Recent Advances in OPHTHALMOLOGY

EDITED BY
SIDNEY I. DAVIDSON

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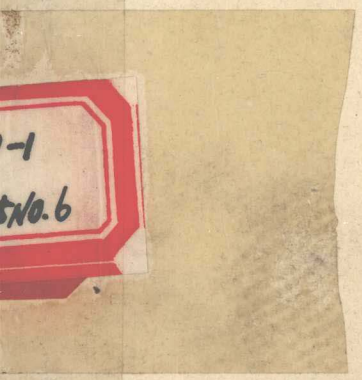

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Preface

With this edition of Recent Advances in Ophthalmology I have endeavoured to revert to the original format as initially conceived by Duke-Elder and Goldsmith by providing an overall view of recent literature in the various branches of ophthalmology, e.g. squint, glaucoma, etc. I felt that this approach would be of greater value to general ophthalmologists who would wish to keep up with recent advances and aspects of the specialty in which they did not have particular expertise. Indeed ophthalmology has shared in the striking advances that have occurred in medicine over the last few years and sub-specialisation is now an accepted fact within the specialty.

As it is intended to publish this volume more frequently no attempt has been made to encompass all the literature since the previous edition, and readers may find the volume somewhat slimmer than its predecessor. This will have the advantage of making it easier to read through completely and so allow the subspecialist who wishes to be informed of current progress in other branches of the specialty to keep up to date.

I had hoped to cover all major aspects of the specialty but unfortunately it did not prove possible to obtain a contribution on the anterior segment in time to allow publication. Indeed my endeavours to obtain such a contribution have delayed this edition more than I would have wished, for I felt that recent developments, particularly in lens surgery, intra-ocular lenses and anterior segment reconstruction, were of paramount importance. It is intended to remedy this omission in the next edition. This delay, in the expectation of providing a chapter on the anterior segment, has resulted in some of the contributions not appearing as up to date as would have been wished but does not reflect on the contributing authors.

I trust that busy clinicians will find this edition of Recent Advances helpful in their practice of our specialty which happily continues to advance, albeit at an accelerating pace.

Liverpool, 1983 S.I.D.

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John D. Scott

INTRODUCTION

It is now sixty-one years since Jules Gonin first operated successfully upon a retinal detachment. Two years later he described to a sceptical ophthalmological world how he had plunged a red-hot needle into the eyes of patients affected by retina detachment successfully closing the break in the retina and curing the problen (Gonin, 1921). History relates the reception which this astounding revelation receive and the manner in which this great surgeon's efforts were developed and finall accepted.

Since those early days of rational treatment much progress has been made and th field of retinal surgery has moved forwards into the vitreous where until very recentl ophthalmic man has feared to tread. What follows is an attempt to assess the preser situation in retinal and vitreous surgery, to see where advances really have been mad and hopefully to assist in putting some controversial issues into perspective. It is not comprehensive discussion of all important aspects of modern retinal practice, an many interesting topics are not mentioned. Certain aspects have been selected as c interest and these are discussed in depth whilst others are briefly described. It i hoped that they will be of interest to general ophthalmic surgeons and help them t understand the issues which their more narrowly orientated friends find stimulatin enough to argue about.

AETIOLOGY

The very close relationship between retina and vitreous has long been recognised a the basis for the production of retinal breaks and detachment, yet the nature of thi liaison is the subject of some confusion and needs to be looked at in more detail.

The high incidence of lattice degeneration in eyes with retinal detachment, betwee 20% and 30% (Michaelson, 1954; Straatsma & Allen, 1962) might be helpful i considering a link between retina and vitreous, however this degeneration occurs i 6% of the normal population (Straatsma & Allen, 1962). It could therefore b concluded that retinal detachment among people with lattice degeneration is very rar indeed. There must then be an additional factor and the evidence suggests that thi lies in the vitreous gel itself which in lattice degeneration is attached to the retin (Byer, 1975). It has been shown that about 12% of eyes affected by an acute an symptomatic vitreous detachment have acute retinal breaks (Tasman, 1968) and tha in those eyes which were found to have lattice degeneration the breaks developed i relation to the degenerative area. Of course retinal breaks occur in the absence o vitreous detachment and in eyes in which no lattice degeneration can be found and i

is for this reason that the idea of the vitreous traction system has been developed (Scott, 1971). It has long been believed that the vitreous is attached to the vitreous base and to the disc and macula and it is certainly true that at the time of vitreous detachment the vitreous separates reluctantly from these attachments. However, before vitreous detachment one can only consider the vitreous gel as attached to the internal limiting membrane of the retina over its entire surface.

It is perhaps easier to think of the vitreous as attached to the retina by connections which have a variable effect on the retina and that this effect is due to changes in the vitreous structure either from normal anatomical variation or to pathological changes. Further one must consider these vitreo-retinal attachments to more remote vitreous gel. In this discussion we shall of course consider pathological attachments.

Predisposing lesions

These are lesions in which ophthalmoscopically obvious degenerations are associated with attachments to vitreous in such a way that vitreous tractions can be exerted upon them; and the commonest lesion in this category is lattice degeneration. However, since 20% to 30% of retinal detachments are associated with lattice degeneration, it follows that more than 50% are not, and although some of these will be associated with non-lattice-like predisposing lesions such as post-inflammatory adhesions, post-vein-occlusive adhesions and familial vitreo-retinal disease, many of them will be eyes with no previously observed disease and therefore showing no predisposing lesion. Of these patients most will be aphakic (Benson et al, 1975); one study showing that in otherwise normal aphakic fellow eyes to eyes affected by aphakic retinal detachment 17 out of 21 detachments occurred as a result of breaks occuring in retina which had previously showed no abnormality. In fact the incidence of lattice degeneration in aphakic retinal detachment is 10% less than in phakic detachment (Ashrafzadeh et al, 1973). Factors other than degenerative vitreo-retinal lesions do therefore play a part in the production of retinal breaks and these are ophthalmoscopically extremely difficult to detect.

Vitreous traction

This is an ill-understood concept, but since it lies at the basis of all retinal break formation leading to retinal detachment, it must be fully discussed. It is in fact quite difficult to produce a retinal break by surgically pulling on the retina. In the diabetic eye for example, trans-vitreal bands of connective tissue slowly shrink and distort the retina but do not cause breaks; in the traumatised eye, similar and at times even stronger bands pull the retina far out of shape but the latter stretches without tearing. In operating upon eyes affected by massive vitreous retraction membranes can be lifted away from retina without causing a break. We must therefore ask how vitreous traction causes a retinal break? The answer would appear to lie in the concept of dynamic vitreous traction. Slow traction over a wide area stretches the retina, and rapid traction in the right direction over a very small area of weakened retina tears it.

Dynamic vitreous traction

Vitreous movement is started by attachments of relatively inelastic gel to the vitreous base and spread by intra-vitreal connections to more distant cortical and nuclear gel (Scott, 1971). The extent of gel movement will depend in the normal eye upon the

mass of gel and the space in which it has to move. The relatively inelastic gel of the base and the vitreous attached to it degenerates slowly and remains a mobile mass, gel elsewhere degenerates as age advances. There is therefore always a mass of gel under the influence of the vitreous base moving with every eye movement. The extent of the mobility of this gel is governed by the change in shape of the gel elsewhere as it moves about, by vitreous degeneration and by abnormalities of the relative vitreous volume itself as in high myopia, trauma involving loss and aphakia especially when complicated by vitreous loss.

Dynamic vitreous traction is therefore a force generated by the vitreous base and propagated through the gel, but acting only when the eye is moving.

A vitreous traction system

This is dynamic traction applied to a point on the retina in which a sheering stress is created at that point of attachment. Whilst this concept allows the idea of vitreo-retinal attachment it must be propagated by means of a system which transfers the force of vitreous movement initiated by the vitreous base to that point on the retina. Thus during sudden vitreous movement the vitreous attached at that point to the retina cannot stretch and a retinal break results.

Trauma

The influence of trauma has long been believed to be paramount in the production of retinal breaks, yet it is the experience of most ophthalmologists that it may be necessary to delve deep into a patient's past before finding any evidence that trauma has occurred, similarly of course patients may experience minor trauma such as a violent cough or minor head injury immediately before the first symptom of the detachment develops. These apparent inconsistencies may be all understood within the concept of dynamic traction upon the retina; whether it be normal rotational eye movement perhaps preloaded by gravity, or by movement of the eye without rotation head movement in the sudden acceleration or deceleration usually associated with injury. Supportive evidence lies in the invariable orientation of a horseshoe break with the operculum anteriorly and the tear lying in the antero-posterior direction.

The influence of gravity has probably been under-estimated and may well have a role to play in the familiar and difficult-to-understand situation familiar to all eye surgeons that breaks most commonly occur in the upper quadrants of the retina.

Break to detachment

The production of a retinal break may now be understood, but the manner in which it goes on to cause a detachment of the retina is much more difficult. In years past it was assumed that all breaks progressed to detachment (Gonin, 1934) but we now know that this is not so. Breaks may be asymptomatic and of these, in one series (Byer, 1974), none of 162 eyes went on to detach; however, in this study only 16% of breaks were operculated. In another series 35% of eyes with symptomatic breaks went on to detach (Davis, 1974). These included 39% with traction breaks and eyes fellow to a detached eye. Different though these studies and figures are, they demonstrate that many retinas with breaks due to traction do not go on to detach. We must therefore ask what is the extra factor which turns a break into a detachment? It is very probably the continuation of the dynamic traction phenomenon already discussed. As the

traction continues to act on the operculum of the break its base is slowly lifted and fluid passes passively beneath the base of the break; the moving mass of gel then begins to include moving retina which in its turn contributes to the inertia system. In time the detachment is propagated by the movements of the retina itself and the final pattern of detachment is determined by a combination of gravity and the position of the break.

Finding the hole

One of the most significant additions to the retinal literature in recent years was presented by Lincoff & Gieser in 1972. It represented the results of testing a concept in retinal detachment contours on 1000 consecutive cases operated upon in one centre. A summary of the findings is given here, but it is strongly urged that all ophthalmic surgeons who operate any retinal detachment should read this work.

Retinal detachments may be divided into three major groups according to the basic contour of the detached area of retina:

1. Superior nasal or temporal detachments localised to one side of the eye.
2. Superior detachments which cross the 12 o'clock meridian and descend on both temporal and nasal sides.
3. Inferior detachments.

The relationship of retinal holes to thes groups of contours is then described.

In superior nasal or temporal detachments the hole is found in a lateral position in the corresponding superior quadrant, and it is found within one and a half hours of the clock of the highest border of the detachment 98% of the time. In a detachment whose distribution of fluid is predominantly down one side of the eye the fluid may cross the midline below and rise on the other side. Should this happen the fluid will rise to the level of the hole on the other side but will not rise to the upper level of the fluid on the other side. A further observation was that in superior breaks lateral to the 11 and 1 o'clock meridians the fluid does not cross the midline of the primary side and there is always a small area of flat retina left related to the 12 o'clock meridian.

Superior detachments that cross the 12 o'clock meridian and spread down both sides of the eye are due to holes which lie close to or at the 12 o'clock position. Further it is these detachments which are total. It was found that 93% of total detachments and those which cross the 12 o'clock meridian were due to holes occurring in a triangle whose apex is at the ora at 12 o'clock and whose base is at the equator between the 11 and 1 o'clock meridians.

Inferior detachments are due to holes below the level of the disc when they are shallow and to holes superiorly when they are bullous. This is a very important observation for it is not at all unusual for a superior hole to spread inferiorly by means of a peripheral sinus of fluid which is hard to appreciate and a mistaken search is made in the lower half of the retina. In inferior detachments due to inferior holes the fluid may rise to different levels on either side of the 6 o'clock meridian; should this occur the side with the higher level of fluid will be the side containing the tear relative to the 6 o'clock meridian. This was found to be true 95% of the time and is therefore a most reliable sign.

The significance of these simple observations cannot be under-estimated and it is surprising that seven years after the initial report some surgeons are not fully

conversant with them. It will be noticed that in no case is the rule 100% reliable and factors such as static traction and retinal lesions modifying the spread of fluid will alter the relationship between retinal detachment contour and position of tear but they lay the foundation for intelligent searching for retinal holes and must thereby improve the standard of detachment surgery.

Primary and secondary breaks

The relation between contour and break described above assumes a single causative lesion in the retina; it is, however, a common observation that often more than one retinal break is present. It is not known whether it is possible for more than one break to be the causative lesion in any one retinal detachment, although I have certainly seen more than one case with two separate detachments due to two individual breaks, one superior and one inferior. However, in detachments with more than one break in the same area of detached retina it is reasonable to assume that only one break actually caused the detachment in the first place. It is for this reason that the concept of primary and secondary breaks is attractive. Eyes are often seen with both asymptomatic and symptomatic breaks in flat retina, but it is unusual for fresh breaks to develop in eyes already affected by flat breaks. In one study 12 eyes out of 176 with flat breaks were found to have additional breaks after the initial examination (Robertson & Norton, 1973); in another study 17 out of 301 eyes developed new breaks from one month to seven years after treatment for an original retinal break (Shapland, 1934) and in yet another study no further breaks were found in a follow-up of 125 eyes with asymptomatic retinal breaks (Byer, 1974).

It might be difficult to decide from these observations whether detachments with multiple breaks develop all of them at the same moment or whether they build up until a particular one provokes a detachment. The last study quoted above would tend to suggest that when breaks develop they do so all at the same time and that one of them acts as the causative or primary lesion. Breaks elsewhere act as secondary lesions which become involved in the detachment as it spreads under the influence of gravity and ocular movement. These secondary breaks however should not be regarded as unimportant and ignored in the treatment of the primary one. If they are not adequately treated they may assume the function of primary breaks and the distribution of fluid will then be related to the position of the new primary break. This is particularly true of surgery without drainage. It is therefore advised that in detachments with multiple breaks all the breaks are treated as potential primaries and that the surgery should be planned with the assumption that all the breaks may contribute equally to the retinal detachment.

Holes, tears and breaks

The terminology of the causative lesion in retinal detachment has caused much confusion over the years. All three terms appear at times to be used interchangeably and may be defined related to symptoms or to the risk of production of a detached retina. No final answer has been forthcoming in the literature and common usage appears to depend largely upon which side of the Atlantic Ocean the problem is discussed. Much of the problem has revolved around which lesion could be responsible for a retinal detachment and which could not, that is a round hole in an area of lattice degeneration was a hole and a horseshoe-shaped hole at the edge of the

same sort of degeneration was a tear; in the former no traction was thought to be involved and in the latter dynamic forces had torn the retina. It is now known that the dividing line between those lesions causing a retinal detachment is much less easy to define, and round apparently degenerative holes may produce a detachment as well as the more obvious operculated tear (Robertson, 1973). It would seem therefore that the use of the term retinal break overcomes the difficulty and allows a single term for a lesion which may be the primary cause of a retinal detachment.

Prophylaxis

Before coming to the interesting subject of surgery for the established detachment and its complications, it would be appropriate to consider ways of preventing it. The problem is that there are several causes of retinal detachment and of the breaks which precede it, and, in addition, as observed already, not all retinal breaks appear to be preceded by a recognisable predisposing lesion. It is therefore only possible to assess a risk in any eye based on a very limited set of factors. These factors are those most likely to produce a retinal detachment, i.e. retinal degenerations known to proceed to break formation; retinal breaks themselves and other non-degenerative high-risk factors.

Argument has long raged regarding the need to treat equatorial retinal degeneration to prevent tearing. However, a close look at well-known figures may help to resolve the problem. Although these degeneration are common in eyes with retinal detachment and are very common in the population at large, the great rarity of retinal detachment in the population would indicate that equatorial degenerations are very rarely associated with the development of detachment and that they only require treatment in high-risk situations (Okun, 1960). These include: patients with a strong family history of retinal detachment in whom equatorial degeneration is found; those in whom cataract extraction is likely in the foreseeable future; and those patients who have had a detachment on the opposite side due to equatorial degeneration, in whose fellow eye the vitreous has not yet detached. These factors are discussed below.

The treatment of established retinal breaks has been well summarised in one study (Byer, 1974). Based on observations on 162 retinal breaks the conclusion was that asymptomatic retinal breaks do not need treating and that those with symptoms attributable to breaks from dynamic traction should be treated prophylactically. It was however recommended that if the asymptomatic break was unusually large, located quite far posteriorly or was associated with a progressing retinal detachment then treatment should be carried out even in the absence of symptoms.

The distinction between an asymptomatic and a symptomatic break is at times difficult to determine, but it is most important since it is reasonable to regard the latter as a break associated with dynamic traction. Symptoms of a fresh break of this kind are those leading the patient to seek medical advice and which are examined within a time limit. Davis (1974) suggested six weeks. In this study the commonest cause of symptoms was vitreous haemorrhage. Less common were floaters and light flashes due to vitreous detachment.

High risk factors include those associated with aphakia. It has been known for very many years that removal of the lens is associated with an increased risk of retinal detachment and that an even higher risk is incurred if vitreous is lost at the time of operation. Quite why this should be has not been adequately explained in the

literature. Discussions have usually revolved around the influence of surgical technique on the likely development of a detachment. Two main points would arise from this assertion, the first being the fact that over a period of thirty years when cataract technique had changed greatly no significant change occurred in the incidence of aphakic retinal detachment (Francois, 1965; Cambiaggi, 1964), only when extra-capsular methods are not followed by capsulotomy is there any apparent protection. The second is that, except in high myopia, there is often a delay of a year or more between cataract surgery and the development of the detachment. For these two reasons and for others it is hard to accept the importance of the role of basic surgical technique in the increased incidence of detachment in aphakic eyes.

We should now look at the influence of myopia on aphakia, for it is known that myopia is an important predisposing factor in phakic detachment (Francois, 1965). If myopic eyes are excluded from consideration in the incidence of aphakic detachment then a more consistent figure of about 0.3% is found. By contrast if eyes with six or more dioptres of myopia are included then one study showed an incidence of nearly 7% retinal detachment after extraction; furthermore, this same study showed that age had an important bearing for all their patients were under 63 years of age and that the average age was only 56 (Hyams et al, 1975). They went on to demonstrate that in their group of myopic and aphakic patients the retinal detachments tended to occur relatively soon after lens surgery.

As in phakic eyes the presence of retinal degeneration or even breaks is not a very helpful indicator for prophylactic treatment. One study found an incidence of retinal breaks in aphakic eyes with myopia of six or more dioptres to be more than 18% (Hyams et al, 1975) and this compares with an incidence of 11% in phakic myopic eyes found by the same observers in another study (Hyams & Neumann, 1969). The latter study is of particular interest for it goes on to demonstrate that in the 18% of eyes with retinal breaks none went on to develop a detachment. Previous work had shown that most detachments were caused by fresh as opposed to established retinal breaks (Hyams et al, 1974), suggesting that in highly myopic aphakic eyes there is a greater tendency for retinal breaks to go straight on to detachment. It follows therefore that if a break is found in an undetached aphakic eye there is little indication for prophylactic treatment.

It will be noticed that in the discussion of the risk factors in aphakia, no mention has been made of equatorial degeneration. This factor is very difficult to assess. It has already been stated that the incidence of equatorial degeneration in detached aphakic eyes is significantly less than in phakic eyes (Ashrafzadeh et al, 1973), but no prospective study has to my knowledge ever been reported. It cannot be said therefore that the presence of equatorial degeneration does or does not indicate an increased likelihood of detachment as a result of cataract extraction.

The role played by posterior vitreous detachment has been studied and some years ago one report looked at fresh and symptomatic vitreous detachment to examine the risk of development of a retinal break (Tasman, 1968). It was found that 12% developed retinal breaks and that these were more likely to occur in relation to equatorial degeneration, if this were present. Retinal breaks could develop up to three weeks after vitreous detachment but in two eyes asymptomatic breaks occurred several months later. This study may be compared with another more recent one (Friedman & Neumann, 1975); here a number of aphakic eyes without vitreous

detachment were observed over a period of up to six years. It was shown that vitreous detachment can proceed slowly in aphakic eyes, that in non-myopic eyes the occurrence of vitreous detachment rarely proceeded to detachment even when fresh break formation occurred and that if vitreous detachment is found to be complete then the risk of fresh break formation is negligible.

One study of particular importance concerned the management of the fellow eye in which the first eye had suffered an aphakic retinal detachment (Benson et al, 1975). This is the first time that such a prospective study had been reported and the findings need to be looked at in detail. The first conclusion by the authors was that the second eye of a patient affected by aphakic detachment in the fellow eye was at risk even before cataract extraction was carried out. Two out of fifty were in this category and of these one had a normal fundus before detaching. The second finding was that of seventy-seven patients who were aphakic bilaterally, twelve went on to detach the second eye and of these, nine had a clinically normal retina. Thirdly, a group of thirty-four patients were examined who underwent cataract extraction in the second eye; of these seven patients detached and three of these had normal fundi. These figures excluded a number of patients in each group who underwent prophylactic treatment to equatorial degeneration and retinal breaks and of these there were two out of eleven in the third.

This important study has shown one most interesting and new phenomenon which is the high rate of retinal detachment in the fellow eye to one affected by aphakic detachment, of detachment before the lens is removed. It also confirms the difficulty in predicting fellow eyes at risk when breaks leading to detachment occur in clinically normal retina. The desirability of treating prophylactically depends on the risk if it is not done, on the complications of treatment and on its effectiveness in preventing the problem.

The desirability of prophylactic treatment has perhaps been demonstrated in the foregoing discussion. The complications of treatment have changes greatly in recent years. For some time photocoagulation was the only modality available and there were well established complications from this form of treatment. Secondary breaks have been described (Meyer-Schwickerath, 1960) and also macular pucker (McDonald & Tasman, 1967), and an occasional report of corneal complications has appeared in the literature (Pfister et al, 1971). Xenon-arc light has been the source of radiation in early series but in the early 1960s this was replaced in some clinics by cryotherapy and later by argon laser radiation. Complications have been described in all modalities of treatment and these are found at a rate of about 2% in each (Benson et al, 1977), but it should emphasised that these figures refer to treated retinal breaks alone, some of which had early and localised retinal detachment. In our department no complications have occurred in 300 prophylactic cryotherapy treatments carried out over the last four years for a variety of indications to be referred to below.

The effectiveness of treatment can only be accepted if no failures whatsoever are found within a reasonable time of follow-up. In all studies of retinal breaks treated prophylactically, so far none can report complete success. The average failure rate is at least 2% or the same as the rate of complication. The reason for this failure is the occurrence of fresh breaks in retina previously considered as normal at the time of initial treatment. A way of anticipating this phenomenon must be found therefore, and I believe that this lies in the technique of treatment. It has been known for some

years that more than 98% of retinal tears lie either at the equator or close to the ora serrata (Lincoff & Gieser, 1972), these two areas can therefore be reasonably expected to harbour most breaks when they occur. Treatment therefore should be aimed at this part of the retina. In the absence of any retinal degeneration the exact spot where a break will develop cannot be determined so all the retinal periphery must be treated and one row, either at the equator or just posterior to the ora, is applied in a continuous ring.

This technique has been the basis of our prophylactic management for the last four years and no complication has arisen as a result of it. In comparing complications of cryotherapy quoted by other authors, one must bear in mind that there is a difference pathologically between an intact retina treated before tear formation and the treatment of an existing retinal break. It may justifiably be said that once the retina is torn one passes from the stage of prophylaxis to the stage of treatment.

From the foregoing argument the following catergories of eye should be treated:

1. Symptomatic retinal breaks should be treated directly, and if other areas of retinal degeneration or break are found they may be treated as well. Breaks can be treated locally with the laser.
2. A fellow eye with equatorial degeneration and without vitreous detachment, where the first eye has been affected by retinal detachment due to a break resulting from equatorial degeneration.
3. A highly myopic eye with or without degeneration likely to undergo cataract extraction in the near future.
4. A fellow eye to one affected by aphakic retinal detachment, if that eye is to undergo cataract extraction or if that eye has equatorial degeneration in the absence of vitreous detachment.
5. An eye with equatorial degeneration and without vitreous detachment about to undergo cataract extraction.
6. Any eye with retinal degeneration of any predisposing kind in the presence of a family history of retinal detachment.
7. The fellow eye to one affected by a giant equatorial retinal break.
8. Any eye previously treated for congenital cataract, and any eye which has lost vitreous gel at the time of cataract surgery.
9. Any eye which has suffered perforating injury involving the vitreous base should be treated as soon as possible after the injury has adequately healed.

It will be seen then that there are a large number of eyes which we believe should be treated prophylactically. The technique will vary between treating equatorial degeneration directly and treating the entire retinal periphery in one layer through 360° in one sitting. The latter method is safe if the lesions are kept to a minimum intensity and it is believed that cryotherapy is the best modality to use. General anaesthesia may often be needed and a conjunctival incision in each quadrant is also justified. It reduces post-operative oedema which always occurs if trans-conjunctival treatment is used and it heals within a few days. Furthermore, the lesion produced by trans-conjunctival cryotherapy is very difficult to control in intensity and is much smaller in diameter. More lesions and therefore more conjunctival oedema will result.

Prophylaxis will mostly be aimed at preventing the more problematical retinal detachments. These will include those associated with aphakia and congenital cataract

surgery and giant breaks, and if these can at least be eliminated from the fellow eye it will have been well worth-while.

The surgery of retinal detachment

The principles of modern retinal surgery are now well accepted, and surgeons using scleral buckling, cryotherapy and perhaps drainage of sub-retinal fluid will expect to achieve a final success rate of well over 90%. It must not be forgotten however that fifty years ago good results with up to 80% success were being obtained in selected cases with diathermy and drainage. Later scleral resection was introduced to reduce eye volume since it was believed that scleral stretch was the prime cause of retinal detachment. Scleral buckling came upon the scene almost by accident when it was discovered that a secondary effect of the scleral resection was to create a bump inside the eye and that with careful manipulation this bump could be made to coincide with the tear. Resected bumps were short lasting, and this led Custodis to develop the plomb. From that time to the present there has been little limit to the variety of material used as a plomb to create the bump and to suture or stick it to the sclera. It is not proposed to review these at all here, the principle is a valid one and is accepted.

It is worth while discussing three important aspects of surgery for uncomplicated detachments which are still the subject for controversy even some years after their first introduction, but first it would be as well to define an *uncomplicated retinal detachment*. This is a detachment due to a retinal break which has not been complicated by secondary vitreous changes, by the effects of gravity or by its position in the fundus so that conventional scleral buckling techniques cannot easily close the break and flatten the retina.

The three aspects referred to are the orientation of the buckle, the need for an encircling element and the need to drain sub-retinal fluid.

The orientation of the buckle

At one time the direction of the longest axis of a buckle was determined by the longest axis of the retinal break, and if there was any doubt a circumferential direction seems to have been preferred. Since those days and particularly since the development of surgery without drainage the tendency has been towards radial buckling whenever possible. The reason for this is probably twofold. Firstly if a buckle is placed radially it is very much easier to localise the exact meridian in which the break lies. If the buckle is placed circumferentially the localisation of the distance from the limbus is very much more difficult to assess and conversely there is less need for accuracy in the choice of meridian. This effect is magnified by the need to buckle in the direction of the traction. This is always in the antero-posterior meridian. The second reason is more rational and concerns itself with the relationship which results from the use of scleral sutures in attaching an episcleral silicone sponge to the eye. The need for mattress sutures with a long intrascleral travel results in a relative shortening of the sclera in the direction of travel of the intrascleral portion of the suture. The part that crosses the silicone sponges is prevented from shortening by the presence of the sponge itself. This unidirectional shortening has the effect of creating a fold in the retina at right angles to the intrascleral portion of the suture. Thus a circumferential plomb will tend to create radial folds and radial ones circumferential folds. If there is any tendency for a horseshoe break to shorten across its operculum as is often the case

then a fold may develop if a circumferential buckle is used which will relate to the centre of the break and a potential leak may be created. This phenomenon is referred to as fish-mouthing and may be prevented by the use of radial sutures over radially placed plombs. This problem is discussed at length in a study devoted to the theoretical and practical aspects of radial buckling (Lincoff & Kreissig, 1975).

There are of course certain breaks which do not lend themselves at all to radial buckles and these may be retinal dialyses or breaks occurring at the posterior edge of lattice degeneration where the long axis of the break may be very long indeed.

In placing radial plomb sutures it may be very difficult to pass the radial intrascleral portion of the suture in the direction from front to back safely, should this be so a very simple and little-known trick is to pass both limbs of the suture from anterior to posterior and to join the posterior loops to create a mattress suture with knots across the plomb both anteriorly and posteriorly.

There will be times when the break is so large that available silicone plombs are too small to cover the break. It is generally recommended that there is an overlap of 1.5 mm on either side of the localised break (Scott, 1970) and it possible to place a maximum of two 5 mm plombs side by side to give about 10 mm of radial buckle. More than this will require special techniques. The method of choice is the episcleral pouch (Paufique et al, 1966; Spitznas et al, 1973) which is constructed from donor sclera and sutured to the sclera with a shortening factor created by stretching the donor sclera as it is attached to the host sclera. The pouch may be packed full with any biologically acceptable material which in practice is usually silicone sponge. The pouch has no limits in size and is extremely useful when commercial sponge is not large enough.

The use of encircling elements

This is much abused and probably stems mainly from a native insecurity in many surgeons minds when they approach anything but the most localised detachment. It is hoped that if a band is placed at the equator it will take account of any unseen retinal break that may lurk to trap the unwary and these will be sealed safely.

Encircling bands were introduced in the late 1950s to treat cases thought to have poor prognosis and which seemed to require a buckle which was permanent (Schepens et al, 1957). The band had the effect of preventing loosening of the sutures holding the scleral resection or the silicone rubber plomb pocket together. Since this time there has been a tendency to believe that any detachment can be treated this way. Nothing could be further from the truth for there are some cases in which an encircling element is definitely contraindicated. An example of this is the retinal dialysis where a localised and extremely anterior buckle is required.

It would be helpful to review our current concepts of the function of encircling elements for they differ considerably from traditional attitudes. An encircling element may take two forms, a silicone rubber band placed at the equator of the eye, fixed to the sclera by two anchor sutures in each quadrant and relying for its constricting effect upon the elastic properties of the rubber. After placement there will be no tension within the band for at equilibrium with the intra-ocular tension pressing equally around the band there will be very little force on each millimetre of the band to stretch it. The second form is an encircling buckle of silicone sponge attached to the sclera by mattress sutures. This may be placed at any distance from the limbus for it relies for