas poorly performed local buckling procedures are likely to result in failure. These are not in themselves good reasons for routinely performing an encircling operation! However, difficulty is experienced in the finding of retinal holes even in the best hands and in some of these cases the encircling procedure is useful.

When considering the advantages of the encirclement procedure comparison has to be made with local buckling operations and to the relative morbidity of the two procedures. Complications following encirclement are greater than those following local implants (Criswick and Brockhurst, 1969). Encirclement carries a higher incidence of anterior segment ischaemia of varying severity, of glaucoma, choroidal detachment and causes subsequent enophthalmos. There is also the likelihood of more postoperative pain following encirclement procedures, and this may be temporary, lasting a week or two, or it may persist for many months. Encircling procedures are particularly contra-indicated when reduction of perfusion of the anterior segment is hazardous, e.g. HbSc disease, (Ryan and Goldberg, 1971). With local procedures a high degree of astigmatism may result when deep radial buckles are raised. Most of the serious complications of encirclement result from deep posterior encircling ridges that are rarely neces-

sary. A much less complicated encircling operation, aiming for a buckle of approximately 2 mm in height and augmented where necessary by local implants, may be performed without drainage of subretinal fluid, hence the more serious complications of fluid drainage are not added to those of the encirclement procedure. The success of the simpler and less traumatic local procedures has led us to employ encirclement with much less frequency. In a recent series (Chignell and Markham, 1978) encirclement was performed in 16% of cases as a primary procedure. However, the encirclement operation, particularly if it can be performed without drainage of fluid, still plays an important part in retinal detachment surgery.

Local Buckling Procedures

Local procedures are particularly suitable when a nondrainage operation is being performed. The combination results in a minimal operative procedure with low risk and morbidity. When a local buckle is to be used there are two main considerations: the size of the buckle and the direction of the buckle.

Size of Buckle

It is usually possible to decide on the size and shape of the buckle before operation. The size of the buckle is mainly determined by the size of the retinal hole to be treated and by the distance separating multiple holes, if present. The buckle produced must be of sufficient width and length to leave a safety margin of approximately 1 mm of retina between the hole and the edge of the buckle (Fig. 3.2). A greater margin of error must be allowed in a non-drainage operation if subretinal fluid is deep and localisation difficult. In this situation overestimation is particularly likely with radial implantation as it is often difficult to see where the posterior limit of the buckle should be. The width of the buckle can be increased, first, by increasing the diameter of a single sponge and, secondly by placing two sponges side by side (Fig. 3.3A) if more width is required. This latter step is used mainly to increase the width of radial buckles. The broadest width that can be attained is by placing two 7 mm sponges parallel to each other with widely placed sutures tied over the top. Two implants of this size will produce a buckle 8 mm wide at the equator which will be sufficient to cope with a retinal tear of approximately 40° at this point (Lincoff et al., 1977). The height of the buckle is determined by the distance by which the limbs of the scleral mattress sutures are separated and the amount by which the sutures are shortened when tied. The ease by which the sutures are shortened depends mainly on the intraocular pressure. If a non-drainage operation is being performed the intraocular pressure rises quickly and considerable tension will be placed upon the sutures to achieve a satisfactory indent. When the eye is softened by drainage, to achieve the

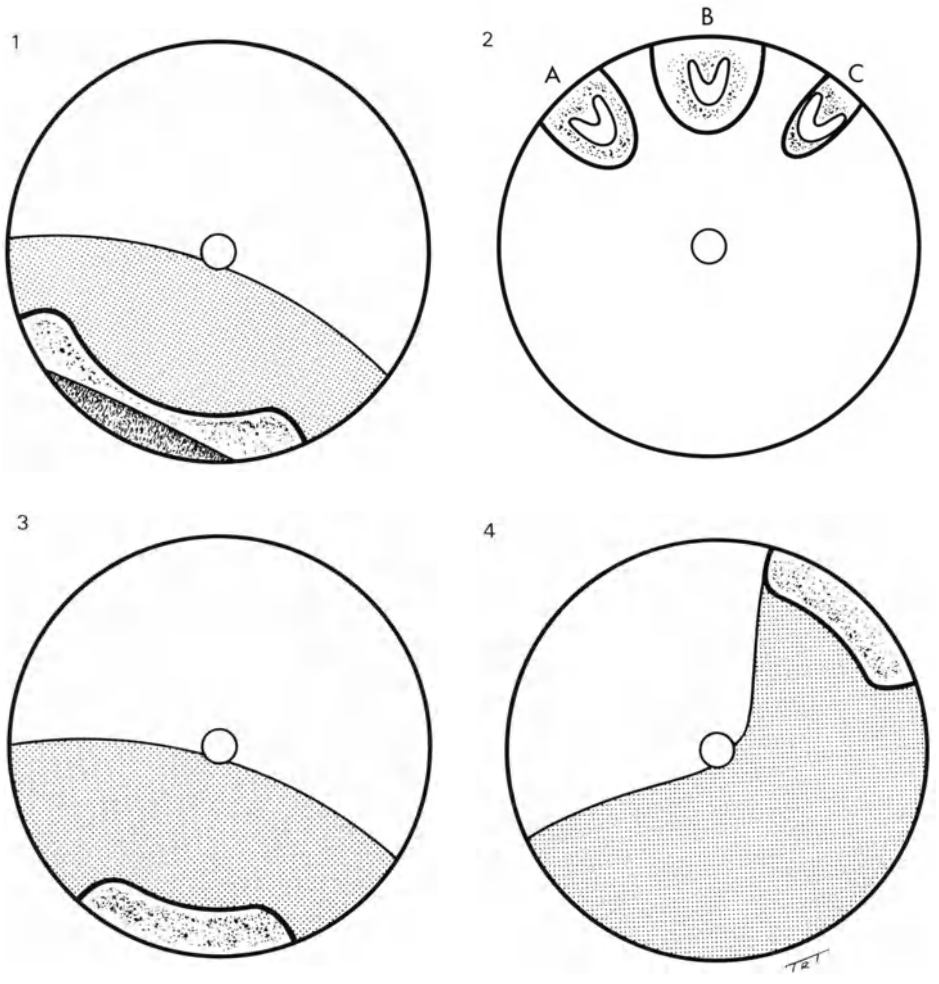

Fig. 3.2. (1) Use of a circumferential buckle to seal a dialysis; (2A) the selection of the radial buckle of appropriate width and depth to seal a horseshoe-shaped tear; (B) a buckle that is unnecessarily wide; (C) a buckle that is too narrow and carries a risk of not sealing part of the tear; (3) A circumferential buckle has been used to seal a presumed hole in the inferior retina; (4) A circumferential buckle has been used to seal a presumed hole in the superior retina

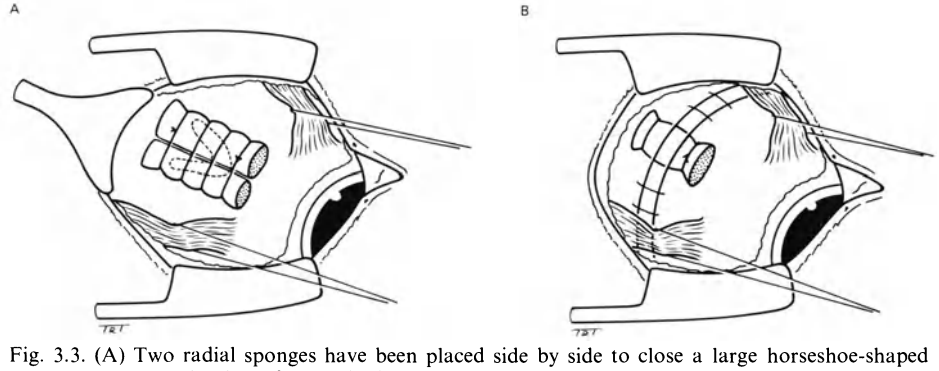

Fig. 3.3. (A) Two radial sponges have been placed side by side to close a large horseshoe-shaped tear; (B) The combination of an encircling band and a radial sponge

same height of buckle the sutures can be tied under much less tension. In practice, however, in most cases the height of the buckle produced by the non-drainage operation is entirely adequate to close the retinal hole successfully.

Direction of Buckle

The direction of the local implant is either radial or circumferential in relationship with the limbus. Obliquely placed implants are sometimes used. Occasionally, the disposition and orientation of the retinal holes may necessitate the combination of circumferential and radial implants and if these are adjacent an 'L' or 'T' shaped configuration results (Fig. 3.4E). The selection of the direction of the implant to be used depends upon the type of retinal hole and the relationship of one hole to another when there is more than one hole. Additional factors to be considered are the disposition of preoperative retinal folds and the relationship of these folds to the retinal holes. Radial implants obviate the risk of troublesome fishmouthing of retinal tears due to the occurrence of retinal folds (Lincoff and Kreissig, 1975a; Lincoff, 1974). In fishmouthing, a fold of retina extends over the buckle from the detachment behind the ridge to a hole, usually horseshoe-shaped, at its anterior extremity. Such folds form at right angles to any buckle that has been raised and are, therefore, particularly troublesome when circumferential buckles have been produced (Pruett, 1977). The folds may appear either de novo in the postoperative period but warning as to the likelihood of their subsequent appearance is given by the preoperative appearance of radial retinal folding in the vicinity of the retinal tears.

In a consecutive series of retinal detachments treated by local buckles (Chignell and Markham, 1977) radial implants were used in 45% of the cases. Radial implants (Fig. 3.4) are preferred in the following situations:

1. Medium or large holes that require a 5 mm buckle or larger;
2. Horseshoe-shaped tears. Radial implants are particularly suitable for buckling this type of hole; the buckle can not only close the tear but by supporting the tear in its long axis can relieve traction on its anterior aspect.
3. Single holes;
4. When the preoperative presence of radially orientated folds suggests a risk of postoperative fishmouthing;
5. When local buckles are to be combined with encirclement procedures (see Fig. 3.4C).

Circumferential buckles are indicated in the following:

1. Dialyses (see Fig. 3.2);
2. Multiple tears (even U-shaped). When close together and preventing the placement of multiple radial buckles.
3. When the position of the retinal holes is uncertain; up to one or two quadrants of retina may have to be buckled (Fig. 3.2 (3)).
4. Giant tears.

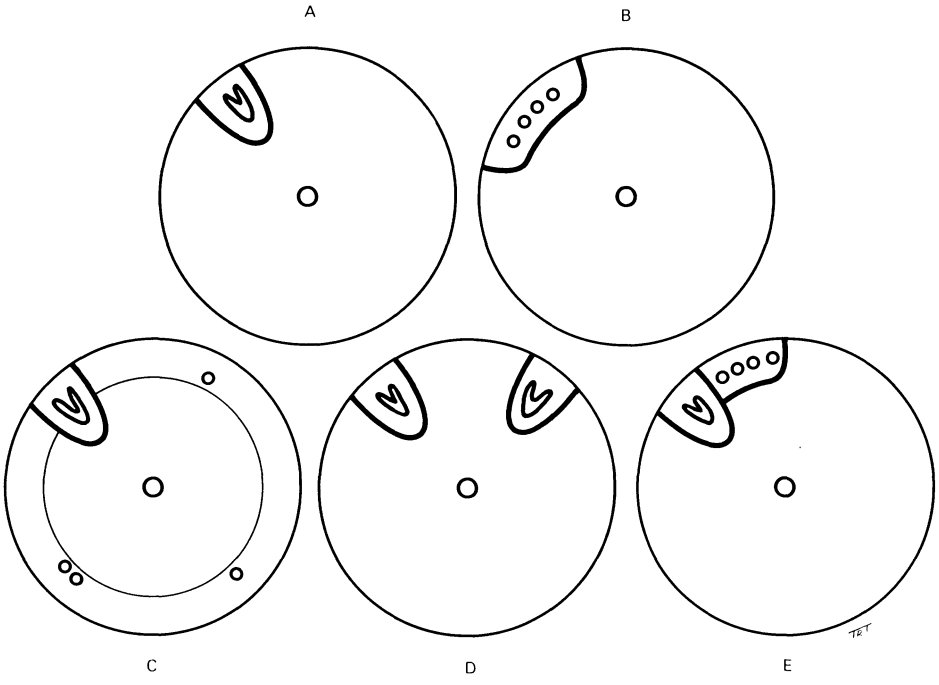

Fig. 3.4A–E. The treatment of different arrangements of retinal holes; (A) a single horseshoe-shaped tear treated by radial buckling; (B) a row of round holes treated by circumferential buckling; (C) multiple retinal holes treated by radial buckling of the horseshoe-shaped tear and an encircling band to close small round holes in three separate quadrants; (D) two separate horseshoe-shaped tears treated by separate radial buckles; (E) horseshoe-shaped tear in close proximity to a row of more anterior round holes treated by radial and circumferential buckles in close apposition

While the selection of the direction of the buckle is important in some situations, in others, particularly when the subretinal fluid is thin and holes are small, the choice may not be of great importance as in either case localisation and closure is easier and fold formation in the postoperative period extremely unlikely.

Encirclement Procedure

Indications for Encirclement

1. Uncertainty of existence of retinal holes. The failure to detect retinal holes may make local buckling unreliable:

a) When the distribution of subretinal fluid (Lincoff and Gieser, 1971) has not provided any useful information regarding the likely site of the retinal hole, e.g. when the contours are distorted by preretinal membrane formation. In a series of cases when it was not possible to find the retinal hole (Crick and Chignell, 1977), 60% were successfully treated with a local procedure.

b) Failed local procedures. In some case of failed local procedures, particularly if the detachment was originally total it may not be possible to find an adequate explanation for failure. In these cases an encirclement procedure will have to be performed.

c) Aphakia. In some aphakic detachments, particularly when the detachment is total, multiple small holes are often present and there is a risk of missing them when a local procedure is employed. Encirclement is preferred in these cases (35% of aphakic cases are treated with an initial encircling procedure, Chignell and Markham, 1977).

2. Multiple Holes. Retinal holes were multiple in 37% of detachment cases (Chignell and Markham, 1977). However, in only 16% of cases were the retinal holes separated by at least 90° of detached retina. Thus, it can be seen that even if multiple, the majority of retinal holes occur reasonably close to each other and can therefore usually be treated by local implants. In the cases in which there was at least one quadrant of retina separating the holes, 40% were treated by an encircling procedure.

3. Static Vitreoretinal Traction. Static transvitreal retinal traction, may best be treated by encirclement as in these cases it may be useful to reduce the diamenter of the globe in the plane of the encirclement. Also, traction may further increase after the operation if contraction of fibrous tissue continues. There is no obvious advantage, however, in treating purely preretinal membrane or retroretinal membranes with encirclement procedures. In these cases reduction of the diameter of the globe has no obvious effect on the traction involved and would appear to have little to offer.

4. Thin Sclera. On rare occasions it is impossible to suture a local implant in place due to extreme thinness of the adjacent sclera. In these cases an encircling band will have to be used.

5. Giant Tears. In most cases with detachment encirclement procedure is advised.

Non-drainage Operation

This operation was introduced initally by Custodis (1953) and later modified and popularised by Lincoff (Lincoff et al., 1965). At first highly controversial, this type of procedure has since slowly gained general acceptance (Weidenthal, 1967; Scott, 1970; Boke and Custodis, 1973; Chignell, 1974; O'Connor, 1976) and is based on the concept that no matter how carefully performed, the drainage of subretinal fluid from the eye still remains the least predictable step to be taken in retinal detachment surgery (Cibis, 1965). Numerous complications may be attributed to the drainage of subretinal fluid – choroidal haemorrhage, retinal incarceration and vitreous loss, hypotony, intraocular infection – and these

complications contribute significantly to failure in retinal detachment surgery, mainly by eventually promoting intraocular fibrosis (Chignell et al., 1973). It is obvious therefore that if this step can be eliminated, the operation becomes an entirely extraocular procedure and thus much safer.

The rationale of the non-drainage operation is that after accurate placement of the scleral buckle, resulting in closure of the retinal hole, either at the time of surgery or in the postoperative period (Fig. 3.5) subretinal fluid will be absorbed spontaneously, rendering it unnecessary to evacuate the fluid itself. If the hole can be closed at the time of surgery, then flow of fluid from the subretinal to the vitreal compartment is impossible and the disappearance of subretinal fluid can only occur in a posterior direction, i.e. across the pigment epithelium towards the choroid the detached retina itself not being well equipped to absorb fluid (Foulds, 1975). A trans-scleral passage of fluid was postulated by Foulds (1976). One of the most remarkable features of a non-drainage operation is that it has not been found necessary in all cases to close the hole at the time of operation, provided the buckle has been accurately sited underneath the hole and is of adequate dimensions. The hole will gradually sink back against the buckle, usually within a period of 24 to 48 hours, to be followed by absorption of the remaining subretinal fluid. In these cases the reduction of subretinal fluid in the early postoperative period is harder to explain. Expansion of the buckle as the high high intraocular operative pressure falls in the postoperative few hours cannot explain the settling back of the retina, which may take up to several days. Foulds (1975) has postulated that a thin layer of cortical gel may be forced into contact with the tear and act as a form of internal tamponage to prevent further recruitment of fluid from the vitreal space. However, it may be difficult to accept this explanation when very large tears are concerned or when there has been extensive detachment of the vitreous preoperatively from the vicinity of the retinal tears, particularly as recent work (Foos, 1977) has shown that in simple posterior vitreous detachment the separation of the vitreous from the retina was in fact complete and no residual layer of cortical

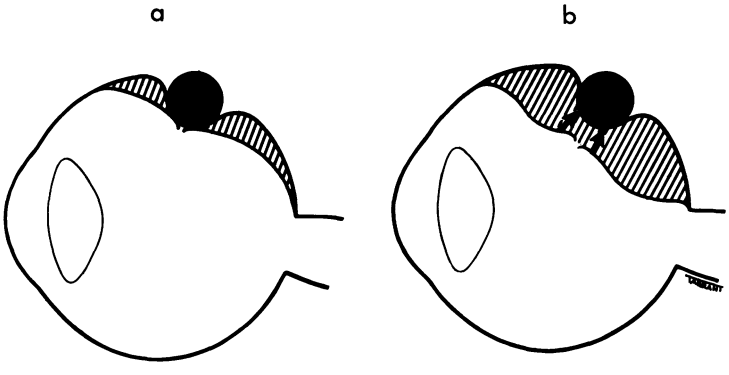

Fig. 3.5a–b. The non-drainage operation; (a) retinal hole closed by buckle at time of surgery; (b) the retinal hole has not been closed and for closure to occur in the postoperative period the detached retina must be mobile enough to fall back on the buckle

gel is left. Detachment of vitreous was incomplete only if preretinal membranes were present before detachment of the gel. It is likely that the simple placement of the buckle, possibly by inducing underlying choroidal ischaemia and secondarily affecting pigment epithelium, induces a posterior movement of fluid even before the holes become closed. Cryotherapy does not influence the absorption of subretinal fluid in the postoperative period, as patients treated without cryotherapy absorb fluid in an identical way (Chignell, 1977b; Zaubermann and Rosell, 1975). In addition to the main asset of avoiding obvious complications, the non-drainage operation has other advantages.

1. A quieter eye is encountered in the postoperative period due to reduction of traumatic uveitis.
2. The time of operation is reduced (usually).
3. In the event of reoperation the surgeon will return to an eye that has been little altered by its previous operation. In particular, there will be no increase in opacities of the media.

The incidence of the non-drainage procedure in a series of retinal detachment operations will to some extent reflect the complexity of the types of case being treated. In one series (Lincoff and Kreissig, 1972) the operation was performed in 88% of cases. Other series, which have a higher incidence of preoperative membrane formation resulting in retinal immobility, show a lower percentage of non-drainage cases, even though this technique is preferred whenever possible (65%; Chignell and Markham, 1977). It has also been found that an earlier operation is not necessarily a contra-indication for the non-drainage operation (Leaver et al., 1966), although it is accepted that in reoperations there is a greater tendency for there to be a fixed retina.

The aphakic detachment will respond as well to a non-drainage procedure as a phakic one (O'Connor, 1976).

Indication for Drainage of Subretinal Fluid

The non-drainage operation, using either local implants or encirclement procedures, may be applied to a wide variety of clinical detachment conditions and for the majority of detachments when retinal fibrosis resulting in retinal immobility is not advanced. However, the procedure is certainly not always appropriate, and drainage of subretinal fluid is indicated in the following situations.

High Intraocular Pressure

During the tightening of the buckle sutures during a non-drainage operation, the intraocular pressure may rise to appoximately 50–60 mm Hg. This pressure returns to normal in the healthy eye within about 15 to 20 minutes. However, there are occasions when it is dangerous to raise the pressure to this limit even for a short time. They include:

Open Angle Glaucoma. The outflow channels may be unable to cope with the raised pressure and demand for increased aqueous outflow. The rise in pressure may therefore be unduly prolonged and result not only in the risk of prolonged ischaemia to the optic disc but also in much longer operating time.

Ipsilateral Poor Ocular Perfusion. This may result in closure of the central retinal artery as soon as there has been even the slightest rise in intraocular pressure, e.g. in case of carotico cavernous fistula, ipsilateral carotid artery, or central retinal arterial disease. Similarly, hypotension as a result of anaesthesia may contribute to reduction of perfusion of the globe. Such an immediate closure of the central retinal artery may make the non-drainage operation impossible.

Rupture of an Anterior Segment Wound. When recent cataract or penetrating corneal graft surgery has been performed the non-drainage operation must be executed with caution. It may be safely performed three weeks to a month after uneventful cataract surgery if the wound has apparently healed well. This period should be extended for several weeks after graft surgery.

Poor View of the Disc. A good view of the optic disc is essential to check arterial patency, usually seen as arterial pulsation when the pressure rises. The view of the optic disc can be obscured by opacities in the media, or by a overhanging retinal detachment itself. The last-named is seldom a problem since by movement of the globe the retina can usually be induced to move out of the way to allow a fleeting view. However, if the optic disc cannot be seen, then the subretinal fluid will have to be drained.

Localisation Difficulties

Localisation of the buckle, particularly when there is considerable depth of subretinal fluid between buckle and hole, is often a matter of considerable judgement, and with increasing experience the accurate siting of the buckle can usually be achieved even when there are difficult holes. However, on occasions, particularly when there are multiple holes at different levels of detached retina (Fig. 3.6), it may be necessary to drain fluid to ensure more accurate localisation.

Immobile Retina

This, in our cases, is the commonest cause of the need to drain subretinal fluid. In a study series on non-drainage detachment cases (Chignell, 1974) it was found that in 79% it was not possible to close even a part of the hole at retinal surgery. Success in these cases depends upon the ability of the detached retina to sink back slowly against the buckle in the postoperative period (see

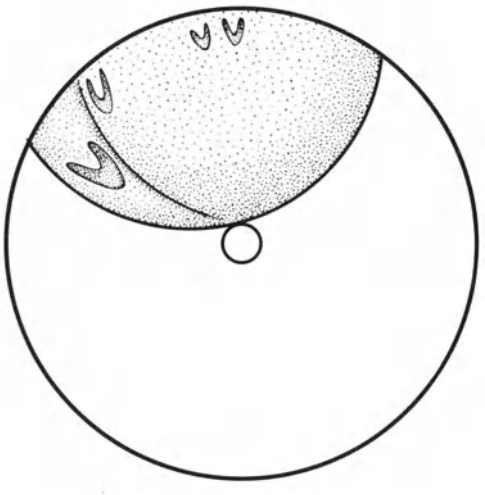

Fig. 3.6. Multiple retinal holes at different levels with highly elevated retina. Drainage was necessary to aid localisation

Fig. 3.5b). If the mobility of the detached retina has been impaired by membrane formation this capacity may be lost. When the retina is immobile, subretinal fluid may have to be drained from the eye so that the hole can be closed. The further the retina has to fall back, the more mobile it must be (Scott, 1972).

Internal Tamponage Procedures

When injection into the vitreous is planned the subretinal fluid will have to be drained in order to reduce the intraocular volume for the injection to be made.

Technical Problems at Operation

Thin Sclera. The raising of the buckle in a non-drainage operation puts considerable tension on the adjacent sclera and in some cases a thinned sclera is quite unable to hold the sutures. Drainage of fluid is then necessary to allow them to be tightened.

Access to Posterior Tears. When retinal tears are extremely posterior (for example in macula or juxtapapillary holes) it may be difficult to achieve adequate exposure of the globe for suturing purposes and placement of the implants. The problem may be further exacerbated in patients who have naturally deep-set eyes. Draining subretinal fluid in these cases reduces the intraocular volume and by allowing the globe to be compressed provides access to the posterior sclera.

It is quite common in cases of retinal detachment to encounter a combination of circumstances that, although they may not individually demand drainage

of fluid, together will add up to a most difficult operative situation; for example, thin sclera with multiple tears on high balloons, a situation that may be much facilitated by drainage of fluid.

Intraocular Injections

In selected types of retinal detachment intraocular injections may not only be of assistance to the surgeon but sometimes the only mechanism by which the retina can be replaced.

Retinal Tamponage

The physical pushing back of the retina into place as a result of vitreous injection is performed only when it is considered that the normal buckling of the retina will be inadequate to secure retinal reattachment. This may be necessary:

a) When there is sufficient periretinal fibrosis to result in a highly elevated immobile detachment of the retina, particularly if such elevation is situated in the vicinity of the retinal hole. In these cases, if high buckling and drainage of subretinal fluid does not approximate the hole to the buckle, intraocular injection is indicated to achieve this. In these cases the injection, by forcing the retina into place against the tensile strength of the membranes, may have the added advantage of helping to break down the surface traction of less well-established membranes.

b) When it is impossible to close a retinal hole by conventional buckling procedures because of the configuration of the retinal hole, e.g. giant or irregular tears.

c) To allow a torn retina to be manipulated into its correct place, e.g. in the treatment of flapped-over giant tears.

Hypotony

Hypotony occurring during retinal surgery may arise inadvertently due to excessive drainage of subretinal fluid, or because it has been necessary, particularly in the presence of fixed retina, to drain a good deal of subretinal fluid from the eye to achieve closure of a hole. Following tightening of the buckle sutures the eye may still remain excessively soft or even collapsed with risk of choroidal haemorrhage. The temptation to restore intraocular pressure further by greatly increasing the height of the buckle should be resisted. This is particularly so if encirclement procedures are to be used, since excessive tightening will result in the globe being grossly constricted. In these cases, therefore, intraocular injection is being used to restore normal intraocular pressure. Restoration of a more normal pressure has the added advantage of flattening out redundant folds of detached retina which are often greatly accentuated when the globe is hypotonic.

Materials for Intraocular Injection

Air or Gas

The practice of injecting air into the vitreous cavity has been used for many years (Rosengren, 1938). The main disadvantage is that it is usually rapidly absorbed from the vitreous cavity during the postoperative period so that the effect of the tamponage is quickly negated. More recently, other intraocular gases have been used, sulpha hexafloride (SF6) having proved to be the most useful. This gas when introduced into the eye absorbs nitrogen from the blood so that the bubble of gas does in fact increase in the postoperative period (it will double its volume in 24 hours). When used undiluted, the expansion may raise the intraocular pressure to a dangerous level, a tendency that can be avoided by using a 40–60 SF6/air mixture at the time of surgery. This will produce an intraocular bubble that will have an effective life of approximately two weeks, if it is a 2.5 ml injection. The advantages of using an intraocular gas in the management of some types of retinal detachments has been stressed by Norton, 1973; and by Chawla and Birchall, 1973. Experimentally it has been found that both air and SF6 can induce posterior capsular cataract changes that may be avoided by making sure that the bubble of gas is not in contact with the posterior lens capsule in the postoperative period. This can be achieved by appropriate posturing of the patient, avoiding the supine position (Fineberg et al., 1975).

Silicone Oil

An eye in which intraocular fibrosis is very extensive or in which end-stage massive periretinal proliferation has in fact been reached, usually after a series of unsuccessful detachment operations, will no longer respond to conventional retinal surgery. Tamponage with air or SF6 may result in temporary reattachment of the retina only to be followed by progrssive contracture of membranes and redetachment. It is for these cases that the use of silicone oil has been advocated (the 1000 cs viscocity is preferred). The object of injecting this material, which is completely transparent and possesses high surface tension, is to fill the vitreous cavity permanently and to push detached retina against the pigment epithelium. Since the introduction of this technique (Cibis et al., 1962) initial enthusiasm for the operation has fallen away (Cockerham et al., 1969) due to what was considered to be an unacceptably high complication rate (Watzke, 1967), complications that repeated themselves in a later series (Grey and Leaver, 1977). The trouble consists of cataract formation due to the contact of silicone oil with the posterior lens capsule, and glaucoma and keratopathy associated with emulsification of silicone oil in the anterior segment. Direct infiltration of emulsified silicone into the retina has been demonstrated experimentally (Mukai et al., 1975) and following enucleation of human eyes that have received silicone oil injections (Sugar and Okamura, 1976). It is not

yet quite clear whether silicone retinopathy exists as a clinical entity and will in itself result in deterioration of vision in an eye that has previously had silicone oil injection and in which there is progressive and unexplained reduction of vision.

It has been pointed out (Scott, 1977) that emulsification of silicone oil is responsible for most of the reported complications and that the problem can be avoided in part by filling the vitreous cavity as completely as possible with oil so that the bubble becomes immobile and there is less tendency for the silicone bubbles to break off and emulsify.

The functional results of silicone oil surgery in these desperate cases has been assessed in different series (Watzke, 1967; Grey and Leaver, 1977). The latter authors found that in 39% of cases there was improved vision after a year and that 41% were unchanged. Unfortunately, however, 20% of the patients demonstrated deterioration of visual acuity to a level lower than that preoperatively. In some cases where there has been excellent anatomical reattachment of the retina, the final visual result is unexpectedly disappointing (Okun et al., 1969).

The use of silicone oil is still strongly championed by some authors (Scott, 1972; 1973; 1974; 1977). There is no doubt that in exceptionally difficult cases where there is massive preretinal retraction, no other operative procedure at the present time offers a better prospect than silicone oil for regaining at least some vision. However, the complication rate and sometime disappointing results of surgery would deter most surgeons from injecting silicone oil if the fellow eye was enabling the patient to lead a normal life. If, on the other hand, this too was severly diseased, then the injection of silicone oil seems justified at the present time.

Selection of Operation in Special Cases

Rhegmatogenous Detachment Without Apparent Holes

In some cases of retinal detachment it may not be possible to find retinal holes. The failure is usually due to a combination of circumstances, e.g. opacities in the media combined with retinal detachments in which there are small holes. Occasionally, extensive opacities in the media may conceal quite large retinal holes. The surgeon is unable to identify a retinal hole, either at the preoperative examination or at the time of surgery. In addition to the more vigorous scleral depression that is possible at the time of surgery, holes may be revealed when suspicious retinal areas are frozen by cryotherapy. If, however, holes are still not demonstrated, a decision then has to be made regarding the type of buckling procedure that will be needed. In these cases surprisingly good results have been obtained (Crick and Chignell, 1977; Griffith et al., 1976).

One of the reasons why this group of cases has unexpectedly been successfully treated is that the size of the retinal holes that could not be detected were probably small and thus easily closed by the buckling procedures. Even though the retinal holes could not be detected, in a high proportion of cases it was possible to deduce from the distribution of subretinal fluid where the hole ought to be and in these cases a circumferential buckling procedure was used, which resulted in a fairly broad buckle from the pre-equatorial region to the ora serrata (see Fig. 3.2). It is usually possible in these cases to be sure that the more centrally placed retina is free from hole formation, but even so, encirclement is performed more often than usual, and there is no particular need to drain subretinal fluid, provided it can be established that there is no marked degree of retinal immobility.

Retinoschisis Complicated by Detachment

If the schisis is entirely separate from the retinal detachment, then the area of retinal detachment should be treated in the usual way and the schisis left completely alone. If, however, the schisis has led to retinal detachment, the usual situation, it is the outer leaf holes that should be treated as in a normal detachment, and it is not necessary to close any inner leaf holes if found.

At the time of surgery, the localisation of the awkwardly shaped outer leaf holes is often difficult and drainage of subretinal fluid is usually necessary to effect a reasonable localisation. The holes themselves are usually confined to one quadrant of the retinal detachment and an encircling procedure is rarely indicated. Satisfactory closure of retinal holes will result in complete absorption of subretinal fluid. Retinoschisis fluid, however, often persists but may completely disappear if the schisis compartment is accidentally drained, or it may resolve spontaneously later due to the effect of cryotherapy on the outer leaf.

Macular Holes

The management of macular holes is difficult. Once a macular hole has actually formed the chances of regaining good central vision are slight; furthermore, most of the operative procedures suggested for the treatment of macular holes in themselves carry a considerable degree of risk (Adams, 1961). Retinal detachments confined to the posterior pole alone, and in which good peripheral vision is still present, are therefore best left untreated as in some cases they may not progress. Progressive detachments, however, due to macular holes extending to at least the equator, and in which there is extensive field loss, require surgical intervention.

Treatment of Macular Holes

If the detachment is one that has been clearly observed to have spread slowly from a central posterior polar detachment, then the macular hole itself must be treated. The spread of subretinal fluid in these cases is usually in a somewhat eccentric way, spreading towards the temporal equator and also inferiorly. The detachment will extend to the ora serrata and, if untreated, eventually proceed to total detachment. Spread from a central area is not usually clearly demonstrated even by successive examinations, and the surgeon may be confronted with what appears to be a full thickness macular hole, a detachment extending to the ora serrata, but no sign of a peripheral hole. In these cases, and those in which a peripheral hole can be found, a peripheral buckling procedure should be performed as the primary step. This is because, although it can usually be established at the slit-lamp examination that a macular hole is in fact full thickness, it is not possible to know if such a hole is in fact propagating retinal detachment. Even cases in which the macular hole is full thickness may respond well if a peripheral hole is sealed. If, however, the peripheral buckling procedure does not result in anatomical reattachment of the retina then the macular hole is treated as a secondary step.

Direct Buckling of the Hole

The type of buckling procedure to be used depends upon the state of the posterior pole of the eye. In high myopia where there is usually a very thin posterior staphyloma, direct suturing of the macular region is hazardous, and there is a high risk of choroidal haemorrhage. In these cases a variety of operations have been devised which have as their common denominator the production of a buckle by avoiding suturing in that area. One current method of treatment is a sling procedure where a band or sponge is passed from the upper nasal quadrant to the lower temporal quadrant. The implant is firmly fixed above, and after drainage of subretinal fluid the inferior arm, sometimes with a relieving strut to the upper temporal quadrant, is tightened which allows the ridge to be raised (Feman et al., 1974). A further type of indentation has been effected by manipulating a circumferentially orientated metallic rim with an arm passing backwards in the upper temporal quadrant to indent the macula (Kloti, 1964). The disadvantage of this procedure is that the clip has to be removed about 10 days after surgery.

In emmetropia, exposure of the posterior part of the globe is not difficult, particularly if a lateral canthotomy is performed and the lateral rectus is disinserted. The sclera is of sufficient thickness to allow suturing without risk and, surprisingly, vessels entering or leaving the eye appear to tolerate the buckling procedure well. It has been found that a 4 or 5 mm radial implant can easily be placed so that its posterior limit is just short of the optic nerve itself (Fig. 3.7). When macular holes are being buckled it is not usually necessary to apply cryotherapy; application will only serve to disturb macular function

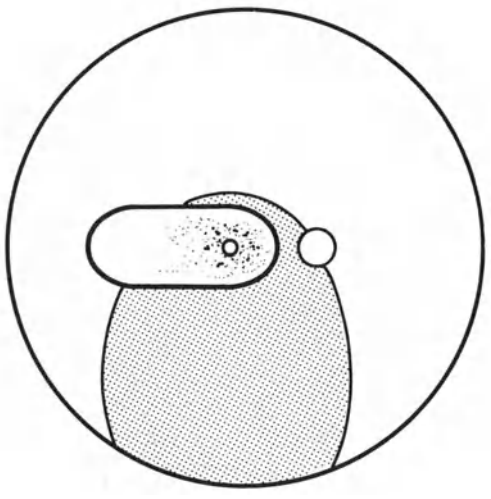

Fig. 3.7. The use of a radial buckle to treat a macular hole in an emmetropic patient

even further. Manipulating buckles at the posterior pole also carries the added risk of damaging the optic nerve, resulting subsequently, in optic atrophy.

Internal Tamponage

This method has been suggested for treating posterior polar tears, and air, gas, and even silicone oil (Scott, 1974) have all been used. The attraction of internal tamponage is that no buckling of the posterior quadrant is necessary with consequent reduction in operative trauma. With air or gas the patient has to be postured postoperatively so that the gas bubble is in contact with the posterior pole.

There is difficulty in assessing the relative merits of various procedures in the treatment of detachments associated with macular holes as the cases are rare and the reported series therefore small. Successful surgery may result in a very useful peripheral field of vision although return of central vision is usually poor (Leaver and Cleary, 1975).

Giant Tears: Principles of Treatment

In cases in which the posterior edge of the tear has not become markedly displaced, there may be no actual retinal detachment so that a prophylactic procedure alone, involving cryotherapy to surround the tear, may be all that is necessary (Fig. 3.8A). These cases are rare and it is usual to find at least some subretinal fluid present. If there is, but the retinal flap has not become markedly displaced, then a simple buckling procedure can be performed. This will take the form of a wide but shallow circumferential implant arranged so that at each end of the tear the circumferential buckle is brought round and arranged in a radial fashion. This will prevent further extension of the

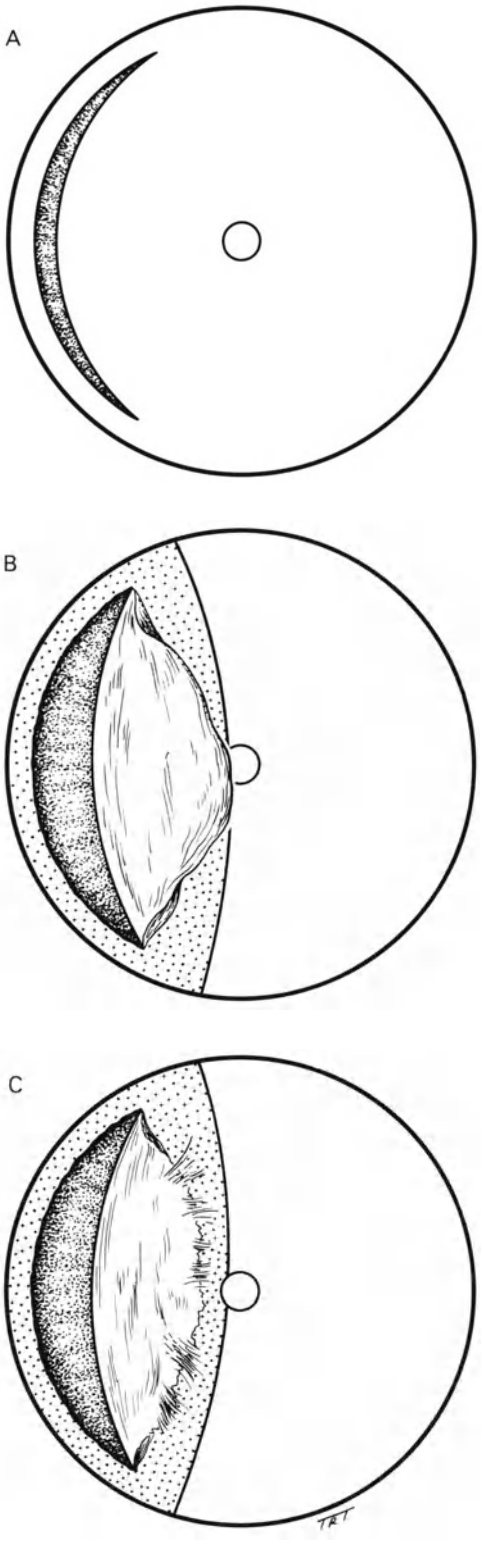

Fig. 3.8A–C. Clinical conditions in giant tears; (A) no displacement and no subretinal fluid; (B) posterior edge of tear has flapped over but is mobile – subretinal fluid has accumulated; (C) posterior edge of tear has become fixed in the retroverted position, preventing unrolling

retinal tear or leakage of subretinal fluid from either end. It is important that the buckle should not be too high as a long circumferential buckle is particularly liable to result in radial folds forming (Lincoff et al., 1977).

In cases in which the posterior flap has become displaced but remains mobile (Fig. 3.8B) the mobility of the tear, which is usually one of not more than 180° and will have been obvious during the preoperative examination of the patient, will indicate that the retina can be pushed back into place by intraocular manipulation, using air or gas. The intraocular gas forces the detached retina back against a prepared bed of cryotherapy and, having achieved unrolling, a low circumferential buckle either local or encircling is then raised. In some cases air or gas and cryotherapy alone can be used (Norton et al., 1969) but my experience has been that a buckle is needed to maintain reattachment. An air/gas mixture is used, usually about 3 ml, which is enough to provide adequate tamponage but not big enough to prevent the flap unrolling. In the postoperative period the patient is postured to encourage contact of the gas bubble with retina at the site of the giant tear.

Sometimes the posterior flap of a giant tear becomes fixed due to the formation of preretinal membrane (Fig. 3.8C). These cases carry an very poor prognosis for reattachment since conventional surgery has no chance of success. Vitrectomy has been advocated to remove the vitreous from the eye and to free adhesions holding down the retinal flap, and having mobilised the flap, air or gas is then introduced into the eye in an attempt to keep it in position; this technique is also necessary when tears are greater than 180°, even if still mobile, as these cases can rarely be unfolded by simple gas-bubble techniques. The use of a rotating operating table to manipulate the intraocular gas has been found to be of benefit (Machemer and Allen, 1976). Silicone oil injection has also been used in the treatment of these most difficult cases (Scott, 1976).

The prognosis for successful reattachment of the retina when a giant tear is of 180° or less is good either when the retinal flap has not become displaced, or when a flap is displaced but is still mobile. Once preretinal membrane has formed, or if the tears are 180° to 360° the prognosis becomes correspondingly poor. High myopia or aphakia in addition appears to carry a particularly poor prognosis (Kanski, 1975). One of the most disappointing features in these cases is the tendency for eyes containing giant tears to pass into massive periretinal proliferation, and this can come about even though the retina has apparently been satisfactorily re-attached for a few weeks. The onset of massive periretinal proliferation is the main cause of failure in these most difficult cases (Freeman, 1969).

Detachments Associated with Uveal Colobomas

Retinal detachment is sometimes seen in these cases and may be of two types:

a) The detachment may arise from holes quite separate from the coloboma itself, in which event the detachment can be treated without regard to the

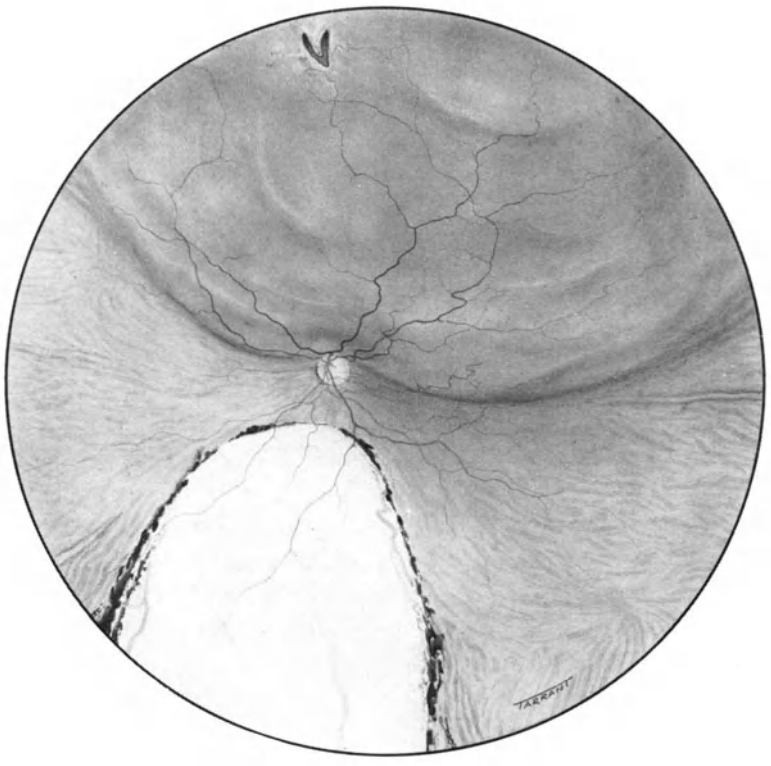

Fig. 3.9. An upper half retinal detachment that is quite separate from an inferior coloboma

coloboma and with a normal chance of reattaching the retina (Fig. 3.9);

b) Sometimes no tears can be seen and it is presumed that they are related to the edge of the coloboma, in which case the two edges of the coloboma are buckled using radial implants in the hope of reattachment. In general, however, this type of detachment carries a rather poor prognosis.

Detachments Associated with Intraocular Fibrosis

It is useful to consider the special demands of patients in whom retinal detachment has been complicated by some degree of intraocular fibrosis.

A simple traction detachment in which vitreo-retinal traction has resulted in a tenting up of the retina at the point where the vitreous is attached, and in which there is no full thickness retinal hole, seldom requires treatment since traction detachments of this type are usually static. If, on the other hand, a traction detachment is complicated by full thickness hole formation with rhegmatogenous detachment occurring as a secondary event, or if a simple rhegmatogenous detachment is complicated by periretinal fibrosis, then surgical intervention is necessary. The considerations for surgical treatment are similar

in both types, although the aetiology and anatomical questions that they pose are somewhat different.

Whatever the method by which fibrosis has arisen within the eye the guidelines for treatment in accordance with the severity of the fibrotic process may be summarised as follows.

If the periretinal membrane formation is not too extensive and the retinal holes can be identified, the holes can be treated by local buckling procedures in the normal way. If the membranes are not in fact closely related to the hole, a non-drainage procedure can usually be adopted. If, however, the membranes are in close proximity to the hole, there may not only be a distorted hole, necessitating a slightly bigger buckle, but there will also be some degree of retinal immobility, and, depending upon the depth of subretinal fluid between the retina and pigment epithelium, a drainage procedure may be necessary so that the hole is closed at the time of surgery. There is little to be gained when membranes are present on the surface of the retina to involve them in the buckling procedures. The only occasion when this is useful is when the membranes are suspected of containing undetected retinal holes.

If, on the other hand, there is extensive membrane formation with highly elevated fixed retina, and there are retinal tears, it may not be possible to close the holes with simple, local buckling, even though the subretinal fluid has been drained off. After drainage there may still be a considerable depth of subretinal fluid between the buckle and the fixed detached retina. In these cases intraocular injection of air and gas is advised to push the detached retina back against the buckle. If the hole is placed in the upper half of the retina, 1 to 2 ml of gas will be adequate. If inferiorly placed, more gas will be necessary to achieve effective tamponage. In these difficult cases there may be extensive equatorial vitreo-retinal traction and the eye will be on the verge of massive periretinal proliferation. In these cases the areas of traction should be supported with circumferential buckling, and if the elevation of the retina is very high or if the areas of traction extend for approximately more than one quadrant in extent, this should take the form of an encirclement, which should be deep enough to approximate detached retina to pigment epithelium — the approximation being assisted by the injection of air and gas and drainage of subretinal fluid.

The treatment of the eye will be rather different if it is not possible to detect a retinal hole. In mild or moderate cases of membrane formation it may again be possible, by the distribution of the subretinal fluid, to deduce the site of the retinal hole although the deduction will be much more difficult when retinal fibrosis is widespread as retinal contours are distorted. A more extensive buckling procedure will usually need to be performed as the membranes may conceal undetected retinal holes. Drainage of subretinal fluid will usually be necessary in these cases. When membrane formation is advanced, particularly if the retinal detachment is total, an equatorial encircling band will have to be used, combined with drainage of subretinal fluid and injection of intraocular gas and air.

In the treatment of cases where transvitreal or vitreo-retinal bands have resulted in extreme elevation and hole formation at the point of attachment to the retina, conventional buckling, even with drainage of subretinal fluid and gas injection, will not close the retinal tear, e.g. in highly elevated fixed inferior retinal detachment following extensive vitreous loss and incarceration of vitreous into the wound at cataract surgery (Norton and Machemer, 1971). In these cases it will be necessary to cut the offending vitreous bands to achieve reattachment by releasing traction. In the past, the bands were severed by means of intravitreal scissors alone, a technique that is now rarely used without performing vitrectomy at the same time. When end-stage, massive periretinal proliferation is present, a stage that is usually arrived at only when the patient has experienced one or more unsuccessful buckling procedures, it may be assumed that a further conventional operation will no longer be successful and a decision must be made as to whether or not further surgery is worthwhile. The choice of operations lies between silicone oil injection and vitrectomy. Vitrectomy via the pars plana combined with injection of air or gas, drainage of subretinal fluid and encirclement, offers hope in some cases (Huamonte et al., 1977) while vitrectomy and peeling of membranes may help in others (Machemer, 1976). However, success rates are not particularly encouraging (Aaberg, 1976) at the moment, but it does seem that improved vitrectomy techniques will offer a better chance of reattachment in the future.

Prophylactic Surgery

The object of prophylaxis is to recognise and treat lesions that lead to retinal detachment. After demonstration of the importance of retinal holes in the aetiology of detachment, improved methods of examination, followed by a relatively safe method of treatment in the form of photocoagulation (Meyer-Schwickerath, 1960, 1964; Zollner, 1964), there was a tendency to treat *all* conditions known to be associated with retinal detachment, an attitude that now has been modified (Charamis and Theodossiadis, 1972). It has been found that not only is it unnecessary to treat all retinal holes and degenerations, because they dot not necessarily lead to detachment, but also because if they are treated there is a risk of complications (Mortimer, 1966; Chignell and Shilling, 1973), and further, it would be quite impracticable to treat the very large number of patients who have in fact clinically detectable holes or degenerate lesions. The consideration of an eye for prophylaxis mainly concerns the treatment of either retinal holes or retinal degeneration which, in turn, may lead to retinal hole formation.

Retinal Holes

The potential risk of untreated retinal holes has been stressed by various authors (Colyear and Pischel, 1960; Lincoff, 1961; Meyer-Schwickerath, 1964) and,

in particular, the vulnerability of the fellow eye of the patient with retinal detachment has been recognised. In these fellow eyes, Merin et al. (1971) strongly advocated prophylaxis (19% of patients had holes in their fellow eyes and 20% of these cases subsequently developed retinal detachment). The incidence of retinal detachment is increased two or three times in aphakic fellow eyes (Davis et al., 1974).

The incidence of retinal holes in the general population, however, is high: it was estimated as 13.7% in volunteer patients without eye disease (Rutnin and Schepens, 1967), 5.8% in a study of 1700 patients were found to contain asymptomatic retinal breaks (Byer, 1967), and an estimate of 10% was given in a study of 250 patients (Halpern, 1966). Myopic eyes were found to contain 11% of retinal holes that had not caused symptoms (Hyams and Neumann, 1969). The incidence of holes was found to be high (9%) in a non-myopic aphakic group (Friedman et al., 1973). These clinical findings of a relatively high incidence of retinal holes have been supported by pathological studies where unsuspected retinal holes were found on post-mortem examination. An incidence of 4.8% was noticed by Okun (1961) and 10.6% by Foos and Allen (1967). Thus, although the incidence of retinal holes in the population is fairly high, the incidence of detachment in the population is low and in a study of a relatively stable population has been estimated (Bohringer, 1956) to occur in 0.004% of population per annum. It is reasonable therefore to assume that only a small proportion of patients with retinal holes develop detachment (Hyams et al., 1974). In a series of 108 eyes that had retinal holes (Neumann and Hyams, 1972) only three patients subsequently developed retinal detachments.

The great majority of the holes detected in the clinical studies described above were found on routine clinical examination and were asymptomatic, and it is felt that these holes carry a much better prognosis than those that present to the ophthalmologist with fresh symptoms (Davis, 1974). This study showed that only 5% of asymptomatic holes progressed to retinal detachment, whereas in the symptomatic hole this incidence was raised to 18%, with horseshoe-shaped tears carrying a much greater risk than round holes. The study also showed that if a hole is to proceed to retinal detachment, it is likely to do so within the first six weeks from its appearance, following which the risk of detachment rapidly decreases. Thus, there appear to be retinal holes with a low risk of detachment, and others that carry a high risk, but even so it is still not possible to forecast with absolute certainty which retinal holes will proceed to retinal detachment and which will not. A balance has to be made therefore between the need to treat retinal holes when detachment seems a real threat and to avoid unnecessary treatment in those cases where there is little risk. The reason symptomatic holes may be more dangerous than asymptomatic ones is probably a reflection of a number of factors – thus, a symptomatic hole is likely to be fresh and have active vitreous traction if U-shaped. It should also be remembered that the distinction between symptomatic and asymptomatic holes may be somewhat artificial. Symptoms themselves may

be trivial, variable, or even forgotten by the patient. In spite of this limitation, however, the following scheme for prophylactic treatment of retinal holes is advised.

Symptomatic Holes

When a patient attends complaining of flashes of light, floaters, or loss of vision due to frank vitreous haemorrhage as a result of recent or continuing vitreous traction and a retinal hole is found, then the risk of the hole proceeding to detachment in this group is relatively high. Progression after prophylactic treatment has been shown, however, to be reduced to 6% (Robertson and Norton, 1973). Prophylactic treatment therefore of all symptomatic holes is advised.

The High-risk Eye

This is the group of patients in which the incidence of retinal detachment is known to be high and in which the chance of an individual retinal hole proceeding to detachment is greater than usual. The high risk conditions are those of aphakia particularly if there has been pre-existing myopia (Hyams et al., 1975), and of fellow eyes of patients with retinal detachment. The risk of detachment in aphakic fellow eyes is even greater (Benson et al., 1975). Prophylactic treatment is therefore recommended to all retinal holes in aphakic or fellow eyes.

Asymptomatic Holes

These holes are discovered at routine examination of the eye in non high-risk situations. They may be horseshoe-shaped or round holes with or without opercula and the absence of recent symptoms suggests that the hole is probably not fresh. Studies of these holes show a low incidence of progression to detachment: thus, none in 26 cases followed up from 3 to 9 years (Byer, 1967), 2% of 60 cases followed between 1 and 6 years (Neumann and Hyams, 1972), and none in 191 eyes followed between 1 and 6 years (Hyams et al., 1974).

Accordingly, prophylactic treatment is not advised for phakic asymptomatic holes unless they have exceptional characteristics, the natural history of which is not fully understood but is unlikely to be benign. These exceptions include retinal dialysis, and giant tears or large holes, particularly if the latter are not situated within the vitreous base itself and therefore not protected from vitreous traction.

Myopia

Specific comment is required regarding asymptomatic holes in myopia. According to studies available, the incidence of progression of these holes to retinal

detachment is low (Neumann and Hyams, 1972). However, the risk of detachment in myopia is well established. The apparent discrepancy can probably be explained by the fact that the majority of myopic patients who progress to detachment also have symptomatic holes which would in any case receive prophylactic treatment. The case for the non-progression of asymptomatic holes in myopia is not completely proven, and although prophylactic treatment of all small asymptomatic holes in this condition is not advised, treatment is usually advised for U-shaped tears. Other factors that may be important in considering prophylactic treatment in this group is the *degree* of myopia, the age of the patient, and the likelihood of being able to keep the patient under observation for a long period. Thus, high myopia and relative youth would be in favour of treatment whereas old age and a low degree of myopia would be factors in favour of conservatism.

Lattice Degeneration

Like retinal holes, the high incidence of lattice-like degenerations noted at autopsy (Straatsma and Allen, 1962) in eyes that have not proceeded to retinal detachment, and also on clinical examination in otherwise normal eyes (Byer, 1965), renders it impractical and unnecessary to treat all patients with this condition. However, true lattice degeneration, by leading to retinal hole formation, is a known precursor of retinal detachment and has been estimated to be a contributory factor in approximately 30% of retinal detachment cases. However, Byer (1974) observed a group of patients who had lattice degeneration in non-fellow asymptomatic eyes and found that, even if retinal holes were present, these cases did not progress to retinal detachment. This relatively benign and non-progressive tendency in the majority of cases has led to an increasingly conservative attitude in the management of patients with lattice degeneration. On the other hand, it was found that in a retinal detachment series (Tillery and Lucier, 1976) that 2.8% of all cases were caused by lattice degeneration associated with round holes that led to an insidious type of retinal detachment, which usually presented only when the macula had become detached. It has also been pointed out (Morse and Scheie, 1975) that when lattice degeneration is complicated by hole formation and is combined with myopia it is particularly dangerous. Treatment of lattice degeneration is therefore advised in the following high risk situations:

1. In the same or the other eye of a patient with retinal detachment.
2. When asymptomatic retinal holes are found and other high risk factors are present (e.g. aphakia).
3. If found in an eye prior to cataract extraction.

If a cataract operation is to be performed and a clear view of peripheral lattice degeneration can be achieved, then prophylactic treatment should be given before surgery because there is an added risk of detachment after cataract extraction in these patients (Morse, 1974). If the cataract is too dense to visualise the lattice lesion accurately, then prophylactic treatment should be carried out

as soon as possible after cataract extraction, which should include a broad iridectomy to facilitate a good view of the ocular fundus.

Even in patients not requiring treatment a regular watch should be kept on the lattice degenerative lesions. Occasionally these will be complicated by symptomatic retinal holes which will need to be treated in the usual way.

Snail-track Degeneration

This type of degeneration is usually associated with round holes and has a strong tendency to progress to retinal detachment (Aaberg and Stevens, 1972). Prophylactic treatment is therefore advised in all cases in which retinal holes are found but it should be confined to the areas that contain the holes. It does not appear necessary to treat all the degenerate areas, which are often extemely widespread.

Other Indications for Prophylaxis

Giant Tears

There is a high incidence of fellow eyes affected by giant tears in atraumatic cases, an incidence that has varied between 32% (Schepens and Freeman, 1967), 48% (Glasspool and Kanski, 1973) and 75% (Scott, 1976). The fellow eye of a patient with a giant tear may contain obvious lattice degenerative change or tear formation, but this is unusual. More often, areas of white with or without pressure or non-specific peripheral choroido-retinal degeneration are found, and sometimes the eye appears to be completely normal. Whatever the findings, 360° circumferential cryotherapy to the equator and pre-equatorial region is advised. Prophylactic encirclement with a silicone rubber band has been advised (Hudson et al., 1973) but this more complicated procedure does not seem to offer any advantage over simple cryotherapy.

To summarise, the indications for prophylactic treatment are as follows:

1. All symptomatic holes.
2. All holes in aphakic eyes.
3. All holes in fellow eyes.
4. Some asymptomatic holes (e.g. retinal dialysis).
5. Holes in snail-track degeneration.
6. Lattice degeneration in fellow eyes, aphakic eyes, some myopic eyes, and in eyes before cataract extraction, even if no holes are present.
7. The fellow eye of an atraumatic giant tear.

References

Aaberg, T.M.: Retinal detachment. Trans. Am. Acad. Ophthalmol. Otolaryngol. *81*, 402–405 (1976)

Aaberg, T.M., Stevens, T.R.: Snail track degeneration of the retina. Am. J. Ophthalmol. *73*, 370–376 (1972)

Abraham, T.K., Shea, M.: Cryopexy for improved results in the prophylaxis of retinal detachment Trans. Ophthalmol. Soc. U.K. *88*, 297–231 (1968)
Adams, G.L., Yeer, D., Hahn, P.M., Pearlman, J.T.: Effect of binocular and monocular patching on eye movements. Arch. Ophthalmol. *90*, 117–120 (1973)
Adams, S.T.: Retinal detachments due to macular and small posterior holes. Arch. Ophthalmol *66*, 528–533 (1961)
Banks, W.S.: Cryosurgery in the treatment of retinal detachment. J. Cryosurg. *2*, 30–42 (1969
Benson, W.E., Grand, M.G., Okun, E.: Aphakic retinal detachment. Arch. Ophthalmol. *93*, 245–249 (1975)
Bietti, G.: Chorio-retiniti adesive da crio-applicazioni episclerali. XIV. Concilium Opthalmologicium, Hispania, 1933
Bloch, D., O'Connor, P., Lincoff, H.A.: The mechanism of the cryosurgical adhesion. Am. J Ophthalmol. *71*, 666–673 (1971)
Bohringer, H.R.: Statistisches zu Häufigkeit und Risiko der Netzhautablösung. Ophthalmologica *131*, 331–334 (1956)
Boke, W., Custodis, E.: Technik in der Netzhautchirurgie. Klin. Monatsbl. Augenheilkd. *162* 147–159 (1973)
Byer, N.E.: Clinical study of lattice degeneration of the retina. Trans. Am. Acad. Ophthalmol Otolaryngol. *69*, 1064–1083 (1965)
Byer, N.E.: Clinical study of retinal breaks. Trans. Am. Acad. Ophthalmol. Otolaryngol. *71* 461–473 (1967)
Byer, N.E.: Prognosis of asymptomatic retinal breaks. Arch. Ophthalmol. *92*, 208–210 (1974)
Charamis, J., Theodossiadis, G.: Which peripheral retinal lesions should have peripheral treatment'. Ann. Ocul. *205*, 209–316 (1972)
Chawla, H.B.: Retinal detachment treated with encircling silastic sponge. Br. J. Ophthalmol. *55* 331–337 (1971)
Chawla, H.B., Birchall, C.H.: Intravitreal air in retinal detachment surgery. Br. J. Ophthalmol *57*, 60–70 (1973)
Chignell, A.H.: Retinal detachment surgery without drainage of subretinal fluid. Am. J. Ophthalmol *77*, 1–5 (1974)
Chignell, A.H.: Retinal mobility and retinal detachment surgery. Br. J. Ophthalmol. *61*, 446–449 (1977a)
Chignell, A.H.: Retinal detachment surgery without cryotherapy. Trans. Ophthalmol. Soc. U.K. *97*, 30–32 (1977b)
Chignell, A.H., Lean, J.S.: The effect of bed rest on depth of subretinal fluid. Unpublished data (1979)
Chignell, A.H., Markham, R.H.C.: Buckling procedures and drainage of subretinal fluid. Trans Ophthalmol. Soc. U.K. *97*, 474–477 (1978)
Chignell, A.H., Shilling, J.S.: Prophylaxis of retinal detachment. Br. J. Ophthalmol. *57*, 291–298 (1973)
Chignell, A.H., Fison, L.G., Davies, E.W.G., Hartley, R.E., Gundry, M.F.: Failure in retinal detachment surgery. Br. J. Ophthalmol. *57*, 525–530 (1973)
Cibis, P.A.: Errors in the management of retinal detachment surgery. Mod. Probl. Ophthalmol. *3*, 284–308 (1965)
Cibis, P.A., Becker, B., Okun, E., Canaan, S.: The use of silicone oil in retinal detachment surgery. Arch. Ophthalmol. *68*, 590–599 (1962)
Cockerham, W., Schepens, C.L., Freeman, H.M.: Silicone injection in retinal detachment. Mod. Probl. Ophthalmol. *8*, 525–540 (1969)
Colyear, B.H.: The initial operation in retinal detachment. Mod. Probl. Ophthalmol. *7*, 228–231 (1968)
Colyear, B.H., Pischel, D.K.: Preventative treatment of retinal detachment by means of light coagulation. Trans. Pac. Coast Otoophthalmol. Soc. *41*, 193–215 (1960)
Crick, M.D.P., Chignell, A.H!: Treatment of rhegmatogenous retinal detachment without apparent holes. Trans. Ophthalmol. Soc. U.K. *97*, 272–275 (1977)
Criswick, V.G., Brockhurst, R.J.: Retinal detachment. 360° scleral buckling as a primary procedure. Arch. Ophthalmol. *82*, 641–650 (1969)
Custodis, E.: Bedeutet die Plombenaufnahung und die Sklera einen Fortschritt in der operativen Behandlung der Netzhautablösung. Ber. Dtsch. Ophthalmol. Ges. *58*, 102–105 (1953)
Davis, M.D.: Natural history of retinal breaks without detachment. Arch. Ophthalmol. *92*, 183–194 (1974)

Davis, M.D., Segal, P.P., McCormack, A.: The natural course followed by the fellow eye in patients with rhegmatogenous retinal detachment. In: Retinal Congress. Pruett, R.C., Regan, C.D.J. (eds.), pp. 643–660. New York: Appleton Century Crofts 1974

Feman, S.S., Hepler, S.S., Straatsma, B.R.: Rhegmatogenous retinal detachment due to macular hole. Arch. Ophthalmol. *91*, 371–372 (1974)

Fineberg, E., Machemer, R., Sullivan, P., Norton, E.W.D., Hamasaki, D., Anderson, D.: Sulfur hexafluoride in owl monkey vitreous cavity. Am. J. Ophthalmol. *79*, 67–76 (1975)

Foos, R.Y.: Vitreoretinal juncture; epiretinal membranes and vitreous. Invest. Ophthalmol. *16*, 416–422 (1977)

Foos, R.Y., Allen, R.H.: Retinal tears and lesser lesions of the peripheral retina in autopsy eyes. Am. J. Ophthalmol. *64*, 643–655 (1967)

Foulds, W.S.: Experimental detachment of the retina and its effect on the intra-ocular fluid dynamics. Mod. Probl. Ophthalmol. *8*, 51–63 (1969)

Foulds, W.S.: Aetiology of retinal detachment. Trans. Ophthalmol. Soc. U.K. *95*, 118–128 (1975)

Foulds, W.S.: Clinical significance of trans-scleral fluid transfer. Trans. Ophthalmol. Soc. U.K. *96*, 290–309 (1976)

Freeman, H.M.: Current management of giant retinal breaks. Symp. of retina and retinal surgery, pp. 171–183. New Orleans: Acad. of Ophthalmol. C.V. Mosby 1969

Freeman, H.M., Hawkins, W.R., Schepens, C.L.: Anterior segment necrosis. Arch. Ophthalmol. *75*, 644–651 (1966)

Friedman, Z., Neumann, E., Hyams, S.: Vitreous and peripheral retina in aphakia. Br. J. Ophthalmol. *57*, 52–57 (1973)

Glasspool, M.G., Kanski, J.J.: Prophylaxis in giant tears. Trans. Ophthalmol. Soc. U.K. *93*, 363–371 (1973)

Grey, R.H.B., Leaver, P.K.: Results of silicone oil injection in massive preretinal retraction. Trans. Ophthalmol. Soc. U.K. *97*, 238–242 (1977)

Griffith, R.D., Ryan, E.A., Hilton, G.F.: Primary retinal detachments without apparent breaks. Am. J. Ophthalmol. *81*, 420–427 (1976)

Hagler, W.S., Ataraliya, V.: Macular pucker after retinal detachment surgery. Br. J. Ophthalmol. *55*, 441–457 (1971)

Halpern, J.I.: Routine screening of the retinal periphery. Am. J. Ophthalmol. *62*, 99–102 (1966)

Hofmann, H., Hanselmayer, H.: Retinal detachment – extent and frequency of redetachment through pre-operative immobilisation. Klin. Monatsbl. Augenheilkd. *162*, 178–185 (1973)

Huamonte, F.U., Peyman, G.A., Goldberg, M.F.: Complicated retinal detachment and its management with pars plana vitrectomy. Br. J. Ophthalmol. *61*, 754–761 (1977)

Hudson, J.R., Kanski, J.J., Elkington, A.R.: Prophylactic encirclement. Br. J. Ophthalmol. *57*, 531–536 (1973)

Hyams, S.W., Neumann, E.: Peripheral retina and myopia. Br. J. Ophthalmol. *53*, 300–306 (1969)

Hyams, S.W., Meir, E., Ivry, M., Krakowski, D., Barkai, S., Jedwab, E., Neumann, E.: Chorioretinal lesions predisposing to retinal detachment. Am. J. Ophthalmol. *78*, 429–437 (1974)

Hyams, S.W., Bialik, M., Neumann, E.: Myopia-Aphakia. Prevalence of retinal detachment. Br. J. Ophthalmol. *59*, 480–482 (1975)

Kanski, J.: Giant retinal tears. Am. J. Ophthalmol. *79*, 846–852 (1975)

Kirkconnell, W.S., Rubin, M.L.: Effect of cryopexy on the vitreous. Arch. Ophthalmol. *74*, 399–402 (1965)

Kloti, R.: An operating method for retinal detachment due to macular holes. Ophthalmologica *148*, 42–56 (1964)

Laqua, H., Machemer, R.: Repair and adhesion mechanism of the cryotherapy lesion in experimental retinal detachment. Am. J. Ophthalmol. *81*, 833–846 (1976)

Leaver, P.K., Cleary, P.E.: Macular holes and retinal detachment. Trans. Ophthalmol. Soc. U.K. *95*, 145–147 (1975)

Leaver, P.K., Chignell, A.H., Fison, L.G., Pyne, J.R., Saunders, S.H.: Role of non-drainage of subretinal fluid in reoperation for retinal detachment. Br. J. Ophthalmol. *75*, 644–650 (1966)

Lincoff, H.A.: The prophylactic treatment of retinal detachment. Arch. Ophthalmol. *66*, 48–60 (1961)

Lincoff, H.A.: The rationale for radial buckling. Mod. Probl. Ophthalmol. *12*, 484–491 (1974)

Lincoff, H.A., Gieser, R.: Finding the retinal hole. Arch. Ophthalmol. *85*, 565–569 (1971)

Lincoff, H.A., Kreissig, I.: The treatment of retinal detachment without drainage of subretinal fluid. Trans. Am. Acad. Ophthalmol. Otolaryngol. *76*, 1221–1233 (1972)

Lincoff, H.A., Kreissig, I.: Advantages of radial buckling. Am. J. Ophthalmol. *79*, 955–957 (1975a)
Lincoff, H.A., Kreissig, I.: The conservative management of vitreous haemorrhage. Trans. Am. Acad. Ophthalmol. Otolaryngol. *79*, 859–864 (1975b)
Lincoff, H.A., McLean, J.M.: Cryosurgical treatment of retinal detachment. Trans. Am. Acad. Ophthalmol. Otolaryngol. *70*, 202–211 (1960)
Lincoff, H.A., McLean, J.M.: Mechanism of the cryosurgical adhesion. Am. J. Ophthalmol. *67*, 477–493 (1969)
Lincoff, H.A., McLean, J.M., Nano, H.: Cryosurgical treatment of retinal detachment. Trans. Am. Acad. Ophthalmol. Otolaryngol. *68*, 412–432 (1964)
Lincoff, H.A., Baras, I., McLean, J.M.: Modifications to the Custodis procedure for retinal detachment. Arch. Ophthalmol. *73*, 160–163 (1965)
Lincoff, H.A., Long, R., Marquardt, J., McLean, J.M.: The cryosurgical adhesion. Trans. Am. Acad. Ophthalmol. Otolaryngol. *72*, 191–202 (1968)
Lincoff, H.A., Kreissig, I., Lafranco, F.: Large retinal tears. Am. J. Ophthalmol. *84*, 501–508 (1977)
Machemer, R.: Removal of pre-retinal membranes. Trans. Am. Acad. Ophthalmol. Otolaryngol. *81*, 420–425 (1976)
Machemer, R., Allen, A.W.: Retinal tears of 180° and greater. Arch. Ophthalmol. *94*, 1340–1346 (1976)
Merin, S., Feiler, V., Hyams, S., Ivry, M., Krakowski, D., Landau, L., Maythar, B., Michaelson, I.C., Scharf, J., Schul, A., Ser, I.: The fate of the fellow eye in retinal detachment. Am. J. Ophthalmol. *71*, 477–481 (1971)
Meyer-Schwickerath, G.: Light coagulation. St. Louis: Mosby 1960
Meyer-Schwickerath, G.: The prophylactic treatment of retinal detachment. Trans. Can. Ophthalmol. Soc. *26*, 9–15 (1964)
Morse, P.H.: Lattice degeneration of the retina and retinal detachment. Am. J. Ophthalmol. *78*, 930–934 (1974)
Morse, P.H., Scheie, H.G.: Rhegmatogenous retinal detachment in patients aged 40 or less. Trans. Ophthalmol. Soc. U.K. *95*, 129–133 (1975)
Mortimer, C.B.: The prevention of retinal detachment. Can. J. Ophthalmol. *1*, 206–212 (1966)
Mukai, N., Lee, P., Oguri, M., Schepens, C.L.: A long term evaluation of silicone retinopathy in monkeys. Can. J. Ophthalmol. *10*, 391–403 (1975)
Neumann, E., Hyams, J.: Conservative management of retinal breaks. Br. J. Ophthalmol. *56*, 482–486 (1972)
Norton, E.W.D.: Present status of cryotherapy in retinal detachment surgery. Trans. Am. Acad. Ophthalmol. Otolaryngol. *73*, 1029–1034 (1969)
Norton, E.W.D.: Intra-ocular gas in the management of selected retinal detachments. Trans. Am. Acad. Ophthalmol. Otolaryngol. *77*, 85–98 (1973)
Norton, E.W.D., Machemer, R.: A new approach to the treatment of selected retinal detachments secondary to vitreous loss at cataract surgery. Am. J. Ophthalmol. *72*, 705–707 (1971)
Norton, E.W.D., Aaberg, T.M., Fung, W., Curtin, V.T.: Giant retinal tears. Am. J. Ophthalmol. *68*, 1011–1021 (1969)
O'Connor, P.R.: External buckling without drainage for selected detachment in aphakic eyes. Am. J. Ophthalmol. *82*, 358–364 (1976)
Okun, E.: Gross and microscopic pathology in autopsy eyes. Retinal breaks without detachment. Am. J. Ophthalmol. *51*, 369–391 (1961)
Okun, E., Arribas, N., Johnston, G.P.: Therapy of retinal detachment complicated by massive preretinal fibroplasia. Mod. Probl. Ophthalmol. *8*, 563–571 (1969)
Pischel, D.K.: How to avoid complicated operations. Mod. Probl. Ophthalmol. *7*, 214–223 (1968)
Pruett, R.C.: The fishmouthing phenomenon. Arch. Ophthalmol. *95*, 1777–1787 (1977)
Ray, G.S., Vanheuven, W.A.J., Patel, D.: Gelatin implants in scleral buckling procedures. Arch. Ophthalmol. *93*, 799–802 (1975)
Robertson, D.M., Norton, E.W.D.: Long term follow up of treated retinal breaks. Am. J. Ophthalmol. *75*, 395–404 (1973)
Rosengren, B.: Results of treatment of detachment of the retina with diathermy and injection of air into the vitreous body. Acta Ophthalmol. (kbh) *16*, 573–579 (1938)
Rubinstein, K., Hayes, B.G.: Scleral indentation without resection. Br. J. Ophthalmol. *53*, 773–777 (1969)
Rutnin, V., Schepens, C.L.: Fundus appearance in normal eyes. IV. Retinal breaks and the findings. Am. J. Ophthalmol. *64*, 1063–1078 (1967)

Ryan, S.J., Goldberg, M.F.: Anterior segment ischaemia following scleral buckling in sickle cell haemaglobinopathy. Am. J. Ophthalmol. *72*, 35–50 (1971)
Schepens, C.L., Freeman, H.M.: Current management of giant retinal breaks. Trans. Am. Acad. Ophthalmol. Otolaryngol. *71*, 474–487 (1967)
Scott, J.D.: Retinal detachment surgery without drainage. Trans. Ophthalmol. Soc. U.K. *90*, 57–77 (1970)
Scott, J.D.: Static and dynamic vitreous traction. Trans. Ophthalmol. Soc. U.K. *91*, 175–188 (1971)
Scott, J.D.: Treatment of the detached immobile retina. Trans. Ophthalmol. Soc. U.K. *92*, 351–357 (1972)
Scott, J.D.: The treatment of massive vitreous retraction. Trans. Ophthalmol. Soc. U.K. *93*, 417–423 (1973)
Scott, J.D.: Macular holes and retinal detachment. Trans. Ophthalmol. Soc. U.K. *94*, 319–324 (1974)
Scott, J.D.: Equatorial giant tears affected by massive vitreous retraction. Trans. Ophthalmol. Soc. U.K. *96*, 309–312 (1976)
Scott, J.D.: A rationale for the use of liquid silicone. Trans. Ophthalmol. Soc. U.K. *97*, 235–237 (1977)
Straatsma, B.R., Allen, R.A.: Lattice degeneration of the retina. Trans. Am. Acad. Ophthalmol. Otolaryngol. *66*, 600–613 (1962)
Sugar, H.S., Okamura, I.D.: Ocular findings six years after intra-vitreal silicone injection. Arch. Ophthalmol. *94*, 612–615 (1976)
Tillery, W.V., Lucier, A.C.: Round atrophic holes in lattice degeneration. Trans. Am. Acad. Ophthalmol. Otolaryngol. *81*, 509–518 (1976)
Watzke, R.: Silicone retinopiesis for retinal detachment. Arch. Ophthalmol. *77*, 185–196 (1967)
Weidenthal, D.T.: Retinal reattachment without release of subretinal fluid. Am. J. Ophthalmol. *63*, 108–112 (1967)
Zauberman, H., Rosell, F.G.: Treatment of retinal detachment without inducing chorioretinal lesions. Trans. Am. Acad. Ophthalmol. Otolaryngol. *79*, 835–844 (1975)
Zollner, F.: Über die prophylaktische Behandlung der Ablatio Retinae durch Lichtkoagulation. Ber. Dtsch. Ophthalmol. Ges. *66*, 24–66 (1964)

Chapter 4

Surgical Details

Initial Dissection

Lateral Canthotomy

A lateral canthotomy may be performed when the palpebral fissure is too narrow to allow reasonable access for comfortable scleral dissection and placement of the buckle sutures. This is more likely to be difficult when an extremely posterior dissection is to be performed.

Corneal Clouding

In most cases the cornea may be left undisturbed throughout the operation although it is necessary to irrigate it regularly with saline. Occasionally, however, if the cornea becomes progressively cloudy, removal of the epithelium is necessary but this will result in considerable postoperative discomfort to the patient and should be performed only if essential.

Conjunctival Incision

A limbal incision of 1 to 2 mm from the limbus (King and Schepens, 1974) is used (Fig. 4.1). This incision running parallel to the limbus is extended to just beyond the extraocular muscle guarding the quadrant of sclera to be exposed.

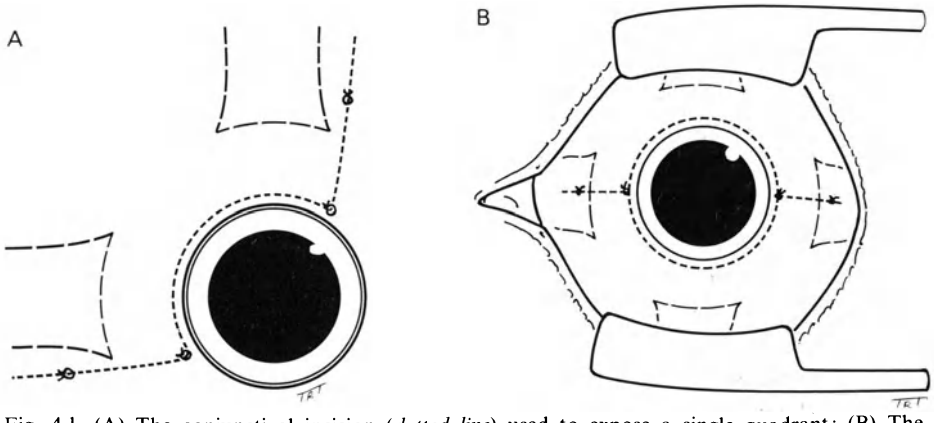

Fig. 4.1. (A) The conjunctival incision (*dotted line*) used to expose a single quadrant; (B) The incision used to expose all quadrants

A relieving incision is made at each end and this is extended backwards towards the fornix (Fig. 4.1 A). If all quadrants have to be exposed a 360° incision is made, with relieving incisions in the 3 and 9 o'clock positions (Fig. 4.1 B). The main advantage of the limbal incision is that it allows the simultaneous reflection of the conjunctival and tenons layers and at the end of operation these two layers are drawn forward together to provide a thick covering over the episcleral implants that are to be used. Occasionally, the more traditional incision 4 to 5 mm from the limbus will be necessary in reoperations if the limbal area is already seriously scarred and adherent, if there has been recent cataract surgery, or if a draining conjunctival bleb from glaucoma surgery has been left. These are best avoided completely by wide circumvention.

Isolation of Muscles and Exposure of Sclera

The rectus muscles on each side of the quadrant to be buckled are isolated by blunt dissection and tagged with 3.0 black silk sutures slipped underneath the muscles on an aneurysm needle. (If an encirclement operation is to be performed all four muscles are isolated.) Tagging allows easy rotation of the globe during operation. This dissection, so easily performed in the unoperated eye, becomes an arduous and sometimes hazardous procedure in eyes that have already been operated on. In these cases it is often useful to place preliminary traction sutures at the insertions of the muscle to allow initial rotation of the globe to facilitate exposure. Great care must be exercised in separating muscles from the underlying sclera which may have been markedly thinned by the previous use of diathermy. Unsuspected anterior staphylomas under muscles are an additional hazard and the forceful passage of squint hooks under muscles may result in accidental perforation of the globe. Vortex veins are also in jeopardy during the initial dissection since they may have been dragged forward by adhesions from previous operations. The inferior rectus muscle usually has the thickest adhesions, and the fine lateral rectus is the most difficult to identify. The inferior oblique muscle will usually be found to be strongly adherent to previous buckling materials in the inferior temporal quadrant.

The disinsertion of rectus muscles is now seldom necessary in retinal surgery. In all but the most difficult exposures scleral sutures may be manipulated beneath the muscles without detaching them from their insertions. Thus, not only will the blood supply to the anterior segment not be interrupted to any serious degree but also the risk of postoperative muscle imbalance, resulting in troublesome diplopia, will be avoided, a problem that is particularly relevant to the vertical rectus muscle. Muscles are removed only if tears are very posterior, e.g. removal of the lateral rectus to gain access to the macular region and, occasionally, when wide radial implants have to be placed immediately under muscles.

Having isolated and tagged the appropriate muscles, the field where the scleral buckle is to be raised is exposed. Previous implants encountered at this

stage should not be removed since by doing so unexpectedly weak sclera, or even choroid itself, may be exposed, introducing a risk of rupture to the globe and loss of subretinal fluid or vitreous. If previous buckles have to be removed for subsequent application of cryotherapy and for the localisation of holes, it is sometimes wise to pre-place mattress sutures (5/0 Dacron) to straddle the buckle and thus facilitate rapid closure of the affected area if necessary. Recent sclerotomy wounds (if identifiable) from which subretinal fluid has been drained should likewise be avoided. If the failure of a retinal detachment is directly related to the existing buckle, then readjustment or replacement of the buckle is necessary. If, however, a hole has been found in a site well away from the old buckling procedure then the previous implant is best left undisturbed. At this stage of the operation the surgeon will now be able to observe the sclera into which he is to place the buckling sutures. Scleral thinning and dehiscences in the sclera, either iatrogenic or as a naturally occurring phenomenon in the myopic eye, will be obvious.

Cryotherapy

Using a well-guarded retinal cryoprobe to avoid freezing the eye lids, cryotherapy is applied under ophthalmoscopic observation. Only rarely is it necessary to apply cryotherapy 'blind'. In addition to the treatment of obvious retinal holes and degenerate areas, cryotherapy may be of great value when the position of the retinal hole is in doubt. Freezing of suspicious areas of the retina will reveal the characteristic appearance of the retinal hole, the surrounding detached area freezing white to leave a central dark patch through which the choroid can be seen. However, if there is preretinal fibrosis or heaping up of retina into irregular folds, uneven freezing occurs so that holes are difficult to identify. The 'end point' of each cryotherapy application will vary; if during indentation

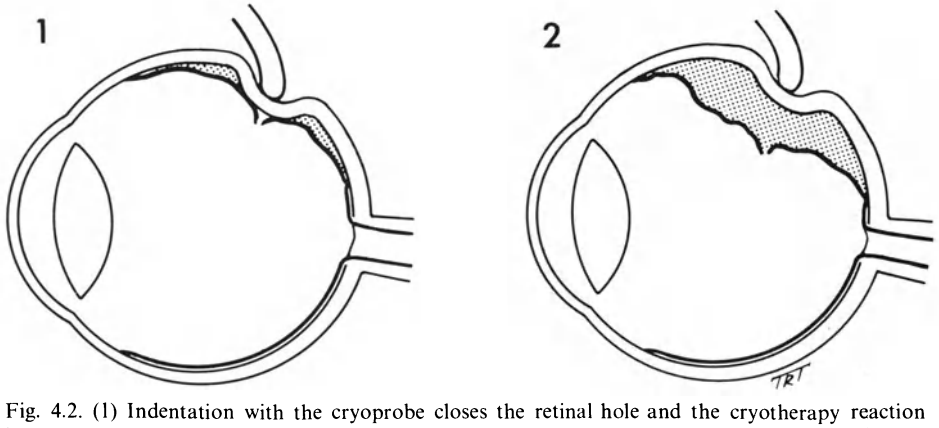

Fig. 4.2. (1) Indentation with the cryoprobe closes the retinal hole and the cryotherapy reaction is easy to see; (2) Indentation does not close the retinal hole if subretinal fluid is deep, and the cryotherapy reaction is difficult to see

with a cryoprobe the retinal hole can be actually closed, then the end point of the cryotherapy reaction may be taken as where the detached retina actually whitens, and this may be allowed to spread for a second or two from the initial freezing area. This ability to close the retinal hole on indentation, previously noted during the preoperative examination, also serves to indicate if the buckle that is to be raised will either close or nearly close the retinal hole on the table, thus often, allowing a non-drainage operation to be performed. If there is deep subretinal fluid between the probe and the detached retina, making it impossible to freeze the detached retina, the cryotherapy reaction is much more difficult to detect (Fig. 4.2), and there may also be no retinal hole through which to see the dull, glassy freezing reaction in pigment epithelium. In these cases, the pale glow of the cryotherapy reaction seen through detached retina must be taken as the end point of the cryotherapy application. Clinically, it does not seem to be necessary to freeze detached retina in order to achieve good adhesion, although it has been suggested that failure to do so will result in a less secure lesion (Laqua and Machemer, 1976). Failure to be able to see the cryotherapy reaction clearly often results in excessive cryotherapy through refreezing the same area or from excessively prolonged application.

A cryotherapy reaction should be confined to the retina immediately surrounding the retinal hole, and it is not necessary to cover all the retina supported by the scleral buckle. Cryotherapy in a case of retinal dialysis needs special attention. Reaction should involve treatment of retina posterior to the dialysis edge itself and also follow the dialysis edge to its insertion at the ora serrata. Gaps in cryotherapy application may result in a risk of communication between the dialysis and subretinal fluid behind the ridge (Chignell, 1973). This is a particular risk when the edge is highly elevated into the vitreous cavity because the retracted edge of retina will not return to the ora serrata after the buckling procedure. With giant tears, it is also advisable to extend the cryotherapy beyond the edge of the tear in order to minimise the risk of subsequent leakage due to extension of the tear.

It is unnecessary to refreeze the same area of retina more than once, although some degree of overlapping is bound to occur in order to obviate the risk of missing areas that should be treated. However, by careful observation of local landmarks refreezing can be kept to an absolute minimum, which again stresses the value of complete familiarity with the local anatomy around the retinal holes, something that can be achieved only by detailed preoperative examination. When mainly linear areas are to be treated, as in lattice degeneration, or in a row of small holes, the cryotherapy reaction should extend on both sides of the lesion and slightly beyond each end.

Operative Complications

Freezing of Eyelids

Inadvertent freezing of the lids due to inadequate insulation of the retinal cryoprobe may cause marked postoperative lid oedema, and in severe cases

actual burns may be seen. Although this does not lead to permanent damage, it can greatly increase discomfort in the postoperative period.

Scleral Rupture

After cryotherapy has been applied, the scleral ice ball that has formed, joining the cryprobe to the eye, must be allowed to melt, and the temptation to crack the probe off the side of the globe before the ice ball has melted must be resisted. Such cracking may result in scleral rupture. This is more likely to occur if the sclera is thin. Similarly, if cryotherapy has been applied through extraocular muscles, there may be partial avulsion of muscle.

Vitreous and Choroidal Haemorrhage

Indentation and freezing underneath the retinal hole, particularly a retinal hole that has a prominent vessel running in the operculum, may result in haemorrhage from the vessel. Although alarming, these haemorrhages are rarely severe. Blood from the vessel will tend to cascade down the back of the detached posterior vitreous and settle at the posterior pole of the eye. Small dot retinal haemorrhages are sometimes seen on the surface of the detached retina after cryotherapy but they do not seem to be of any particular significance. Choroidal haemorrhage may occur if cryotherapy is being applied in the region of the vortex veins in the equatorial region. These haemorrhages are rare but are occasionally severe. Vortex veins themselves may also be damaged as they emerge from the globe, resulting in extraocular haemorrhage.

Serous Choroidal Detachment

This rare event appears a few minutes after cryotherapy has been applied and may be associated with some degree of choroidal haemorrhage. The serous effusion, which may become bullous, usually occurs after cryotherapy in the region of the vortex veins although the actual mechanism of effusions is not fully understood. Again, although alarming, provided they are not attended by extensive choroidal haemorrhage, they will usually allow the surgeon to proceed and perform a normal retinal detachment operation. They will disappear spontaneously in the postoperative period.

Pigment Fallout

Pigment fallout occurs from excessive cryotherapy (Sudarsky and Yannuzzi, 1970; Abraham and Shea, 1968) and is due either to gross overfreezing or to repeated freezing of the pigment epithelium (Chignell et al., 1971). The evidence for overfreezing in these cases was the appearance of an unusually dense, white scar in the postoperative period caused by the complete destruction of pigment epithelium and choriocapillaris, which left only major choroidal vessels

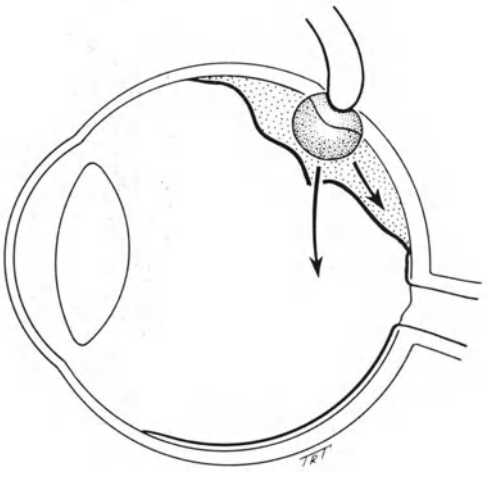

Fig. 4.3. Pigment fallout from excessive cryotherapy spreads into the subretinal space or into the vitreous cavity

intact. Heavy cryotherapy causes the pigment epithelium to explode and to release into the subretinal space pigment granules which are deposited at the most dependent part of the detachment; when the patient is supine at operation this is usually at the posterior pole of the eye. Occasionally, pigment passes directly through the retinal hole into the vitreous and is deposited on the optic disc (Fig. 4.3). Although pigment fallout is a dramatic sight at operation, or in the postoperative period when it is fully revealed as the subretinal fluid is absorbed, it does not seem to jeopardise visual acuity even if it occurs in the paramacular region (Hilton, 1974). It may, however, be possible to detect a relative scotoma at a later date (Abraham and Shea, 1969). In cases in which subretinal fluid is to be drained following excessive cryotherapy, pigment granules may be seen to emerge from the subretinal fluid as it flows from the eye.

Although excessive cryotherapy results in an extremely thin atrophic scar there is no apparent tendency for retinal holes to occur on the edge of such scars.

Localisation of Retinal Holes

Retinal holes are localised by using a modified scleral depressor (Gass, 1966) which when indented vertically on to the sclera leaves a small circular mark. This mark is then gently touched with a methylene-blue marking pencil, which leaves an easily indentifiable spot on the sclera (Fig. 4.4). The depressor itself must be sharp enough to mark the sclera without too much pressure having to be applied to it, but not so sharp as to perforate the sclera if it is thin. If the retinal hole is large, the extremities

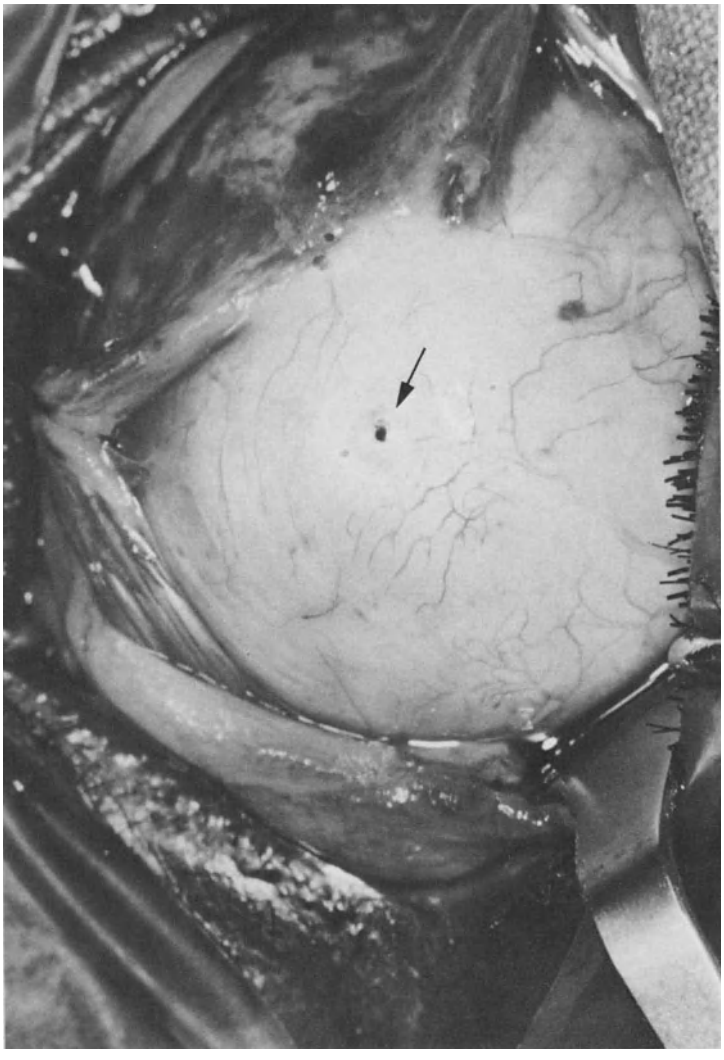

Fig. 4.4. Circular mark on sclera left by scleral depressor (*arrow*) and the identifying spot of methylene blue

of the hole must be localised by making as many separate markings as are considered necessary. In large horseshoe-shaped tears this usually takes the form of one mark at the posterior limit and one each on both of the lateral anterior extremities. In dialyses, two anterior marks are made at each end and one mark at the mid point of its posterior edge. This method of marking may not be suitable in all cases. In reoperations, the scleral surface is uneven and may not be easily marked. Also, if the eye is somewhat enophthalmic and the hole is posterior, some difficulty may be found in rotating the scleral depressor so that a mark can be made on the sclera. In these conditions it is easy to apply undue force to the tip of the depressor and risk perforation.

When the scleral depressor is not satisfactory the localisation can be made by using an episcleral stitch, holding the stitch in forceps that are used as a scleral depressor (Fison, 1975). This latter method, however, although accurate is time consuming. The markings of the retinal hole provide a further check for the proposed size of the buckle and, if necessary, the distance between the marks can be measured so that the correct width and length of the buckle can be selected.

After the retinal holes have been localised the proposed site of drainage of subretinal fluid is similarly indicated with a scleral mark. This reassessment of the subretinal fluid at the time of operation will avoid unnecessary failure in evacuating fluid, particularly if there has been a change from the preoperative picture; after the patient has been supine for a time during the operative procedure, fluid may have shifted and tracked more posteriorly to leave a relatively shallow peripheral detachment.

Buckle Sutures

Placement of Buckle Sutures

When placing the suture, the sclera is made firm and stationary by counter traction with non-tooth forceps on an adjacent rectus muscle insertion. This movement will also tend to straighten the sclera so that the normal curvature of the globe is less pronounced, making for easier passage of the needle. The needle should assume its running depth as soon as possible, without a long entry or exit course before the depth is reached. This will prevent weakness

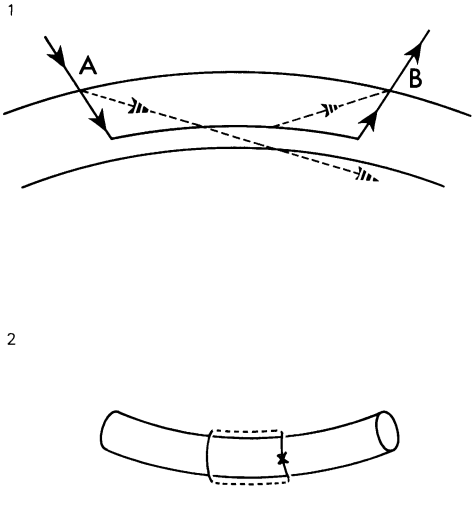

Fig. 4.5. (1) intrascleral course of the needle should follow the unbroken line from *A* to *B*. The interrupted line shows two suture defects; one resulting in a shelving suture with tendency to cut out when tied, and the other in accidental penetration of the globe; (2) small mattress suture used to anchor silicone rubber bands

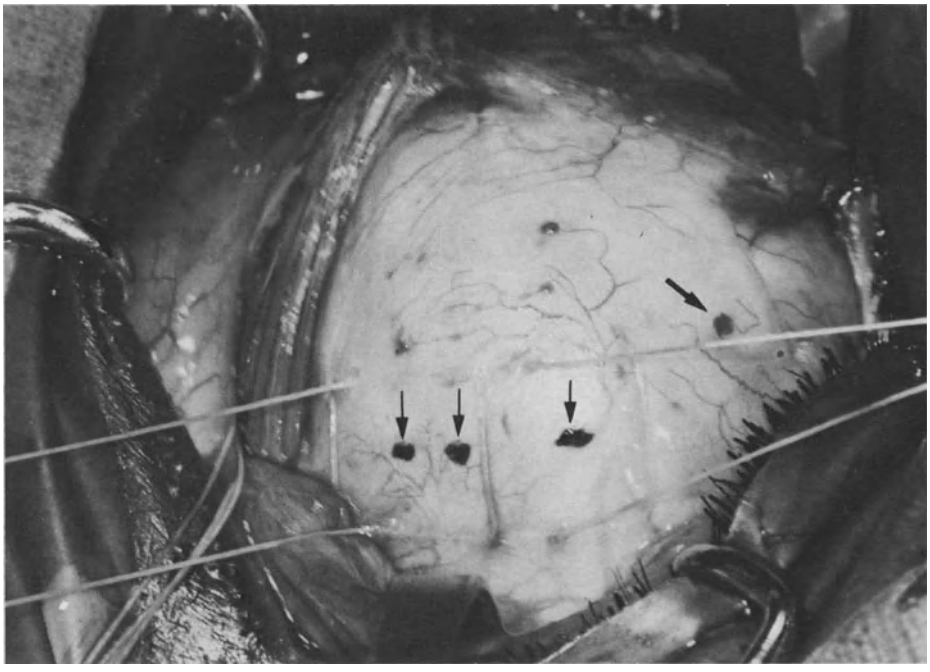

Fig. 4.6. Three holes have been localised (*thin arrows*) and two mattress sutures placed in position – the limbs of the sutures equidistant from the scleral marks. The intended site for SRF drainage has also been localised and marked (*thick arrow*)

at one or both ends of the suture track and a tendency for the suture to cut out when tied under tension. The placement of these sutures is a matter of considerable judgement; too deep a suture will result in accidental perforation with subsequent release of subretinal fluid, and too shallow a suture will result in cutting out when the suture is tied (Fig. 4.5; see also Fig. 4.10). The sutures are arranged in mattress fashion to straddle the intended implant (Fig. 4.6) and, until tied, are provisionally held in bulldog clips (it is convenient to have these individually marked so that rapid identification can be achieved later). In sclera of normal thickness the suture should run at approximately one-half to two-thirds of the scleral thickness in depth. This will result in the suture just being detectable in its intrascleral course, although this appearance will vary with the thickness of the sclera and will be much more clearly visible when the sclera is thin.

During suturing, the vortex veins must be carefully avoided and they are in jeopardy in the following ways.

1. Their intrascleral course may be damaged directly by the needle as it passes through the sclera.

2. As the needle is actually removed from the sclera, difficulty in retrieving it may be encountered in posterior sutures and the tip of the needle may damage vortex veins during the manoeuvre.

3. As the suture is pulled from the sclera the vortex veins, which are adherent to the loose periocular fascia, may be dragged into the suture track by the thread.

If the needle is nearing a tributory of the vortex vein it is removed from the sclera at this point – which leaves a gap in the suture track – passed over the vortex vein, and reintroduced into the sclera on the other side of the vein. A similar technique is employed when the sclera is thin, the surgeon being able to take only small bites of the more healthy sclera where it can be found. When radial sponges are used to seal posterior holes, the most posterior suture is often difficult to place; its placement is easier to effect by using a double-ended suture and allowing both needles to be passed, in turn, in an anterior-posterior direction.

During placement of the scleral sutures the subretinal space may be accidentally entered with release of subretinal fluid. However, due to the very small puncture site, incarceration of the retina and vitreous loss will rarely occur unless the performation has occurred over flat retina. If accidental drainage occurs, the perforating suture should be removed and the leak of subretinal fluid sealed by oversewing with a more widely placed suture. Accidental perforation will be a particular problem if the pressure in the eye has already been slightly elevated by the tightening of previously placed buckle sutures. In these cases release of subretinal fluid will be rapid and the eye will become hypotonic quickly. The pressure must be restored by tightening the buckle sutures as soon as possible after the offending stitch has been removed.

Episcleral Sponges

The most satisfactory sutures are 5/0 Dacron on a spatulated quarter or half-circle needle (manufactured by Davis and Geck). In most cases the quarter-circle needle is better because it is much easier to get a longer scleral bite with it. However, the half-circle needle is useful when access to the dissection site is difficult (e.g. under muscle or in posterior position). When placing the sutures the intrascleral course of the suture should be as long as possible (5 mm or so), reducing the risk of the sutures cutting out when they are tightened during operation or in the postoperative period. If sutures cut out they contribute to extrusion of the sponge. The separation of the limbs of the sutures will vary according to the width of the implant being used. A good general rule is to place the sutures approximately half as much apart as the width of the implant. Thus, for a 4 mm implant the limbs of the sutures are placed 6 mm apart, for a 5 mm implant 8 mm apart, and for a 7 mm implant 10 mm apart. It should be borne in mind that increasing the distance between the limbs of the sutures for any implant will not increase the width of the resulting buckle but only the height. A small gap of approximately 1 mm is left between the end of one mattress suture and the beginning of another. In general, it is found that most radial sponges will require two sutures, the number required when a circumferential implant is used depending very much on the extent

of the buckling procedure needed; usually three sutures are required per quadrant of retina buckled.

Encircling Bands

Small mattress sutures are used for gently tethering the episcleral band in the four quadrants, and the width apart of the limbs of these sutures should be just greater than that of the band used (see Fig. 4.5). The suture should not be so tied that the suture material comes to lie between the band and the sclera because this may result in subsequent erosion inwards of the suture due to pressure from the band. If the band is to be placed anterior to the equator in any quadrant, it is often necessary to include two anchoring sutures in the same quadrant as there is some tendency for the strap to bow-string forward between sutures if they are too widely separated. The site of the encircling band will have been localised in the four quadrants as previously described for retinal holes. If the encircling band is being used to relieve vitreo-retinal traction it will generally be found to be equatorial in its distribution and be lying approximately 12 mm. from the limbus. If on the other hand the band is being used mainly to seal visible or suspected retinal holes then the band must be positioned so that these areas will come to rest just on the anterior slope of the indent produced. This technique is suitable only for small holes, and in most cases when there are retinal holes the encircling band is augmented by either radial episcleral sponges or by a underlying silicone rubber gutter if a longer segment of sclera needs additional buckling. The ends of the encircling band are gripped lightly by means of a Watzke sleeve of silicone rubber (Fig. 4.7) which allows for easy adjustment in the tension of the band. It is convenient not to tie the strap in the same quadrant in which there is to be an additional

Fig. 4.7. The Watzke sleeve (*arrow*) is stretched in cross-action forceps and allows the two ends of the silicone band to be passed through

implant and, in general, it will be found that the lower temporal quadrant allowing good exposure is a convenient site.

Combining Local and Encircling Procedures

An encirclement procedure is not often performed without the addition of episcleral sponges (Fig. 4.8). Thus, the encircling ridge can be heightened in places in order to seal retinal holes, and this will obviate the need for tightening the encircling element excessively. A small local heightening of the encircling strap may be achieved simply by oversewing the strap with a mattress suture. This technique is not suitable, however, in the majority of cases. When combined techniques are used, the usual arrangement is that of a radial implant with an overlying silicone rubber band, or that of the rubber band with an underlying silicone rubber gutter. If a sponge implant is to be used the band is tethered on each side of the implant and the resulting buckles from such an arrangement result in an even and continuous internal ridge with no intervening 'dead ground' of unbuckled retina on either side of the local implant.

When a combined local sponge and encircling strap operation is to be carried out, the local implant sutures are placed first, as some difficulty may be found in placing them if the encircling element is already in position. The episcleral band is then passed round the globe, secured by its anchoring sutures, and

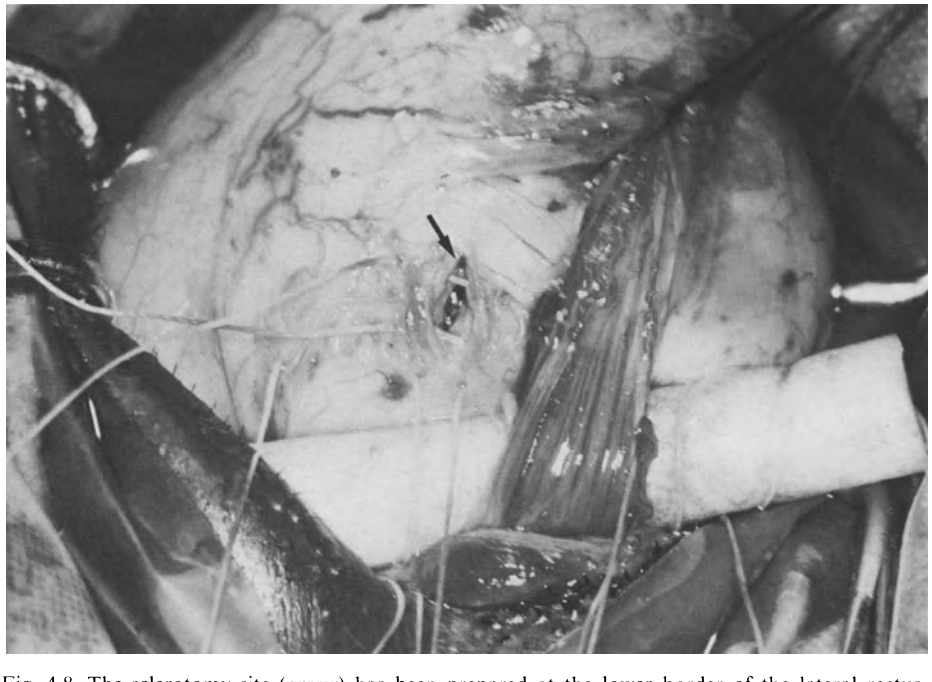

Fig. 4.8. The sclerotomy site (*arrow*) has been prepared at the lower border of the lateral rectus muscle. There is good exposure of the choroidal knuckle. A mattress suture has been placed across the incision to enable ease of closure

then lightly tied with a Watzke sleeve. The episcleral sponge is then placed in position and its sutures tightened if drainage is not going to be employed. If the eye is to be drained, no sutures are tightened until after this has been done.

Handling the Sponge

During the operation the sponge should not be removed from its sterile container until just before it is needed. This reduces the risk of its becoming contaminated before it is introduced to the eye. Prior to its placement in the eye it is soaked in gentamicin and during manipulation on the sclera it is held in non-toothed forceps to avoid damage and disruption to its cellular structure.

Drainage of Subretinal Fluid

If subretinal fluid is to be drained from the eye it is done after cryotherapy, localisation of retinal holes, and the placement of the buckling sutures.

Selecting the Site

The selection of the site for drainage of subretinal fluid, which is usually possible at the time of the preoperative examination, is made according to the following criteria.

1. Where there is a good depth of subretinal fluid.
2. Preferably in the lower half of the globe because in the event of haemorrhage blood will tend to track away from the macula if the patient is sat upright in the postoperative period.
3. If possible, drainage of subretinal fluid should not be through areas that have just been treated with cryotherapy, as this results in vasodilatation of the underlying choroid and an increase in the risk of haemorrhage.
4. Major choroidal vessels must be avoided.
5. Drainage should not be performed in the vicinity of large holes for fear of losing vitreous through the hole, with subsequent incarceration of formed gel and risk of traction band formation.
6. Sites of cryotherapy from previous operations from which retina has not detached must be avoided since vitreous will be lost if such sites are inadvertently punctured.
7. If possible, drainage of subretinal fluid should not be carried out posterior to the buckle because if complications arise they will be more difficult to deal with.
8. It is safe to drain under fibrotic detached retina because such fibrosis will have resulted in immobility of the retina, which tends to keep the retina splinted away from the drainage site.

9. Excessively thick sclera should be avoided because adequate exposure of the choroid is difficult to achieve.

10. Avoid placing the sclerotomy site directly underneath a silastic sponge as it will be very difficult to reopen the sclerotomy site if this is required after the sponge has been sutured into position.

In general, it will be found that one of the best sites for draining subretinal fluid is at the lower border of the lateral rectus muscle. Exposure is easy and the sclera in this site thin, which allows easy access to the choroid and examination of the choroidal knuckle by transillumination. Conversely, drainage on the nasal side of the globe is more difficult.

Technique of Drainage

A radial incision is made at the selected site. The incision should be long enough to allow a good view of the underlying choroid (approximately 2 mm in length) and the sclera is gently dissected until the knuckle of choroid is exposed (Fig. 4.8). At this point a mattress suture is placed across the lips of the wound, which may be elevated and separated by gently traction on the suture. The drainage site is then transilluminated by shining a light in through the pupil via the cornea (indirect ophthalmoscopy, or fibreoptic light is suitable). Transillumination is performed to see whether there are any large choroidal blood vessels crossing the knuckle of choroid that is to be perforated. If there are, or if during the preparation of the drainage site there has been spontaneous external bleeding from the choroid, then the wound is secured and a further site selected. If no blood vessels can be seen the choroidal knuckle is then gently cauterised to discourage subsequent bleeding and during the course of the cauterisation subretinal fluid is usually released. Application of cautery can be difficult if there is inadequate exposure of the choroid. If subretinal fluid is not released after the cautery of the choroid, it is often difficult to appreciate whether the subretinal space is being approached or not, and in this situation no further cauterisation should take place since it may necessitate the cautery being pushed deeper into the scleral wound, when direct visualisation of the tip of the cautery could be lost. This can result in sudden perforation and risk damage to the retina. If cautery has not resulted in release of subretinal fluid, the subretinal space is better entered by simple perforation with the tip of a sharp needle.

In recent detachments, the subretinal fluid will be watery and have a colourless appearance, whereas in long-standing cases it will be much thicker and xanthrochromic. Pigment particles resulting from cryotherapy or in cases in which there is advanced periretinal membrane formation with metaplastic activity of pigment epithelium may also seem to emerge in the stream of subretinal fluid. The amount of subretinal fluid to be drained depends on the reasons for drainage. Sometimes only a small quantity of fluid need be released (e.g.

to allow the optic disc to be seen). At other times as much fluid as possible has to be withdrawn (e.g. in cases of marked intraocular fibrosis), to allow high buckles to be raised or to allow intraocular injections to be performed.

Complications

Cessation of Flow

Flow of subretinal fluid may cease abruptly, and if it does, the surgeon should check the intraocular situation. If it is judged that enough drainage of subretinal fluid has been achieved, then the drainage site can be closed and the buckle sutures tightened. On the other hand, it may be that the flow of fluid has stopped before evacuation is adequate. Indirect ophthalmoscopy will show whether or not there is any depth of subretinal fluid at the drainage site. If in fact the retina appears to be flattened or very nearly so, then it is wise to drain more fluid from another site. If, on the other hand, there appears to be a reasonable depth of subretinal fluid overlying the drainage site, then the flow of fluid may be started again by gently manipulating the lips of the wound. Sometimes, simple separation and elevation of the lips of the wound with two pairs of forceps will encourage flow by keeping retina away from the site, and it can also be encouraged by holding one lip of the wound in a pair of forceps and applying gentle pressure to another quadrant of the globe. Occasionally, the choroid itself appears to act in a flap-like way, tending to shut off the flow of subretinal fluid, and this may necessitate reintroduction of the needle to promote further evacuation.

Choroidal Haemorrhage

Choroidal haemorrhage is the most important and dangerous complication of retinal detachment surgery and it may occur directly, at the time of perforation, and release subretinal fluid, or after fluid has been drained. The first instance is usually caused by direct perforation of a choroidal blood vessel, and the second probably as a result of hypotony or manipulation of the globe subsequent to the release of fluid. Occasionally, choroidal haemorrhage will begin before the subretinal space has in fact been entered. Under these circumstances blood will not enter the subretinal space and will emerge through the sclerotomy hole. When this happens it is safe to close the site and perform drainage in another position. If the subretinal space has been entered the extent to which choroidal haemorrhage will spread internally is variable. Sometimes this is only slight, most of the haemorrhage emerging as part of the stream of subretinal fluid. The blood that has entered the eye will be seen to produce a blackish reflex in the subretinal space, or red if it has gained the vitreous cavity via the retinal hole. In severe cases haemorrhage will be extensive and the globe, softened by the drainage of subretinal fluid, will suddenly become harder. Once

choroidal haemorrhage has been detected it is advisable to tighten the buckle sutures as quickly as possible to promote rapid restoration of normal or even raised intraocular pressure to discourage further bleeding. If, however, the intraocular haemorrhage has been severe enough to raise the intraocular pressure in its own right, tightening of the buckle sutures may be impossible. In this event some evacuation of the haemorrhage may be achieved via the drainage site itself as this may at least allow the buckle sutures to be tightened.

Retinal Haemorrhage

Occasionally, severe haemorrhage will result from direct trauma by the perforating needle or cautery to the underlying retinal blood vessels. Such trauma will also usually result in the formation of jagged iatrogenic retinal tears.

Retinal Incarceration

The retina may become incarcerated in the drainage site in a number of different ways.

If a site where the retina is in fact flat is chosen for drainage the perforation of the choroid will therefore occur coincidentally with that of the retina, with resultant loss of vitreous at the time the globe is perforated.

If the subretinal fluid is very shallow at the site of drainage, retina may become incarcerated after the flow of subretinal fluid has started. It is not usually possible to release the retina from such an incarceration, and unless the incarceration is recognised by ophthalmoscopy further manipulation or probing of the sclerotomy site with a needle may produce perforation of the retina and a flow of vitreous.

If a normal evacuation of subretinal fluid has occurred, a subsequent rise of intraocular pressure from tightening of buckle sutures may result in incarcer-

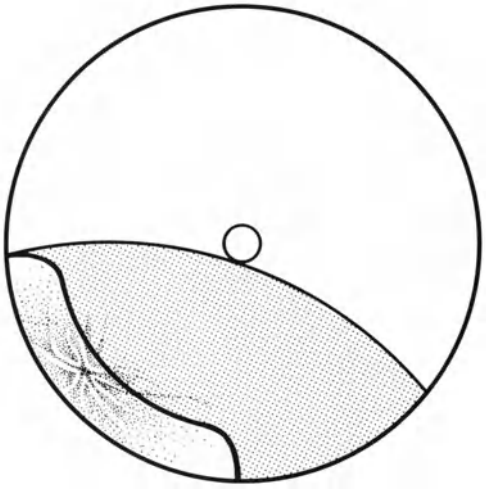

Fig. 4.9. Incarceration of the retina is seen as a stellate puckering of retina into the drainage site. The incarceration should be supported by a buckle

ation if the sclerotomy site has inadvertently been left open or has been poorly secured.

The occurrence of a retinal incarceration is usually easily recognised; the retina has a thin greyish appearance at the sclerotomy site, and on ophthalmoscopy the typical stellate formation of retina can be seen with the centre at the sclerotomy site itself (Fig. 4.9). Such an appearance may be further accentuated if vitreous, too, has been incarcerated and lost.

If incarceration occurs, no attempt should be made to disengage the retina, as this may produce iatrogenic retinal tears. The incarceration should be supported by a buckle, to counteract inevitable traction at the site.

Tightening the Buckle Sutures

As soon as drainage of subretinal fluid has been deemed adequate the sclerotomy site is closed with a temporary knot and the buckle sutures are tightened. Undue delay in tightening the sutures may contribute to choroidal haemorrhage by prolonging hypotony. If a local sponge implant has been used in association with an encircling band the local buckle sutures are tightened first, followed by tightening of the strap. If the strap has been used in combination with a gutter the encircling band is tightened first followed by those buckling the gutter. During tightening of the buckle sutures or of the encircling band, it may be decided that the drainage of subretinal fluid has not been adequate. More fluid may be allowed to escape from the eye by reopening the sclerotomy wound, but this must not be done before the tension of the buckle sutures is released and the eye is softened, since an elevated pressure will encourage retinal incarceration into the sclerotomy when drainage is restarted.

When a non-drainage operation is performed the technique of tightening the buckle sutures is different and more difficult because the eye (particularly the non-myopic eye) offers much greater resistance to being buckled and, apart from a tendency for poorly placed sutures to cut out (Fig. 4.10), various other problems may be encountered. The rise of intraocular pressure may prejudice the retinal circulation and the vessels of the optic disc must be carefully watched for patency as soon as the buckle sutures have been tightened to any degree. These sutures are best secured with temporary knots (a single tucked reef knot) which can be released easily in the event of the central retinal artery being occluded.

Sometimes several minutes may elapse after the tying of one suture, to allow the intraocular pressure to lower itself before a further suture can be tightened. Gentle massage on the side of the globe may encourage a more speedy reduction of the intraocular pressure, the reduction of which is assisted by administering intravenous Diamox (500 mg) at the beginning of the operation. The rapid rise of intraocular pressure that occurs as each suture is tightened

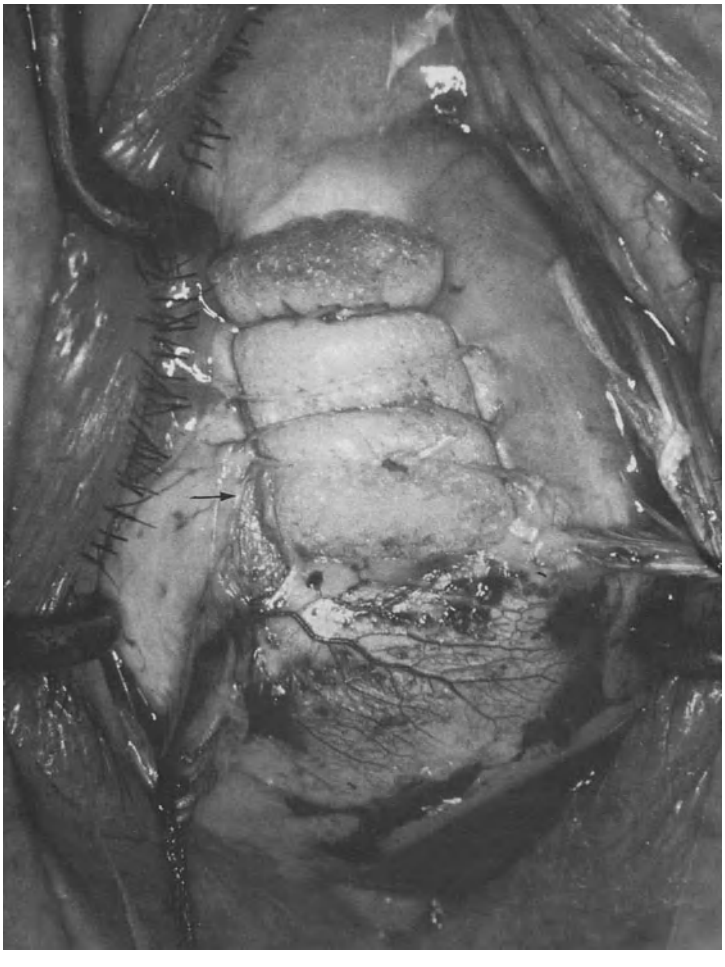

Fig. 4.10. A radial sponge has been sutured in position. The suturing is uneven and due to poor technique the anterior suture has partially cut out from the sclera (*arrow*)

may contribute to the production of transient corneal oedema, particularly in older patients. The oedema usually clears as the intraocular pressure falls and is seldom severe enough to warrant removal of the corneal epithelium.

After the sponge sutures have been tightened, attention is then turned to the encircling band, if one is to be used. In the non-drainage operation there is usually some delay while the eye is restored to a normal intraocular pressure before any attempt at tightening the band can usefully be made. In order to produce an internal indentation of approximately 2 mm the encircling band must be shortened by approximately 15% of its circumference. This works out at approximately 12 mm which is not markedly altered by variations in the ocular size because the changes in circumferential shortening in these ranges is very small (Lincoff et al., 1976). Accurate measurement to the nearest millimetre when shortening the band is difficult, particularly as the initial resting

point of the band around the globe is hard to establish accurately. One method in use is to tie two sutures on each end of the encircling band, and as the band is tightened the increasing distance between the two sutures can be measured. It is important to attempt measurement in the non-drainage operation as the band will not achieve its maximum indentation until the intraocular pressure is restored to normal, which means that its final height is not seen at the time of surgery. This is likely to be a problem when the retina is totally detached and the scleral buckle can only be faintly discernable through the subretinal fluid. When there is some flat retina, however, the effect of tightening the buckle may best be observed in this flat retina and at least some indentation of the buckle can be seen at the time of surgery. The tightening of the band will result in a rise of intraocular pressure in a non-drainage procedure and it is often necessary to wait several minutes in between each few millimetres of tightening before the desired shortening of the band has been achieved. Measurement of the amount by which the encirclement is tightened is not necessary when subretinal fluid has been drained because the height of the buckle is readily seen at the time of surgery. When the eye is soft, however, great care must be taken not to over-constrict the eye with an encirclement procedure. This is likely when hypotony is present as there is a tendency to restore the intraocular pressure by excessive tightening of the band. After the encirclement element has been tied, the Watzke sleeve is further reinforced by two additional sutures on each side of the sleeve.

Problems in Placing the Buckle

Care taken with the initial localisation of the retinal holes and appropriate selection of the size and direction of the implant to be used will be repaid by the rapid production of a buckle of satisfactory height and width accurately located under the retinal holes. A buckle that has been poorly placed or is of inadequate width will have to be repositioned or entirely replaced. This will involve replacing all the scleral buckling sutures. In the non-drainage operation this can usually be done without much difficutly but if drainage has been effected then the surgeon will usually be suturing into a much softened globe. This is not only technically difficult, but additional manipulation of such a globe may contribute to the precipitation of choroidal haemorrhage.

If the buckle is correctly positioned and of appropriate dimensions but its height is too low the latter may be increased simply by oversewing the buckle with more widely placed mattress sutures.

Fishmouthing of Holes

This phenomenon arises as a result of the sudden formation of a radial fold when a circumferential implant is being used, or when preoperative radial folding is accentuated, usually as a result of draining off subretinal fluid. These folds are best avoided by radial buckling (Pruett, 1977).

Retinal Dialysis

In cases of retinal dialysis the anterior ends of the circumferential buckle should be extended to the ora serrata so that no anterior leakage of subretinal fluid will occur. The retracted edge of a long-standing dialysis often extends posteriorly and will not fall back into place at the ora serrata as subretinal fluid is absorbed, or released hence the buckle will need to be arched backwards so that the centre of the implant corresponds with the edge of the dialysis.

Giant Tears

The circumferential buckle is sutured into position to produce a shallow indentation at a point just posterior to where the posterior flap has come to lie after it has been pressed into position by the use of intraocular air or gas. As in a dialysis, the ends of the buckle are brought round and extended to the ora serrata. This, too, will tend to prevent extension to the giant tear or leakage of subretinal fluid at its ends. Occasionally, separate radial implants may be necessary to effect such a closure if the tear has extended posteriorly at its extremities.

Special Surgical Procedures

Intraocular Injection

Air or Gas (SF6)

An intraocular injection of air or gas performed after the drainage of subretinal fluid and usually after the buckle sutures have been tightened (with the exception of when air is being used to manipulate a giant retinal tear) is usually most conveniently performed on the temporal side of the globe as this allows for easy access of syringe and needle. A line opposite the lower border of the lateral rectus is a suitable one. The surgeon is sited opposite the injection site, hence, the needle is introduced towards him, and the injection is made 4 to 5 mm from the limbus with a 5 ml syringe and with a sharp-pointed 25 gauge needle (25 mm long). A small preliminary scratch incision through about half the thickness of sclera will facilitate the introduction of the needle, particularly when the eye is soft.

The air or gas (sterilised by aspirating through a 0.22 millipore filter and contained in a sterile freely running dry syringe) is introduced through the incision, with counter traction on the lips of the incision or from the neighbouring lateral rectus insertion, and is pushed through the pars plana and basal gel in a backward direction towards the centre of the vitreous body to avoid damage to the posterior lens capsule. The tip of the needle, guided by the

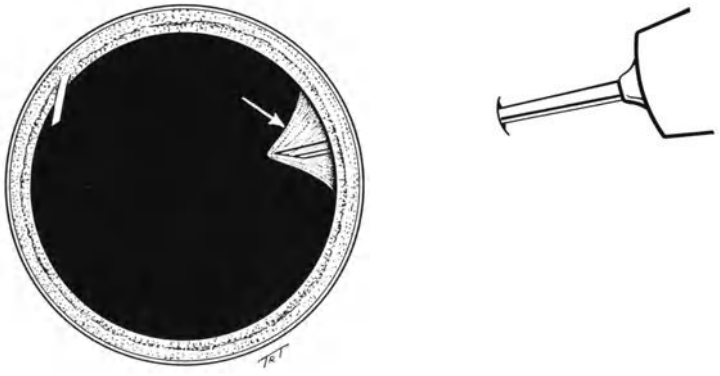

Fig. 4.11. Air is to be injected into the vitreous cavity, but the needle has not yet reached the vitreous cavity and is tenting up non-pigmented pars plana epithelium (*arrow*)

grip of the surgeon on the lower part of the syringe will become readily observable through the pupil.

The injection by an assistant is not begun until it has been clearly established that the tip of the needle has in fact reached the vitreous cavity and is not tenting up a fine grey layer of tissue, which represents the non-pigmented part of the pars plana epithelium or the vitreous base, or both (Fig. 4.11). A sharp needle will usually penetrate these layers, but occasionally a short thrust may be necessary to enter the vitreous cavity. After the injection is started there is an initial stream of small bubbles (less likely if injection is fairly rapid) which rise up behind the lens. In the aphakic eye the bubbles may come forward into the anterior chamber, but they tend to coalesce into a single bubble as the intraocular pressure rises. The needle is then kept in the central bubble and the injection continued until the desired amount of air has been injected. If it is found that the eye is not soft enough to allow an adequate injection to be made then more subretinal fluid may have to be released by reopening the sclerotomy site. In spite of these precautions, in the aphakic eye, particularly when there are irido-capsular remnants, small bubbles entering the anterior chamber may make subsequent visualisation very difficult. The difficulty of not being able to see retinal details after this procedure means that it should be left until the last possible moment since alteration of buckle positions can be most troublesome. In the case of a giant tear, the tear will have to be manipulated back into position after the injection of the air/gas mixture; this is achieved by rotating the patient on the operating table. When this has been done, the buckle that is to be used (usually an encirclement) can then be raised.

Silicone Oil

A silicone oil injection, for which the simple screw-top syringe is suitable (Kelly, 1969), is initially performed in a similar way to that for air. However, to accommodate the wider needle a preliminary scratch incision, almost down to the choroid, is advisable. After the introduction of the needle into the vitreous

cavity, and making sure that the needle has been completely filled with bubble-free silicone oil, the tip of the needle, which must be sharp in order to penetrate what is often thickened vitreous membranes, is passed into the centre of the vitreous cavity. The injection then proceeds in one of two ways. If there is a thick posterior equatorial membrane, and this can be identified lying anterior to the detached retina, the needle can be pushed through the thickened membrane and the injection begins in the position overlying the optic disc (Fig. 4.12). The subsequent injection of oil will produce progressive flattening of the detached retina and also push forward the equatorial membrane and collapsed gel. If, however, the vitreous organisation is even greater, or there is no posterior vitreous detachment so that a transverse vitreal membrane cannot be identified, then the injection of silicone oil will begin in the anterior vitreous. As the oil bubble expands, the retina will be pushed backwards and the fibrotic membranes greatly stretched. They will then tend to rupture, allowing the needle to be passed further backwards towards the disc and the injection to be continued in a more posterior position. One of the most important features of silicone oil injection is to inject into the eye as much oil as possible and to flatten the retina as completely as possible. On some occasions, when the view is adequate, preliminary aspiration of vitreous from the posterior gel may provide additional space for the oil injection. Before silicone oil is injected the subretinal fluid drainage site will have been prepared and a limited amount of subretinal fluid drained off. If the eye is allowed to become too soft, introduction of the needle and the injection of oil will be found to be most difficult. As the oil injection is continued, more subretinal fluid is drained from the eye and the

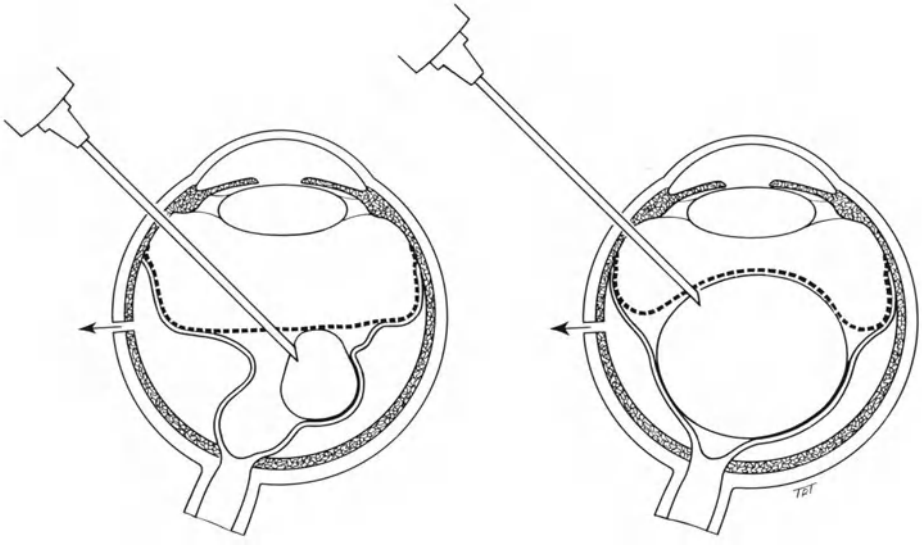

Fig. 4.12. Silicone oil is injected behind a thick transvitreal equatorial membrane (*dotted line*) and a drainage site for subretinal fluid has been prepared. The expanding bubble flattens the retina and pushes transvitreal membrane and collapsed gel forwards, keeping a layer of condensed gel between silicone and lens

process continued until the maximum amount of subretinal fluid has been drained. This may necessitate the preparation of more than one drainage site. Such sites should be kept somewhat smaller than usual to try to reduce the risk of retinal incarceration and perforation with loss of silicone oil via the drainage site. An attempt is made to prevent the tendency of silicone oil to break up into multiple bubbles by keeping the needle in the centre of the silicone mass. As the injection proceeds, retina will become progressively flatter and the optic disc will become visible. When the disc can be seen, vessel pulsation can be monitored in the usual way to avoid over-injection and a high intraocular pressure, and when it is deemed that as much oil as possible has been put into the eye the needle is withdrawn and the preplaced mattress suture at the site of injection is pulled up to avoid loss of silicone from the site of entry.

Operative Complications of Vitreous Injections

Complications other than those due to the specific material being injected are mainly caused by damage to the needle within the eye, and include damage to the lens by the tip of the needle, damage to the retina, with the risk of retinal haemorrhage and retinal hole formation. Failure to enter the vitreous cavity may result in the material being injected into the subretinal space or layers of the pars plana.

Closure of the central retinal vessels due to a sudden rise of intraocular pressure may occur, as also may anterior shift of the lens iris diaphragm which may produce a dangerous narrowing of the anterior chamber. Late complications may include the formation of fibrous strands along the track of the needle.

Closure of the Wound

The implants that have been used are neatly trimmed, particularly silicone sponges, and the limbal incision is then closed by drawing forward the conjunctiva and Tenon's layer into one layer, and securing it with two or three interrupted sutures of 6/0 collagen. A subtenon injection of Genticin is given in all cases (20 mg for adults).

Methods of Prophylactic Treatment

The majority of cases now receiving prophylactic treatment are those in whom the second eye is treated at the time a retinal detachment is being dealt with in the other eye. It is convenient to treat the fellow eye at the same time because the patient will in any case usually be receiving a general anaesthetic and will not have to suffer a further procedure at a later date. To avoid missing

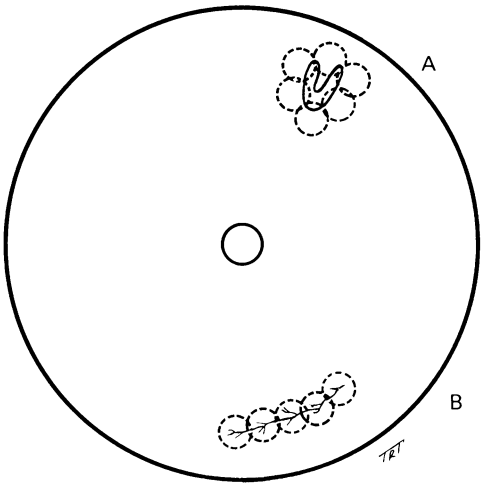

Fig. 4.13A–B. Prophylatic treatment; (A) A large horseshoe-shaped tear; (B) A linear area of lattice degeneration

this opportunity for treatment, the patient undergoing retinal surgery always has the fellow eye examined while under anaesthesia. This is important in patients in whom preoperation examination of the fellow eye has not been satisfactory and there has not been complete examination of the peripheral retina. In these cases prophylaxis consists of transconjunctival cryotherapy (Abraham and Shea, 1968). Most of the lesions being treated will not be posterior to the equator so that the conjunctiva will not have to be opened.

When small retinal holes are being treated, the cryoprobe may be placed directly underneath the hole and, usually, only a single application of the probe will be necessary to freeze the hole. When much larger holes are being treated several applications will be necessary to surround the hole completely (Fig. 4.13). It is not necessary to freeze an area more than once; the risk of overtreatment with prophylaxis is not great since the freezing reaction can readily be seen.

When linear areas have to be treated, the cryoprobe is manipulated to indent the centre of the area, and this will result in freezing the retina on both sides of the lesion (Fig. 4.13). Careful observation of local retinal landmarks will be necessary to see that areas are not unnecessarily heavily treated or missed altogether. When large areas have to be treated, for example in the 360° type of equatorial type of cryotherapy used in the preventive treatment of giant tears, the treatment is divided into two sessions, treating first the upper and then the lower half at each session. The time interval between each session is usually one to two months and the purpose of dividing the treatment is to minimise the amount of cryotherapy used at any one time and thus, hopefully, to reduce any tendency to stimulate preretinal membranes, particularly in the form of macular pucker.

When prophylaxis is needed and the patient is not receiving a general anaesthetic for detachment surgery, in most cases a local anaesthetic can be used.

The treatment can usually be conducted on an out-patient basis: Argon laser photocoagulation has been found to be the most convenient method and

is now preferrred to Xenon photocoagulation. Only topical anaesthesia is required in Argon laser photocoagulation. This enables the surgeon to place lesions accurately and with the minimum amount of energy, the treatment being applied through a three-mirror contact lens; the 500 spot size is used and the retinal hole completely surrounded by photocoagulation burns, the individual spots so placed that the edges of the rings are nearly touching each other. Sometimes photocoagulation treatment is found to be unsatisfactory, mainly when there are considerable opacities in the media, or when a horseshoe tear is extremely anterior, preventing adequate treatment of its anterior aspect. In these cases, prophylaxis is best performed with cryotherapy. If only a small relatively anterior area is to be treated this can often be dealt with by application of surface anaesthetic drops (Amethocaine 1%). The two or three applications of cryotherapy necessary will usually be tolerated by the patient with very little discomfort. If more extensive or posterior treatment is required, a retrobulbar anaesthetic will have to be given (2 to 3 ml of lignocaine 1%). This treatment, again, can usually be conducted on an out-patient basis. In the more nervous patient, premedication is necessary (oral or systemic Valium). Rarely, a general anasethetic will be necessary, for which the patient will have to be admitted to hospital for a period of one or two days. Following prophylactic treatment, pigmentation around the area begins, usually after four or five days, and a firm adhesion achieved after about two weeks. During this time patients are advised not to indulge in violent exercise, although a period of absence from sedentary work is unnecessary.

Complications of Prophylaxis

Cryotherapy is a very safe method of prophylaxis. Most side effects reported are minor and appear not to have any permanent sequelae, e.g. conjunctival chemosis, small conjunctival lacerations, or small retinal or choroidal haemorrhages. If cryotherapy is extensive transient choroidal detachment may occur.

Only rarely have more serious side effects such as macular pucker (Chignell and Shilling, 1973) or cystoid maculopathy (Ryan, 1973) been reported. These more serious side effects appear to be much less likely following prophylactic Argon laser photocoagulation.

References

Abraham, R.K., Shea, M.: Avoidance of depigmentation and retinal atrophy following cryoretinopexy. J. Cryosurg. *1*, 190–192 (1968)

Abraham, R.K., Shea, M.: Significance of pigment dispersion following cryoretinopexy. Scotomata and atrophy. Mod. Probl. Ophthalmol. *8*, 455–461 (1969)

Chignell, A.H.: Retinal dialysis. Br. J. Ophthalmol. *57*, 572–577 (1973)

Chignell, A.H., Shilling, J.S.: Prophylaxis of retinal detachment. Br. J. Ophthalmol. *57*, 291–298 (1973)

Chignell, A.H., Revie, I.H.S., Clemett, R.S.: Complications of retinal cryotherapy. Trans. Ophthalmol. Soc. U.K. *91*, 635–651 (1971)
Fison, L.G.: Localisation in retinal surgery. Trans. Ophthalmol. Soc. U.K. *95*, 113–114 (1975)
Gass, J.D.M.: Scleral marker for retinal detachment surgery. Arch. Ophthalmol. *76*, 700–701 (1966)
Hilton, G.F.: Subretinal pigment migration. Arch. Ophthalmol. *91*, 445–450 (1974)
Kelly, T.S.B.: Remote screw type syringe for intravitreous injection. Mod. Probl. Ophthalmol. *8*, 577–580 (1969)
King, L.M., Schepens, C.L.: Limbal peritomy in retinal detachment surgery. Arch. Ophthalmol. *91*, 295–298 (1974)
Laqua, H., Machemer, R.: Repair and adhesion mechanism of the cryotherapy lesion in experimental retinal detachment. Am. J. Ophthalmol. *81*, 833–846 (1976)
Lincoff, H.A., Kreissig, I., Parver, L.: Limits of constriction in the treatment of retinal detachment. Arch. Ophthalmol. *94*, 1473–1477 (1976)
Pruett, R.C.: The fishmouthing phenomenon. Arch. Ophthalmol. *95*, 1777–1787 (1977)
Ryan, S.J.: Cystoid maculopathy in phakic retinal detachment procedures. Am. J. Ophthalmol. *76*, 519–522 (1973)
Sudarsky, R.D., Yannuzzi, L.A.: Cryodemarcation line and pigment migration after retinal cryosurgery. Arch. Ophthalmol. *83*, 395–401 (1970)

Chapter 5

Postoperative Management and Complications

General Management

Patients are mobilised as soon as possible following surgery, usually on the first postoperative day. This is important in older patients in order to avoid deep-vein thrombosis in the lower limbs. The only exception to this rule is in cases in which intravitreal air or gas has been used at the time of surgery and postoperative posturing of the patient is necessary to position the gas correctly within the eye, that is to keep the bubble up against the part of the retina needing support, e.g. for superior tamponage the patient is kept upright with the head inclined slightly forward. In the event of the patient moving out of the correct position he must be shown how to get back into the correct one by himself. With giant tears correct positioning must be maintained for at least a week from the time of surgery.

There is a variable amount of discomfort in and around the eye following surgery and there is usually some lid swelling and chemosis. The external symptoms and signs are related to the type of procedure that has been performed and also to whether the surgery employed has been the first operation. In a case of retinal detachment when there has been only a local buckling procedure without drainage of subretinal fluid, and in which there has been no previous surgery, then extraocular tissue disturbance is minimal. If, on the other hand, there has been a rather long and more complicated procedure involving, say, encirclement, cryotherapy and drainage of subretinal fluid, then the extraocular tissue signs are much more marked and there is usually an associated mild traumatic uveitis. When a dissection has been particularly posterior, then some degree of proptosis is often produced in the postoperative period.

In uncomplicated cases the patient is able to leave hospital a few days after surgery. A further period of recovery of approximately two weeks is spent at home before work, if of a sedentary kind, or other activities are resumed. Strenuous physical work involving lifting heavy loads, or sporting activities such as riding, or squash racquets should be avoided for approximately two months. Some sports should be abandoned altogether, e.g. boxing and high diving.

Recovery of Vision

In the event of successful reattachment of the retina, subjective improvement in the quality of the field of vision is noticed by the patient almost immediately.

Other preoperative symptoms may persist in the postoperative period. Thus, floaters as a result of vitreous haemorrhage may persist for weeks or months, and although diminshing they may never completely disappear. Flashes of light may likewise persist for many weeks or months following retinal detachment surgery and may even appear in the postoperative period in patients in whom they had not been manifest preoperatively. It is assumed in these cases that the appearance of these symptoms is due to the buckling procedure itself. Retinal recovery after surgery has been studied by a variety of workers. In one study (Chisholm et al., 1975) it was found that the longer the detachment had been present, the poorer was the eventual recovery as assessed by visual acuity, visual field, and colour discrimination. The same workers also found that the recovery of central vision after the macula had been detached might be prolonged up to three years. Patients should be warned about the slow recovery of central vision when they are being followed as out-patients. In a previous study (Jay, 1971) it was found that recovery of macular function as measured by central visual acuity was likely to be approximately 35–40% of its preoperative level but was worse if the macula had been detached for a long period; for example, a preoperation vision of 6/60 might be expected to improve to 6/18. Sometimes, and somewhat unexpectedly, visual acuity will return to normal after macular reattachment.

Although it was found by Grupposo (1975) that the level of vision achieved postoperatively was not statistically correlated to the duration of macular detachment unless the latter had been present for greater than eight weeks, most surgeons would favour reattachment as soon as is reasonably possible. A similar poor recovery of central vision after a long detachment of the macular has also been confirmed by others (Kutschera, 1968; Davies, 1972). The relationship between the length of time of macular detachment and visual recovery was confirmed by Kreissig and Lincoff (1974) and the less satisfactory return of central vision in old age and myopia was also found (Kreissig, 1977).

Following surgery, the response of the eye will be mainly judged by alterations in the volume of subretinal fluid and, where appropriate, the relationship between the buckle and the hole. The cryoreaction around the treated areas will also be seen. Retinal oedema is normally detectable for approximately one to two days as a slight whitening of the detached retina, and pigmentation of the cryotherapy lesion appears at variable times from surgery, depending on whether detached retina has been frozen at the time of surgery or not. In prophylactic procedures, when the retina is flat, a pepper and salt pigmentation appears four or five days after surgery, but in a deeply detached retina the pigmentation may not be seen until a week or ten days after surgery. The eventual appearance of the cryotherapy scar will vary according to the intensity of application. The initial pepper and salt pigmentation gradually gives way over a period of weeks to a somewhat coarser arrangement of pigment, but if the choriocapillaris and retina has not been completely destroyed the effected area is apparently of normal thickness and has a pinkish colour. When the cryotherapy lesions have been extremely heavy, pigment clumps are found on the edge of the

lesion, which is itself very pale and thin due to destruction of choriocapillaris and neuroepithelium. The larger choroidal vessels, however, invariably remain intact and can be seen in the scar. The appearance of pigment in the cryotherapy lesion is due to clumps of macrophages and is not related to the adhesive quality of the lesion (Lincoff and McLean, 1969). However, the appearance of the lesion is an indication of the severity of the cryotherapy treatment and, therefore, the intrinsic strength of the lesion. In the experimental animal (Lincoff et al., 1970) it was found that the maximum strength of the cryotherapy lesion in flat rabbit retina studies was achieved in eleven days.

Behaviour of Subretinal Fluid

Postoperative absorption of subretinal fluid mainly depends upon the relationship of the hole to the buckle at the end of the operation. If the hole has been completely closed at the time of surgery, then subretinal fluid absorption will usually be rapid and complete within two days of operation and will take place regardless of the length of time there has been detachment preoperatively: an event that is readily demonstrated by the absorption of subretinal fluid following retinal dialysis surgery in which subretinal fluid, which may have been present for many years, can be absorbed within 24 hours (Nadel et al., 1971). If it has not been possible to close the hole at the time of surgery then the absorption of subretinal fluid will take longer, depending upon the time it takes the hole to close (Chignell and Talbot, 1978). The distance between the buckle and the hole will gradually become reduced but this may take up to a week from the time of surgery in deeply detached cases. During the settling back of the retinal hole towards the buckle, there is some absorption of subretinal fluid but complete absorption will be achieved only after the hole has been sealed. Subretinal fluid may persist for a long or indefinite time if periretinal fibrosis prevents complete settling back of the detached retina. The absorption of subretinal fluid is not influenced by the age of the patient, the presence of myopia, or the initial volume of subretinal fluid.

Even in the absence of retinal fibrosis or vitreoretinal traction, reabsorption of subretinal fluid may unexpectedly take several weeks (Leaver et al., 1976) and the surgeon must not be tempted to reoperate just because there is subretinal fluid, unless an unsealed retinal hole causing the fluid can be demonstrated. The reason for the persistence and slow absorption of subretinal fluid in these cases, in spite of closure of the retinal hole, is poorly understood. Initial reabsorption of fluid, followed by reaccumulation, indicates the presence of an imperfectly sealed hole or a missed hole. If the retina does not flatten around the site of the buckle then the original hole is usually at fault, whereas if retina around the buckle becomes flat but detachment persists elsewhere the presence of a hole at another site is indicated.

The complications of retinal detachment surgery may be divided into 'early' and 'late'. In early complications the problems arise usually within a week of surgery (Chignell, 1975) but if late, they may arise any time after the patient has been discharged from hospital.

Early Complications

Sudden Blindness

Sudden blindness in the postoperative period is happily extremely rare. It is usually detected at the first postoperative dressing when the patient is unable to perceive light in the operated eye. Central retinal artery occlusion may occur as a result of over-tightening of buckle sutures causing an excessive and prolonged rise of intraocular pressure, possibly augmented by systemic hypotension from anaesthesia during operation or in the immediate postoperative period. Direct damage to the optic nerve at the time of surgery due to deep dissection within the orbit has happened occasionally. In some cases the cause of sudden blindness is hard to determine (Jarrett and Brockhurst, 1965). There is no treatment.

Infection

Infection is extra- or intraocular in nature. In non-drainage surgery infection occurring in the immediate postoperative period is always extraocular but if drainage of subretinal fluid has taken place there may be intraocular spread of infection. The overall incidence of infection in the immediate postoperative period was 2.4% in a series of 416 consecutive retinal detachment operations (Hitchings et al., 1974).

Extraocular Infection

The onset is one to five days from the time of surgery. Pain is a marked feature and usually starts in the immediate postoperative period with associated lid swelling and chemosis. The diagnostic feature is a mucopurulent discharge which when it appears usually gives relief from pain. The treatment is to remove the implant or any other causative foreign body, such as a retained swab, as soon as it is considered safe to do so, bearing in mind the state of the detached retina. It is safe to remove an implant a week after the retina has been reattached with little risk of redetachment. The course of infection is very little influenced by the administration of either local or systemic antibiotics.

Prevention of most of these early infections appears to be achieved by the routine injection of sub-tenon genticin (20 mg) at the end of surgery, a regime that was successful in preventing early infection in 243 consecutive detachment operations (Lean and Chignell, 1977).

Intraocular Infection

Severe intraocular infection is now extremely rare. The onset is one to five days from the time of surgery and is accompanied by considerable pain and lid swelling, with chemosis. Flare and cells in the anterior chamber are prominent features and may progress to hypopyon formation. Increasing haze in the posterior segment may progress to vitreous abscess. Treatment is by the intensive administration of antibiotics, both local and systemic. (One regime is to administer topical chloromycetin drops, sub-conjunctival gentamicin on alternate days, and systemic flucloxacillin, the latter being given parenterally if necessary.)

Local and systemic steroids are introduced after approximately 48 hours in an attempt to reduce intraocular fibrosis as a consequence of the infection: 50 mg of prednisolone a day is given for a few days and then gradually reduced and discontinued over a two to three week period. If an extrascleral sponge has been used as part of the buckling procedure, it is probably better, if a mucopurulent discharge has appeared, to remove this implant a few days after the infection has started. In such cases, however, the removal of the buckling elements may not always have much apparent effect on the course of the intraocular infection.

Scleral abscess, seen when diathermy was practised, is no longer a problem since the introduction of cryotherapy (Lincoff et al., 1965).

Anterior Segment Ischaemia

Ischaemia of the anterior segment following retinal detachment surgery is a result of poor perfusion of the anterior segment and arises because of a reduction of arterial inflow from the anterior ciliary or the long ciliary arteries, or from venous drainage by the vortex veins, or both. Encircling procedures, by their very nature to compress the whole globe are more likely to produce changes than local buckling operations. Clinical syndromes produced by ischaemia of the anterior segment vary greatly from mild ischaemia to severe anterior segment necrosis (Crock, 1967).

Clinical Syndromes

The onset is two to five days after surgery. In mild cases there is slight corneal oedema, flare and cells in the anterior chamber, and the rapid appearance

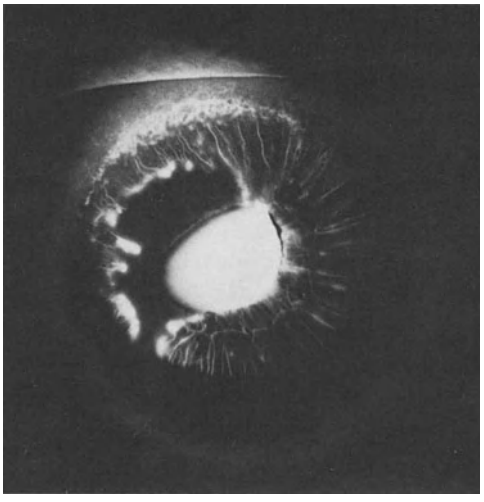

Fig. 5.1. Anterior segment ischaemia following a local buckling procedure. There is nonfilling of a sector of iris and corresponding sectorial atrophy of the iris sphincter

of segmental atrophic change in the iris, the last-named manifesting itself initially as a sluggish pupillary reaction in the affected segment. Often there is thick flare and cells, marked iris atrophy, the iris also assuming a somewhat greenish tinge, posterior synechiae, hypotony and subsequent cataract formation. Diagnosis of the ischaemic states may be greatly facilitated by making use of iris fluorescein angiography which will show areas of the iris that are poorly perfused (Fig. 5.1) (Chignell and Easty, 1971; Easty and Chignell, 1973).

Treatment

In mild ischaemia the reaction in the anterior chamber can be reduced by administration of local steroids, and there is no serious long-term effect. In severe cases the intravenous infusion of rheomacradex (low molecular weight dextran) to increase perfusion over a 24 to 48 hour period has been advocated (O'Day et al., 1966). The effect of this substance has never been fully assessed but clinical impressions suggest that at least in some cases it may have a beneficial effect. In very severe cases removal of the implant (usually an encircling band) is advisable if there is no improvement after 48 hours treatment with rheomacradex.

Prevention

a) Preoperative examination may reveal a high risk anterior segment perfusion problem, e.g. an eye with ipsilateral carotid stenosis, a carotico-cavernous fistula or, a patient suffering from a general haematological problem such as HbSc disease. These groups of patients may be expected to tolerate poorly even slight interference with ocular perfusion, hence encircling bands must be avoided and local procedures used whenever possible.

b) At operation, extraocular muscles should not be detached unless absolutely necessary to preserve the anterior ciliary arteries, and damage to vortex veins must be avoided.

Progressive favouring of local procedures has greatly reduced the incidence of severe postoperative anterior segment ischaemia so that it is now a rare problem.

Sterile Uveitis

This is due either to trauma or to excessive cryotherapy.

Trauma

The type of uveitis due to trauma is found to a varying degree following most types of retinal detachment surgery. It may be confined to the anterior segment or be combined with some degree of vitreous haze. It usually appears within the first one or two days after surgery and there is progressive improvement within the first postoperative week. The uveitis is more marked if there has been any degree of intraocular haemorrhage either at operation or in the post-operative period. Serous choroidal detachments are also usually accompanied by some degree of uveitis.

Treatment. Local steroids are supplemented by systemic steroids if there is any significant degree of vitreous haze.

Cryotherapy-induced Uveitis

This type of uveitis appears only after excessive cryotherapy (Chignell et al., 1971), in cases in which it has been particularly difficult to apply, e.g. in a ballooned detachment with multiple retinal holes. In these cases, not only is the end point of cryotherapy difficult to see, which calls for an excessively long application, but there is a tendency to refreeze an area several times. Following operation there is a latent period of approximately four to five days, the eye then becoming painful and signs of uveitis appearing. Vitreous haze is seen over the implant (Fig. 5.2) and may spread extensively through the vitreous body. Treatment is by vigorous local and systemic steroids with a starting dose of 60 mg of prednisolone a day for three days. After a few days the vitreous will start to clear and over a period of approximately two weeks the systemic steroids can be tailed off and discontinued. There will be other signs of excessive cryotherapy: in particular there will be pigment fallout, and there may be persistence of retinal oedema for more than the usual one or two days in the postoperative period. Eventually an atrophic retinal scar is produced.

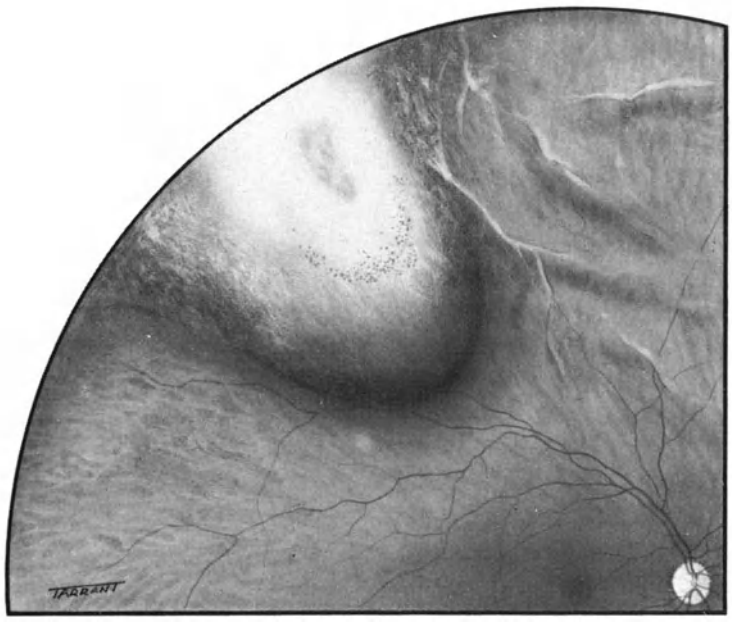

Fig. 5.2. Cryotherapy-induced uveitis five days after a nondrainage operation. There is vitreous haze over the implant and the retina is still oedematous around the tear

In the three conditions, infection, ischaemia, and sterile uveitis, prompt diagnosis is desirable not only to reduce postoperative morbidity but also to institute corrective treatment as quickly as possible.

These conditions may all present considerable difficulty in differential diagnosis as all may present as a painful eye with flare and cells in the anterior chamber. The main points of differential diagnosis are:

1. The appearance of a mucopurulent discharge indicates infection, which will remain extraocular if a non-drainage operation has been performed.

2. Iris fluorescein angiography will help to demonstrate ischaemic conditions.

3. The early appearance of uveitis with progressive improvement indicates a simple traumatic aetiology.

4. The late appearance of uveitis suggests a cryotherapy induced reaction.

5. A rapidly worsening uveitis with hypopyon formation suggests infection.

In spite of careful observation a clear-cut diagnosis may sometimes be impossible to establish and, indeed, mixed pictures may be present; for example, traumatic uveitis following a long and complicated detachment procedure which involves, say, encirclement, cryotherapy, and drainage may be further complicated by ischaemic features or the presence of secondary glaucoma. Secondary glaucoma makes the interpretation of iris angiography unreliable since it will in its own right produce ischaemic changes in the anterior segment. It may also be difficult to rule out early infection in these cases and it will be necessary to administer both steroids and antibiotics locally and systemically. Failure to withhold steroids may increase the risk of proliferation of periretinal membranes.

Glaucoma

Severe glaucoma was found to occur postoperatively in less than 1% of cases of (Chignell et al., 1973) but an incidence of 4% was found by others (Kreiger et al., 1971). The glaucoma that appears in the postoperative period can be closed or open angle in nature. In both types of glaucoma the eye is painful and is injected but more so in angle closure.

Closed Angle Glaucoma

This type of glaucoma is the result of:

a) An anterior shift of the lens-iris diaphragm, by deep buckling procedures (usually encircling) in the posterior segment of the globe. This movement of the lens-iris diaphragm (observed by Hartley and Marsh, 1973) may be further accentuated by compression of the vortex veins, with subsequent venous congestion of the posterior segment. Experimental work has demonstrated the congestion as a result of vortex vein obstruction (Hayreh and Baines, 1973). In this type of glaucoma the onset can usually be seen within one or two days of retinal surgery.

b) Serous choroidal detachment. If this is extensive, the inevitable associated detachment of the ciliary body may result in the ciliary body hingeing forward on the scleral spur with a resultant shallowing of the anterior chamber and the risk of angle closure. This type of glaucoma may reveal itself at any time up to a week after retinal detachment surgery and usually occurs subsequent to the appearance of obvious choroidal detachment.

Open Angle Glaucoma

This is usually secondary to uveitis, less commonly secondary to extensive vitreous haemorrhage, and is more likely to occur in cases where there is underlying aqueous outflow obstruction, e.g. in chronic simple glaucoma or in an eye in which the angle has been markedly recessed preoperatively as a result of previous trauma.

Prevention

The occurrence of glaucoma, particularly the angle closure type, underlines the need for careful assessment of the angle by gonioscopy at the time of the preoperative three mirror examination. If the angle is found to be narrow then deep posterior segment buckles, if possible, should be avoided.

At operation, hypotony should be reduced to a minimum by prompt restoration of intraocular pressure by tightening the buckle sutures as soon as subretinal fluid has been drained. This will reduce the likelihood of postoperative serous

choroidal and ciliary body detachment. Judicious use of cryotherapy may also contribute to reducing the incidence of choroidal detachments.

The depth of the anterior chamber in suspect cases should be carefully watched in the postoperative period, observations that may be augmented by serial measurement with a pacometer.

Management

In all types of glaucoma the management should be conservative and surgical interference should be avoided at all costs.

Diamox 250 mg is given four times a day. In the closed angle type, miotics may be tried but they appear to be of little benefit. In the open angle type mydriasis is advised. Conservative management will usually result in controlling intraocular pressure at a reasonable level. The rise in pressure will generally improve spontaneously and become normal within a week or two of surgery.

The postoperative complications of infection, uveitis, ischaemia, and glaucoma highlight the need for regular slit-lamp examination of the retinal detachment patient in the postoperative period.

Choroidal Detachment

Choroidal detachment observed in the postoperative period may be serous or haemorrhagic, the former being much the more common.

Serous Choroidal Detachment

These detachments usually arise in the first 24 to 48 hours following surgery but their appearance may be delayed by up to a week. They are smooth dome-shaped elevations which may or may not be in direct relationship to the buckle (Fig. 5.3), and they may remain localised or extend to involve the whole of the peripheral retina. The elevations themselves have a brownish colour and the fluid within them does not shift. They rarely extend posterior to the equator, and by pushing forward the peripheral retina the ora serrata is characteristically exposed and can be seen with ease without scleral depression. Having appeared, choroidal detachments absorb spontaneously within a period of one to two weeks. If the choroidal detachment is only slight in extent there is no residual change to be seen in the retina. If, however, the detachments have been extensive there is often a residual pepper-and-salt pigmentary disturbance which may take the form of thin demarcation-like lines.

In a large series of retinal detachments the incidence of choroidal detachments was 23% (Hawkins and Schepens, 1966). These authors found that the factors increasing the likelihood of such detachments were vortex vein interference,

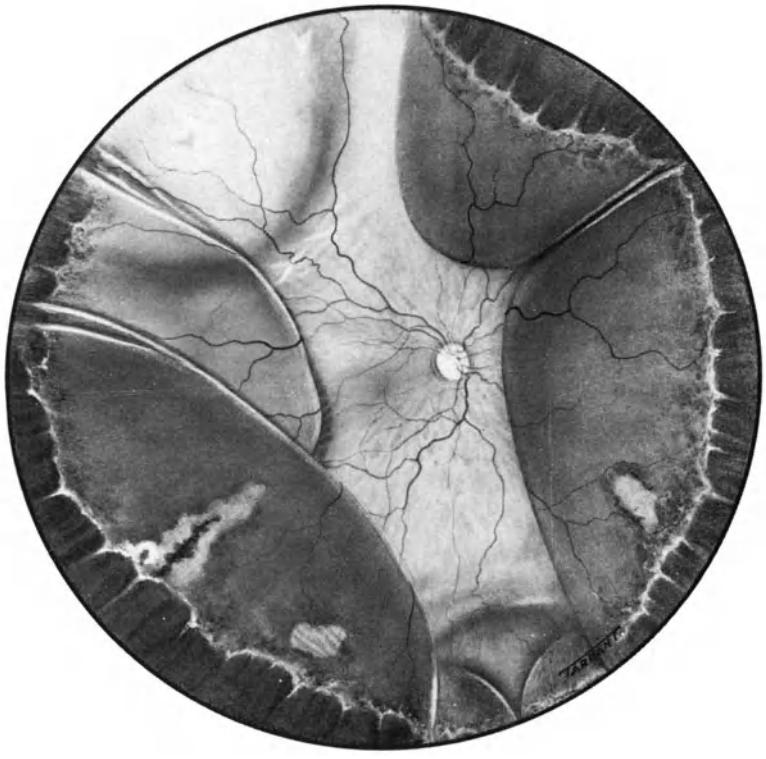

Fig. 5.3. Extensive serous choroidal detachment following a nondrainage retinal detachment operation

myopia, the use of diathermy, and the drainage of subretinal fluid, and there is an increasing tendency in the older age group (supported by Hilton and Norton, 1969). More recent series favouring the non-drainage technique show a much lower incidence, and it is noteworthy that choroidal detachments following non-drainage procedure are rare (Chignell, 1972). This suggests that hypotony resulting from drainage of subretinal fluid is the main factor in producing choroidal detachment in these cases. Vortex vein interference is also important since posterior buckling procedures are more likely to result in serous choroidal detachment.

As has already been seen these choroidal detachments may, if associated with ciliary body detachment, rarely progress to angle closure glaucoma, but do not otherwise have any untoward effect. If an eye has had a postoperative choroidal detachment that has reabsorbed, in the event of a further detachment operation the choroidal detachment can be predicted with certainty to occur again in the postoperative period.

Haemorrhagic Choroidal Detachment

This type of detachment almost invariably arises as a result of an operative complication and rarely arises de novo in the postoperative period. It is often

associated with some degree of overlying serous detachment. The haemorrhagic detachment is seen as a black, rounded mass underneath the retina and, although it generally becomes progressively flatter it may undergo some degree of enlargement in the postoperative few days. If severe, choroidal haemorrhage usually spreads via the retinal hole into the vitreous, which becomes diffusely cloudy two or three days after surgery, obscuring the view of the retina. Postoperative absorption of the choroidal haemorrhage may take several weeks and increases the likelihood of failure of reattachment by encouraging intraocular fibrosis.

Vitreous Haemorrhage

Vitreous haemorrhage in the postoperative period arises as a result of extension of a choroidal haemorrhage or, more rarely, from a further haemorrhage from a retinal tear. The latter is very unusual but on the rare occasions when it happens in the postoperative period the haemorrhages may be multiple, occurring for many months or even years after the time of operation. In most cases the fresh haemorrhages are seen to arise from the vessel in the edge of the operculum. Severe vitreous haemorrhages may sometimes be complicated by secondary open angle glaucoma (haemolytic glaucoma).

Exudative Retinal Detachment

This rare type of detachment has been attributed to the use of cryotherapy, particularly if it has been excessive (Aaberg and Pawlowski, 1972). Subretinal fluid starts to accumulate on about the second postoperative day and may or may not be continuous with the retinal holes. The fluid shifts markedly, like other exudative detachments, and it is invariably accompanied by vitreous flare and cells, and usually also by an anterior uveitis. A similar type of exudation is sometimes seen after cryotherapy or photocoagulation of peripheral vascular angiomatous tumours. The exudative response, which may extend to involve practically the whole retina, has a benign course with spontaneous absorption of fluid within a week or two after its appearance. The administration of systemic steroids may induce a more rapid resolution.

Late Complications

Implant Extrusion and Infection

Many months after surgery there may be a tendency for silastic sponge implants to work themselves loose and to assume a rather bulky swelling of the conjunc-

tiva. This does not require treatment although patients are sometimes alarmed by the appearance of a sub-conjunctival lump. If the sponge actually extrudes through the conjunctiva itself it is likely that infection has played at least a contributory part for extrusion of sponges that were obviously infected was common when these materials were first introduced (an incidence of 24% extrusion was reported by Russo and Ruiz, 1971) but is now rare. This improvement is due to meticulous suturing techniques aimed at maintaining the integrity of the scleral suture by using as long an intrascleral course as possible, use of a limbal incision (to provide adequate cover for the sponge, and the reduction of infection by the use of subtenon antibiotics at the end of operation (Flindall et al., 1971; Lean and Chignell, 1977).

Since the use of cryotherapy and full scleral buckling, infection of silastic sponge implants appears as an extraocular syndrome: as a subconjunctival haemorrhage of a recurrent nature with the formation of a granuloma at the site of the original conjuctival suture line (Fig. 5.4), or with the appearance and persistence of a mucopurulent discharge (Langston et al., 1965; Lincoff et al., 1965). Occasionally the methods of presentation are mixed. An infection may manifest itself many months after surgery but is presumably still a result of organisms introduced at the time of operation. Provided infection does not occur within the immediate postoperative period and subretinal fluid has been drained, there are no intraocular signs.

The eye with an infected sponge is usually red and irritable with a mucopurulent discharge. On examination, the conjunctival fistula can usually be detected and granuloma formation if present, easily seen. The administration of antibiotics, either local or systemic, has no obvious effect in ameliorating the infection, the treatment of which is to remove the implant. Sometimes the infected

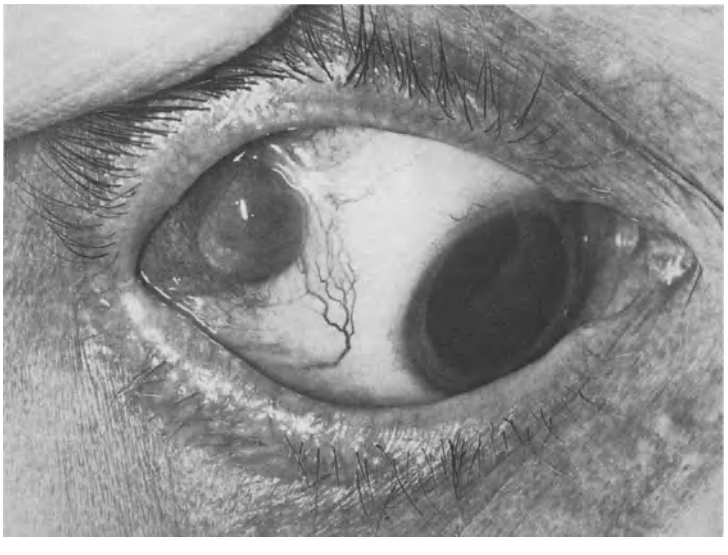

Fig. 5.4. An infected sponge has led to granuloma formation

implant will extrude itself spontaneously, the infection clearing rapidly. According to one study, infections are less likely when solid implants are used and are more likely in reoperations (Ulrich and Burton, 1974). The same study also showed a very high risk of redetachment when these implants were removed (33%). A similar incidence of redetachment has been noted by other workers (Schwartz and Pruett, 1977) who reported an incidence of 28%. This extraordinarily high redetachment rate following removal of implants has not been observed by other workers (Rouchy, 1971), indeed, I have found that redetachment is quite exceptional. The apparent discrepancy in the figures may be that there is a greater risk of infection in complicated reoperations at which there may already be a significant degree of intraocular fibrosis, and in these cases the loss of the buckle may contribute to redetachment.

The treatment of an infected implant is to remove it. If the sponge has started to extrude through the lips of the conjunctival incision it may be removed, after the application of topical anaesthetic drops (Amethocaine 1%), simply by grasping it firmly and gently pulling. If, on the other hand, the sponge is buried and covered by granulomatous tissue, then removal is best performed under general anaesthesia. When the sponge is removed scleral sutures should likewise be removed. The underlying sclera is normal.

Intraocular Erosion of Implants

The use of full thickness scleral buckles and sponge implants has greatly reduced the risk of intraocular erosion. Also, improvement in the materials used for encirclement and the appreciation that tight encirling bands are now rarely necessary, have likewise reduced this tendency. Thus, the sight of an encircling element lying underneath the retina or in the vitreous cavity itself is now seldom seen. Scleral sutures may, however, erode with the passage of time, particularly if they are placed underneath the encircling band; the appearance of these sutures on routine examination of the retina is of no more than passing interest as they rarely cause problems or need to be removed. If in fact an implant has eroded within the eye, symptoms are seldom produced. On rare occasions redetachment may occur, as may recurrent vitreous haemorrhage. In spite of these complications, however, removal of the implant is not advised due to the risk of further damage to the retina itself or of precipitating further haemorrhage.

Choroidal Folds

Choroidal folds may be seen after retinal surgery and although usually confined to the retina just posterior to a radial buckle, they may be extensive in their distribution and involve the posterior pole. They often persist for months and

possibly for years. They are usually detected as an incidental finding if fluorescein angiography is performed but the effect on subsequent return of vision is not known, although they do not apparently effect visual recovery.

Postoperative Pain

Pain following retinal detachment procedures may be very considerable and last for many weeks or even months following operation. The pain usually takes the form of a dull ache around the eye and may be referred to the side of the head in the distribution of the ophthalmic branch of the trigeminal nerve. Although intraocular problems (such as ischaemia of the anterior segment, glaucoma, or uveitis) may be the cause of the pain, it may be impossible to demonstrate any intraocular cause, the pain apparently being due to constriction by the encircling band. Usually most severe following deep encirclement procedures the pain is rarely of much significance following local buckles. In the majority of cases pain encountered in the postoperative week or two wears off within a further month, but if unremitting it may be necessary to cut or loosen the encircling band.

Postoperative Diplopia

Diplopia following retinal detachment surgery has become an increasingly rare complication due to the tendency not to disinsert muscles at the time of surgery, although the association between diplopia and muscle disinsertion has been denied by some authors (Sewell et al., 1974). In a successful retinal detachment series diplopia was estimated to occur in approximately 3.3% of cases (Kanski et al., 1973) but cleared spontaneously in most of them. The vertical rectus muscles are the most susceptible to interference but occasionally problems may arise from disturbance at the insertion of the superior oblique, a site that is often visited by the surgeon in the upper temporal quadrant. If diplopia is severe, temporary symptoms may be controlled by the use of prisms, it is exceptional for muscle surgery to be necessary.

Refractive Changes

In the great majority of cases in which silastic sponges have been used significant spherical changes of refractive error do not occur because the actual length of the globe is unchanged (Weidenthal, 1971). With encircling bands there is a tendency to increase myopia due to elongation of the globe (Dalgleish, 1966; Weidenthal, 1971) but the more gentle encircling ridges favoured nowdays

cause little change. However, when there is a deep encircling indent the actual length is surprisingly shortened and there is, therefore, a resultant shift towards hypermetropia (Rubin, 1975). Deep radial buckles may produce high degrees of astigmatism (Burton, 1973; Chignell and Crewdson, 1978). Such large indents are used only exceptionally but may occasionally be inadvertently produced, particularly if fluid is drained from the eye and extra indentation is employed to compensate for subsequent hypotony. The high degree of astigmatism produced may be unacceptable to the patient in the form of a spectacle lens; removal of the implant then becomes necessary.

Macular Changes

Macular Pucker

This complication, which is due to the presence of preretinal membrane at the macula, has been described following all forms of retinal detachment procedures (Crews, 1968) and also following prophylaxis with both light coagulation and cryotherapy (Lincoff and McLean, 1969). When macular pucker occurs in a case in which the macular has not previously been detached prior to surgery the effects are particularly devastating. About six weeks after surgery, there is reduction of central vision with micropsia and metamorphopsia. The macular changes begin as oedema, with disturbance of the normal macular reflex and wrinkling of the internal limiting membrane (cellophaning). Fine retinal folds form and arise either in a general pattern or from a focus. These folds, initially invisible, thicken to form perceptible white strands (Wise, 1972; Hamilton, 1972; Wise et al., 1975). As the whitish membrane contracts there is distortion of neighbouring blood vessels which are drawn into the centre of the membrane in a tortuous manner with associated straightening of the more central part of the vessels. The posterior vitreous is usually found to be detached, and intraretinal white dots, small haemorrhages, and, later, small exudates, may be found with serous fluid underneath the neuro-epithelium. In the majority of cases of macular pucker vitreous traction does not appear to play a significant role although occasionally thin bands are found extending from the area of macular pucker to the posterior vitreous face (Tanenbaum et al., 1970). Fluorescein angiography in the initial stages will show no abnormality, apart from slight tortuosity of the vessels, but in more advanced cases will demonstrate massive leakage in the area of the membrane and spread into adjacent subretinal fluid (Fig. 5.5). Postmortem studies (Roth and Foos, 1971) have suggested that the cells responsible for these membranes are derived from the retinal glial cells. Usually, the pucker that has formed remains confined to the macular region but sometimes extensive radiating fibrous sheets may form and involve the whole posterior pole of the eye.

In mild cases, when the reduction of central vision has been only relatively slight, resolution and subsequent return of good vision may be achieved. Resolu-

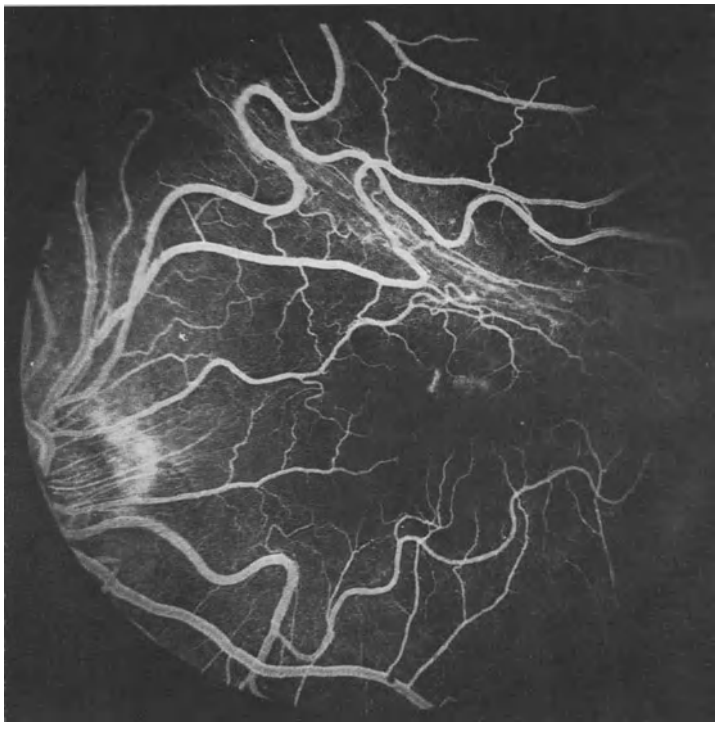

Fig. 5.5. Macular pucker resulting in gross distortion of retinal vessels

tion is sometimes associated with detachment of the posterior vitreous face from the posterior pole. In more severe cases the loss of central vision is irreversible.

Macular pucker of some degree occurs much more frequently in cases in which the retinal detachment involved the macula in the preoperative period, and presumably the actual presence of subretinal fluid (beneath the macula) combined with the trauma of detachment and reattachment is the stimulus to the formation of membranes in these cases. In these same cases the macular changes are often noticed when the patient fails to achieve any significant degree of return of central vision after successful surgey. The condition is usually untreatable, although vitrectomy and peeling of the offending membrane has occasionally been successfully performed and may become popular.

Other Macular Changes

Changes in the posterior pole following successful reattachment of the retina are not uncommon (Sarin and McDonald, 1970). They are mainly non-specific pigment epithelial changes and reveal themselves as small areas of proliferation interspersed with areas of pigment epithelial atrophy. Cystoid macular change and non-specific macular oedema are much less common, and macular hole

formation is rare. Pigment epithelial changes are particularly likely to be found following long-standing detachment of the macula and may jeopardise the final visual result (Cleary and Leaver, 1977). Pigmentary changes and scarring at the macula can also occur secondary to subretinal haemorrhage arising as a result of chroidal haemorrhage entering the subretinal space and settling at the macula.

In most cases in which the expected level of return of visual acuity following retinal detachment surgery fails to occur, the failure can be related to identifiable changes at the macula. However, in some cases prompt reattachment of a detached macula will not result in such improvement even when the macula appears to be quite normal on biomicroscopic and fluorescein angiography examinations. In these cases failure of photoreceptor regeneration has to be invoked to explain the visual deficit.

Periretinal Membrane Changes

The response of periretinal membranes to surgery is variable. If, for example, in preretinal membrane formation the process is not advanced and the membranes are still weak, then successful closure of the retinal hole will result in reattachment of the retina with subsequent regression of the membranes so that in the postoperative period and on follow-up examinations they are scarcely detectable. The retroretinal strands seen in long-standing detachments, while not interfering with retinal reattachment, may still be clearly seen and are often sharply demarcated on the back of the reattached retina (Fig. 5.6). More advanced full thickness retinal folds may likewise undergo almost complete regression, leaving only a small whitish scar on the surface of the retina to indicate where they had been situated. When membrane formation is extensive it may prevent complete reattachment of the retina and in these cases there is some partial retention of subretinal fluid which may persist and remain static for months or years. If vitreous bands are present they will not alter in appearance unless divided at vitrectomy, and if further contraction occurs redetachment will be prevented only if reattachment of the retina has been firmly established with a high buckle and choroidoretinal adhesion.

Production of massive periretinal proliferation remains the greatest problem to the retinal surgeon. It can arise in the postoperative period in a variety of ways and has been found to occur more often when there have been serious operative complications (Leaver et al., 1975). The end stage situation may arise as follows:

1. A remorseless progression of membranes that were present preoperatively in which retinal surgery has not managed to reattach the retina. Indeed, in some of these cases retinal surgery may have accelerated the progression of the membranes. This is now the commonest clinical situation in which end-stage massive periretinal proliferation comes about.

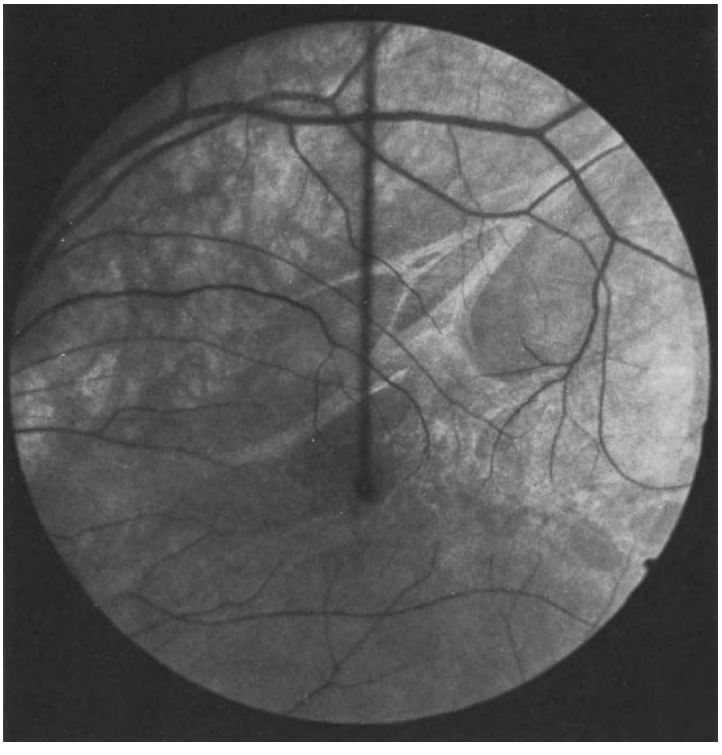

Fig. 5.6. Retroretinal strands seen on the back of a reattached retina

2. The preoperative fibrotic process appears to have been halted by initially successful retinal surgery and the retina is completely reattached for a few weeks only to be followed by a sudden onset of massive fibrosis about two months after surgery.

3. The postoperative appearance of massive fibrosis in what had previously been a retinal detachment uncomplicated by intraocular membranes and apparently successfully treated. This is now an exceedingly rare complication of routine surgery, nevertheless particularly likely to happen with giant tears.

4. The onset of intraocular fibrosis in cases in which there has been no preoperative evidence of such changes but in which there had been failure to reattach the retina. In most of these cases in which the eye is subjected to one or more unsuccessful operative procedures, sooner or later intraocular fibrosis is the inevitable result, emphasising the importance of the initial operation.

Once membranes have appeared in the postoperative period, the usual progression is either to limited traction detachment or more usually to massive periretinal proliferation, unless reattachment can be achieved. Exceptionally, the early appearance of preretinal membrane may spontaneously resolve (Byer, 1973).

Failure in Retinal Detachment Surgery

In most published studies of retinal detachment surgery, success has been defined as anatomical reattachment of the retina for a period of six months from the time of surgery. Judged by these criteria, recent series show a high rate of success and of fresh cases more than 90% can be expected to be successful. In some of the so-called successes, however, return of vision is so poor that it has not made much of a contribution to the patient's life even if the other eye is seeing well, for occasionally a patient will complain that poor vision

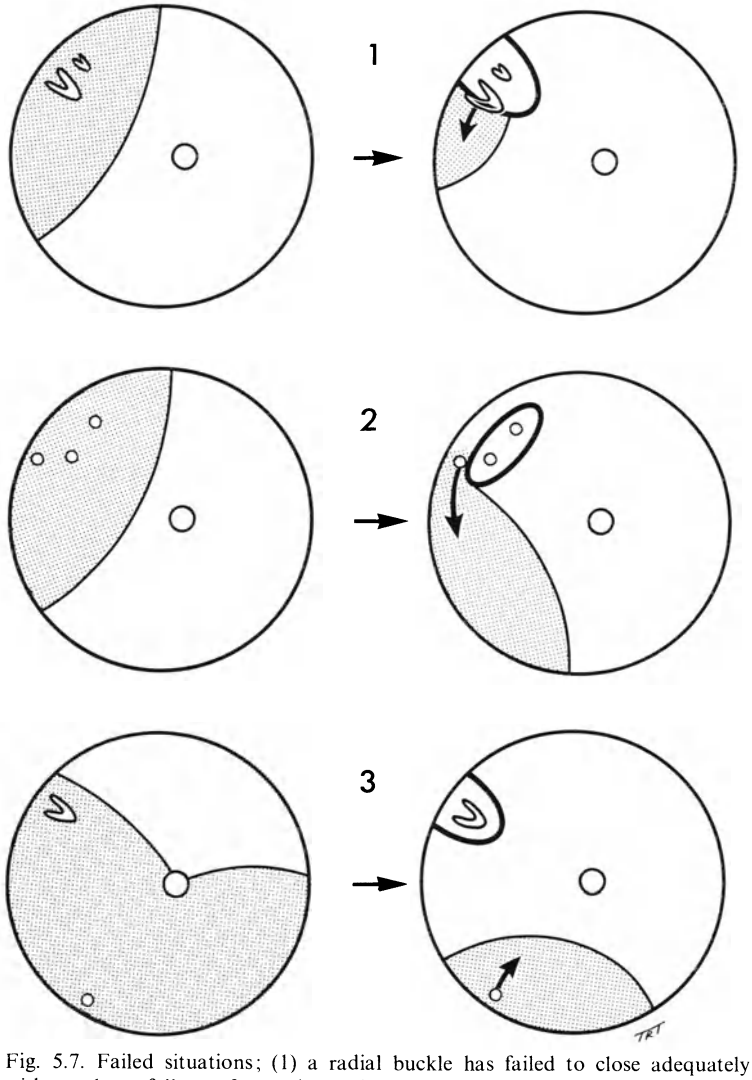

Fig. 5.7. Failed situations; (1) a radial buckle has failed to close adequately a U-shaped tear, with resultant failure of complete subretinal fluid absorption; (2) a circumferential buckle has failed to seal a small anterior round hole; (3) an undetected round hole has prevented the absorption of subretinal fluid

in the operated eye actually interferes with normal vision in the good eye. However, as a rule a successfully reattached retina will give the patient at least an improved field of vision and will also serve as an insurance policy should the other eye subsequently become diseased. In failed cases it becomes a matter of clinical judgement as to whether further surgery should be advised, especially if there is good vision in the other eye. Each case must be carefully considered on its individual merits, and this mainly depends on the patient's age, visual demands, and the state of the other eye. In younger patients reoperation should be advised unless there is no hope of reattachment, but in old patients the enthusiasm of the surgeon to achieve reattachment must be tempered by careful consideration of the risk to the patient and the likelihood of worthwhile return of worthwhile return of visual improvement. In considering the causes of failure of retinal detachment surgery two main groups may be recognised.

Failure to Close the Retinal Hole (Fig. 5.7)

This may arise as a result of

a) uncertainty of the position of the retinal hole;

b) the difficulty of localising the holes at the time of retinal surgery so that a buckle of inadequate dimensions or one that is poorly positioned results;

c) the failure to appreciate the degree of retinal mobility if, when non-drainage operation is used, the hole cannot be closed at the time of surgery, and the retina will not come to lie against the buckle in the postoperative period;

d) the injudicious use of a high circumferential buckle which causes the formation of a radial communicating fold (Fig. 5.8) which has failed to close spontaneously;

e) failure of intraocular gas to tamponade a large or giant tear.

In most of these cases, failure of the detachment operation will usually be apparent within a week or two of the operation, although as a rule there is a preliminary and partial absorption of subretinal fluid followed by reaccumulation. This initial absorption is particularly impressive when the hole has been nartially sealed by the buckling process.

Progressive Intraocular Fibrosis

Not only does progressive traction tend to drag the retina towards the centre of the eye, but it also pulls previously sealed holes away from buckles.

Various series have reported on the reasons for failure and most of these crystallise the day-to-day clinical experience of the retinal detachment surgeon. In one series (Chignell et al., 1973), failure with a first operation was more likely to occur in aphakia and other cases when there was uncertainty in the closure of the retinal holes; for example when there was extensive detachment,

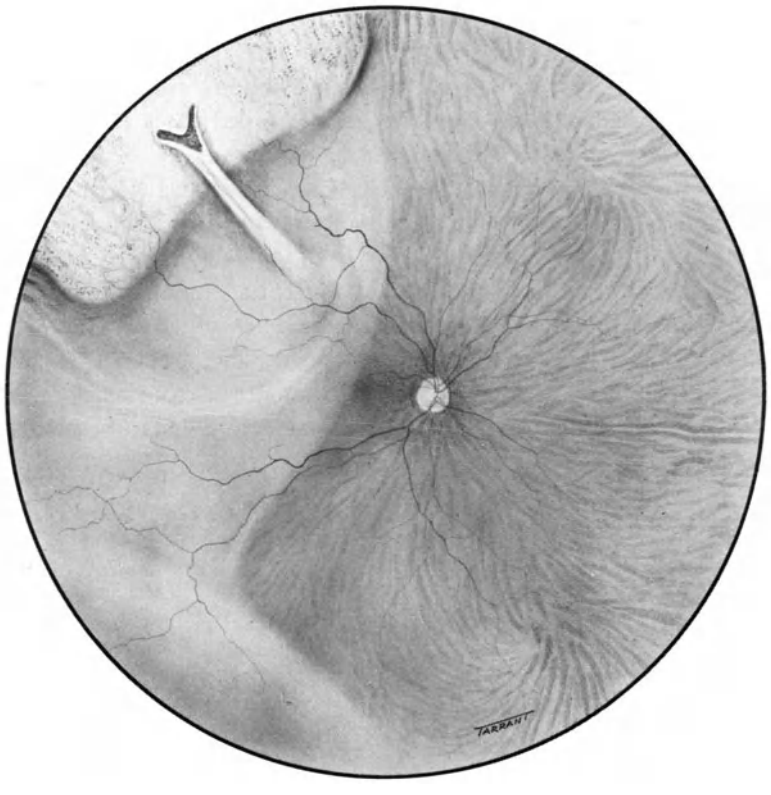

Fig. 5.8. A circumferential buckle has been used to attempt to close a U-shaped tear. A fold connects the tear with subretinal fluid behind the buckle

when there was deep subretinal fluid underneath the retinal tears, making localisation difficult, and when there was marked preoperative membrane formation defying reattachment. Many of these cases were reattached at a second operation but of those that were eventually complete failures the majority had developed massive periretinal proliferation. The high incidence of massive periretinal proliferation in failed cases was also confirmed by Morse (1977).

Surgery of Failed Cases

If no further hole can be seen and there is no increase in the amount of subretinal fluid, it is inadvisable to reoperate unless a cause of failure can be clearly established. On occasions the absorption of subretinal fluid may be slow hence unnecessary intervention should be avoided. Failure is best judged by the reaccumulation of subretinal fluid after initial reabsorption, or by increase in preoperative volume of subretinal fluid after operation. However, when it is clear that the operation has failed and that a further operation will be of benefit, surgery should be carried out as soon as is reasonably possible.

Before reoperation the case must be entirely reassessed with examination by indirect ophthalmoscopy, scleral depression if possible, retinal charting and three-mirror gonioscopy.

Selection of Operation

Photocoagulation

Photocoagulation, using either the Xenon arc or Argon laser – the former is more effective as it is more powerful – can sometimes be used in the postoperative period to promote closure of a retinal hole (Curtin et al., 1967). For this manoeuvre to be successful the buckle must be in the correct position beneath the hole and separated from the hole by only a thin layer of subretinal fluid. Photocoagulation will not be successful in the presence of deep subretinal fluid because the photocoagulation reaction will not produce any observable change in the detached retina. Photocoagulation may be of value when there are radial folds leading from the hole to subretinal fluid behind the buckle (fishmouthing). These folds can sometimes be closed by application of photocoagulation to the area of retina immediately surrounding the hole and will sometimes bring about reabsorption of residual subretinal fluid.

Local retrobulbar anaesthesia is used with patients undergoing photocagulation. The reaction should completely surround the retinal hole, the white rings of reaction in the detached retina just touching each other. Successful closure of the hole will result in rapid disappearance of residual subretinal fluid. Excessively heavy applications should be avoided as there is a risk of inducing the formation of preretinal membrane, or of iatrogenic holes in the retina.

Further Buckling

In most cases it is possible to establish the cause of failure. If an offending hole is identified, then a logical reoperation can be planned. If the hole had previously been missed altogether or had been inadequately buckled with an implant inaccurately placed or of insufficient proportions, then a further simple buckling procedure is all that is necessary to achieve success. In this situation a non-drainage operation may be performed in a substantial number of cases (Leaver et al., 1975). However, it is probable that these are cases that are more likely to have a fibrotic fixed retina, necessitating drainage, than ones in which there has been no previous surgery. If the retinal hole has not been closed in spite of the use of an accurately placed buckle of apparently adequate proportions the failure is usually a result of insufficient mobility of the detached retina so that the retina has not come to lie against the buckle in the postoperative period. In these cases the reoperation should be planned so that closure of the hole is achieved at the time of surgery – often by deepening the buckle,

by drainage of subretinal fluid and, if this does not close the hole, by intraocular injection of air or gas.

If the cause of failure can be established by finding a further hole, then the buckling procedure will be local. An encircling procedure will only be carried out if the site of the retinal hole cannot be seen or deduced, or if marked vitreo-retinal traction has developed.

Follow-up of Patients

Patients should be followed up with the length of time between visits increased until eventually all that is necessary will be an annual assessment to check up on both the operated and the fellow eye. A previous retinal detachment operation should not act as a deterrent to subsequent cataract surgery, should it be necessary, as a very low incidence of redetachment has been reported following extraction (Ackerman et al., 1970), nor should the occurrence of an aphakic detachment prevent cataract extraction in the other eye (Benson et al., 1975).

References

Aaberg, T.M., Pawlowski, G.J.: Exudative retinal detachments following scleral buckling with cryotherapy. Am. J. Opthalmol. *74*, 245–251 (1972)

Ackerman, A.L., Seelenfreund, M.H., Freeman, H.M., Schepens, C.L.: Cataract extraction following retinal detachment surgery. Arch. Ophthalmol. *84*, 41–44 (1970)

Benson, W.E., Grand, A., Okun, E.: Aphakic retinal detachment. Arch. Opthalmol. *93*, 245–249 (1975)

Burton, T.C.: Irregular astigmatism following espicleral buckling procedures. Arch. Opthalmol. *90*, 447–448 (1973)

Byer, N.E.: Spontaneous disappearance of early postoperative preretinal retraction. Arch. Ophthalmol. *90*, 133–135 (1973)

Chignell, A.H.: Choroidal detachment following retinal detachment surgery without drainage of subretinal fluid. Am. J. Ophthalmol. *73*, 860–862 (1972)

Chignell, A.H.: Complications after retinal detachment surgery. Trans. Ophthalmol. Soc. U.K. *95*, 134–137 (1975)

Chignell, A.H., Crewdson, J.F.: Unpublished data (1978)

Chignell, A.H., Easty, D.L.: Iris fluoroscein photography following retinal detachment surgery and in certain ocular ischaemic disorders. Trans. Ophthalmol. Soc. U.K. *91*, 243–259 (1971)

Chignell, A.H., Talbot, J.: Subretinal fluid absorption. Arch. Ophthalmol. *96*, 635–638 (1978)

Chignell, A.H., Revie, I.H.S., Clemett, R.S.: Complications of retinal cryotherapy. Trans. Ophthalmol. Soc. U.K. *91*, 635–651 (1971)

Chignell, A.H., Fison, L.G., Davies, E.W.G., Hartley, R.E., Gundry, M.F.: Failure in retinal detachment surgery. Br. J. Ophthalmol. *57*, 525–530 (1973)

Chisholm, I.A., McClure, E., Foulds, W.S.: Functional recovery of the retina after retinal detachment. Trans. Ophthalmol. Soc. U.K. *95*, 167–172 (1975)

Cleary, P.E., Leaver, P.K.: Macular morphology in the reattached retina. Mod. Probl. Ophthalmol. *18*, 400–407 (1977)

Crews, S.J.: Macular changes following retinal detachment procedures. Mod. Probl. Ophthalmol. *7*, 337–347 (1968)

Crock, G.: Clinical syndromes of anterior segment ischaemia. Trans. Ophthalmol. Soc. U.K. *87*, 513–536 (1967)

Curtin, V.T., Norton, E.W.D., Gass, J.D.H.: Photocoagulation: its use in the prevention of reoperation after scleral buckling operations. Trans. Am. Acad. Ophthalmol. Otolaryngol. *71*, 432–441 (1967)

Dalgleish, R.: Assessment of the intra-scleral silicone rubber implant with encircling band in retinal detachment surgery. Br. J. Ophthalmol. *50*, 245–250 (1966)

Davies, E.W.G.: Factors affecting recovery of visual acuity following detachment. Trans. Ophthalmol. Soc. U.K. *92*, 335–344 (1972)

Easty, D.L., Chignell, A.H.: Fluoroscein angiography in anterior segment ischaemia. Br. J. Ophthalmol. *57*, 18–26 (1973)

Flindall, R.J., Norton, E.W.D., Curtin, V.T. Gass, J.D.M.: Reduction of extrusion and infection following episcleral silicone implants and cryopexy in retinal detachment surgery. Am. J. Ophthalmol. *71*, 835–837 (1971)

Grupposo, S.S.: Visual acuity following surgery for retinal detachments. Arch. Ophthalmol. *93*, 327–330 (1975)

Hamilton, A.M.: Preretinal traction membranes. Trans. Ophthalmol. Soc. U.K. *92*, 387–393 (1972)

Hartley, R.E., Marsh, R.J.: Anterior chamber depth changes after retinal detachment surgery. Br. J. Ophthalmol. *57*, 546 550 (1973)

Hawkins, W.R., Schepens, C.L.: Choroidal detachment and retinal surgery. Am. J. Ophthalmol. *62*, 813–819 (1966)

Hayreh, S.S., Baines, J.A.B.: Occlusion of the vortex veins – an experimental study. Br. J. Ophthalmol. *57*, 217–238 (1973)

Hilton, G.G., Norton, E.W.D.: Juvenile retinal detachment. Mod. Probl. Ophthalmol. *8*, 325–341 (1969)

Hitchings, R.A., Levy, I.S., Chignell, A.H.: Acute infection after retinal detachment surgery. Br. J. Ophthalmol. *58*, 588–590 (1974)

Jarrett, W.H., Brockhurst, R.J.: Unexplained blindness and optic atrophy following retinal detachment surgery. Arch. Ophthalmol. *73*, 782–791 (1965)

Jay, B.: The functional cure of retinal detachments. Trans. Ophthalmol. Soc. U.K. *85*, 101–110 (1971)

Kanski, J.J., Elkington, A.R., Davies, M.S.: Diplopia after retinal detachment surgery. Am. J. Ophthalmol. *76*, 38–40 (1973)

Kreiger, A.E., Hokgkinson, B.J., Fredrick, A.R., Smith, T.R.: Results of retinal detachment surgery. Arch. Ophthalmol. *86*, 385–394 (1971)

Kreissig, I.: Prognosis of return of macular function after retinal reattachment. Mod. Probl. Ophthalmol. *18*, 415–429 (1977)

Kreissig, I., Lincoff, H.A.: Die unaufschiebbare Ablatiooperation. Klin. Monatsbl. Augenheilkd. *163*, 315–318 (1974)

Kutschera, E.: Macular changes in cured retinal detachments. Klin. Monatsbl. Augenheilkd. *152*, 195–203 (1968)

Langston, R., Lincoff, H.A., McLean, J.M.: Scleral abscess 2. Arch. Ophthalmol. *74*, 665–668 (1965)

Lean, J.S., Chignell, A.H.: Infection following retinal detachment surgery. Br. J. Ophthalmol. *61*, 593–594 (1977)

Leaver, P.K., Chignell, A.H., Fison, L.G., Pyne, J.R., Saunders, S.H.: Role of non-drainage of subretinal fluid in reoperation for retinal detachment. Br. J. Ophthalmol. *59*, 252–254 (1975)

Leaver, P.K., Chester, G.K., Saunders, S.H.: Failures influencing absorption of subretinal fluid. Br. J. Ophthalmol. *60*, 559–560 (1976)

Lincoff, H.A., McLean, J.M.: Mechanism of the cryosurgical adhesion. Am. J. Ophthalmol. *67*, 477–493 (1969)

Lincoff, H.A., McLean, J.M., Nano, H.: Scleral abscess 1. Arch. Ophthalmol. *74*, 641–648 (1965)

Lincoff, H.A., O'Connor, P., Bloch, D., Nadel, A., Kreissig, I., Grindberg, M.: The cryosurgical adhesion. Trans. Am. Acad. Ophthalmol. Otolaryngol. *74*, 98–107 (1970)

Morse, P.H.: Encirclement versus 360° buckling and prognostic factors in retinal separation surgery. Trans. Ophthalmol. Soc. U.K. *97*, 36–38 (1977)

Nadel, A., Gieser, R., Lincoff, H.A.: Inferior retinal detachment in young patients. Ann. Ophthalmol. **3**, 1311–1316 (1971)
O'Day, D.M., Galbraith, J.E.K., Crock, G.W., Cairns, J.D.: Treatment of anterior segment necrosis of the eye with low molecular weight Dextran. Lancet *1*, 401–402 (1966)
Roth, A.M., Foos, R.T.: Surface wrinkling retinopathy in eyes enucleated at autopsy. Trans. Am. Acad. Ophthalmol. Otolaryngol. *75*, 1047–1058 (1971)
Rouchy, J.P.: Intolerance to silicone sponges in surgery of reattachment of the retina. Ann. Ocul. *204*, 55–65 (1971)
Rubin, M.L. The induction of refractive errors by retinal detachment surgery. Trans. Am. Ophthalmol. Soc. *3*, 452–490 (1975)
Russo, C.E., Ruiz, R.S.: Silicone sponge rejection. Arch. Ophthalmol. *85*, 647–650 (1971)
Sarin, L.K., McDonald, P.R.: Changes in the posterior pole following successful reattachment of the retina. Trans. Am. Acad. Ophthalmol. Otolaryngol. *74*, 75–79 (1970)
Schwartz, P.L., Pruett, R.C.: Factors influencing retinal detachment after removal of buckling elements. Arch. Ophthalmol. *95*, 804–808 (1977)
Sewell, J., Knobloch, W.H., Eifrig, D.E.: Intraocular muscle imbalance after surgical treatment for retinal detachment. Am. J. Ophthalmol. *78*, 321–323 (1974)
Tanenbaum, H.L., Schepens, C.L., Elzeneiny, I., Freeman, H.M.: Macular pucker following retinal surgery. Arch. Ophthalmol. *83*, 286–293 (1970)
Ulrich, R.A., Burton, T.C.: Infection following scleral buckling procedures. Arch. Ophthalmol. *92*, 213–215 (1974)
Weidenthal, D.: Silicone sponges. Arch. Ophthalmol. *86*, 726 (1971)
Wise, G.N.: Preretinal macular fibrosis. Trans. Ophthalmol. Soc. U.K. *92*, 131–140 (1972)
Wise, A.N., Bellhorn, M.B., Friedman, A.H., Henkind, P.: Preretinal macular fibrosis. Am. J. Ophthalmol. *79*, 349–373 (1975)

Subject Index

S. N. Hassani

Real Time Ophthalmic Ultrasonography

1978. 423 figures. XXI, 214 pages
ISBN 3-540-90138-6

Here is a comprehensive survey of the principles of ultrasound physics and the practical aspects of scanning procedures in the eye and orbit. A new identification system optimizes localization of lesions in these two areas, simplifying-dimensional depiction of ophthalmic pathology.

Methods of examination and the analysis of diagnostic findings are precisely explained with the ophthalmologist and radiologist in mind. A chapter devoted to ophthalmic ultrasonic anatomy highlights normal variants often mistaken for pathologic entities. And a section on ocular manifestations of systemic disorders is only one feature of this book which makes it an authoritative reference for the practicing clinican. *Real Time Ophthalmic Ultrasonography* serves as a link between sonographic imaging and ophthalmic pathology.

Springer-Verlag
Berlin
Heidelberg
New York

Recent Advances in Glaucoma

International Glaucoma Symposium, Prague 1976

Editors: S. Rehák, M. M. Krasnov, G. D. Paterson
1977. 77 figures, 46 tables.
XIII, 295 pages.
(Co-production with Avicenum Verlag für Medizin, Prague)
ISBN 3-540-07944-0
Distribution rights for the socialist countries: Avicenum Verlag für Medizin, Prague

B. von Barsewisch

Perinatal Retinal Haemorrhages

Morphology, Aetiology and Significance

Foreword by O.-E. Lund
1979. 64 figures, 13 plates, 29 tables.
XII, 184 pages
ISBN 3-540-09167-X

Glaucoma Update

International Glaucoma Symposium, Nara/Japan, May 7–11, 1978

Editors: G. K. Krieglstein, W. Leydhecker
1979. 48 figures, 27 tables.
XII, 224 pages
ISBN 3-540-09350-8

Current Research in Ophthalmic Electron Microscopy

Editor: M. Spitznas
1977. 132 figures. VI, 183 pages
ISBN 3-540-08508-4
(Monograph edition of the journal "Albrecht von Graefes Archiv für klinische und experimentelle Ophthalmologie", Vol. 203, issue 3/4)

Current Research in Ophthalmic Electron Microscopy 2

Editor: M. Spitznas
1978. 175 figures, 14 tables.
IV, 216 pages
ISBN 3-540-09160-2
(Monograph edition of the journal "Albrecht von Graefes Archiv für klinische und experimentelle Ophthalmologie", Vol. 208, issues 1–3)

Springer-Verlag
Berlin
Heidelberg
New York