Eye (2002) 16, 349–352

www.nature.com/eye

Future perspectives in primary retinal detachment repair

JD Scott

Eye (2002) **16,** 349–352. doi:10.1038/sj.eye.6700187

It is an interesting challenge to be asked to look into the future. To try to achieve this requires some understanding of the past and the present, in order to analyse the rate of change. From here one can try to predict how things might evolve in the future.

About 100 years ago the International Congress in Paris officially declared that retinal detachment was untreatable. It is significant that the President of the Congress at that time was Professor Marc Dufour. Professor Dufour's First Assistant at that time was Dr Jules Gonin. This was certainly the stimulus for his going back to Lausanne to look for an effective treatment. The fact that he was successful is history and well known to every one of us here today. It is also noteworthy that he published a paper in that very year, 1904, on the role of the vitreous in different types of retinal detachment.

In 1920 Gonin described, for the first time, successful surgery for the repair of retinal detachment by treating the causative retinal break. The French Society was outraged by this suggestion and it took nearly 10 years before the rest of the ophthalmic world believed him.

What is worth emphasising however is the fact that Gonin was able to get over 60% of all his cases successfully reattached and using only accurate localisation, drainage of fluid using cautery directly beneath the retinal break, and postoperative bed rest. His friend and collaborator Dr Weve reported an 80% success rate in what he called 'favourable cases', and remarkably this was without scleral buckles, or any form of tamponade.

So Gonin and his friends had an overall failure rate of about 30% using comparatively simple but very accurate techniques. So are we any better off after a lapse of 70 or so years? The answer is perhaps not quite as much as you might think. We still have an overall primary failure rate of about 20%, despite all our efforts. There are possibly several reasons for this apparent lack of progress.

Inaccuracies in technique and avoidable surgical complications lead to failure. This has been demonstrated in two papers published 25 years apart in which primary failure was about the same at 25%,[1,2] and this was acknowledged to be due mostly to avoidable problems. In 1989 at the inaugural meeting of the College of Ophthalmologists I looked at the causes of failure in patients referred for the treatment of recurrent detachment.[3] Most were due to inaccurate or inappropriate surgery, some to a failure to find the causative break and only 10% to presumed breaks that I could not find at subsequent surgery.

Our own experience here in Cambridge has shown we are unable to do better than an overall 8% primary failure rate, reducing to 1% by the second operation. These figures are however affected by a number of recurrences following silicone removal as the second stage of a planned two-stage procedure for more complex problems so the overall failure rate remains at about 10%.

Is it just possible therefore that the solution to some causes for failure may lie in the hands of the surgeon, and not as a result of factors beyond our control?

In comparing our figures with those achieved by Gonin and his colleagues one might think that their surprisingly good results were due to case selection. Gonin's book published in 1934 demonstrates very clearly that this is not the case. He operated on all comers and understood all about the problems of inaccurate surgery and even of proliferative vitreoretinopathy (PVR).

Which brings us on to the problem of proliferation. It is taught that about 10% of retinal detachment operations fail due to PVR, and the implication seems to be that there is very little that can be done about it. Various

Department of Ophthalmology
Addenbrooke's Hospital NHS Trust
Cambridge, UK

Correspondence:
JD Scott MA FRCS FRCOphth
Low Brooms
38 High Street
Little Shelford
Cambridge CB2 5ES
UK
Tel: 10223 840558
Fax: 01223 845434
E-mail: john.scott@retina-service.freeserve.co.uk

attempts have been made in the past to blame aspects of technique such as drainage of fluid and cryotherapy. These were not supported by reliable evidence and served only to deflect our attentions from the reality that PVR is mostly the result of failed surgery, and not the cause of it.

Proliferation complicates possibly all retinal detachment to a greater or lesser extent. What is important is that it should be controlled and not allowed to affect the outcome. This means that appropriate and successful primary surgery should be carried out as soon as possible by competent, experienced and well trained surgeons.

We actually understand very little about proliferation complicating retinal detachment. We do know something about the cells involved and how they behave, and we know that there are many growth factors, which may be involved in proliferation. However we know nothing about the biochemical cascade, which must be involved, or even how it originates as the retina detaches.

We do not know why or when it is switched on, nor how or even whether it is switched off. Until we understand all of these problems I believe that the development of protocols for the treatment of PVR using anti-proliferative agents should be restricted to the complications of penetrating trauma.

Prevention is always likely to be far more effective than treatment using potentially toxic medicine, and here in Cambridge an independently conducted audit of our results has demonstrated that the incidence of PVR complicating primary retinal detachment can be reduced to less than 0.5% by more effective and timely primary surgery.

The third and no less important factor in our discussion of failure is an understanding of the causation of retinal tears and their progress to detachment. There have been many hypotheses but very little good evidence as to causation.

There are many ways in which a retinal break may occur, with varying pathogenesis. Posterior vitreous detachment (PVD) is responsible for horseshoe-shaped tears, very large ragged tears and giant oral tears. Round retinal holes and retinal dialyses are not due to PVD. The question of the causation of PVD is very important since this process is involved in other sight-threatening disease processes, such as the late complications of diabetic retinopathy.

What then exactly is PVD? Why does the vitreous separate from the retina on a certain day in a person's life. Why is most PVD asymptomatic, and why do only a few present with a flashing light and floaters? Why do only very few people develop retinal tearing as a complication of an event which affects more than two thirds of us during our lifetime? So what is different about those retinas that tear, compared with those that do not? So many questions and so few answers and I will return to this problem later.

The next question relates to how the break progresses to retinal detachment. It has long been recognised that many retinal breaks do not progress, so why should this be? Why can a very small break in the 12 o'clock meridian cause a total detachment and yet a very large ragged break not do so. These are intriguing questions the answer to which is vital to our management of retinal breaks and their resultant detachment.

Next, what are the significant risk factors that are involved in those people who suffer pathological posterior vitreous detachment? We are still taught that cataract surgery and YAG laser capsulotomy are risk factors. We know that retinal detachments occurring in patients who have undergone such procedures are invariably due to PVD-related tears. It follows then that if there is any causative factor which is related to cataract surgery then it must be demonstrated that not only does this factor lead to PVD but that the separation of the vitreous from the retina becomes pathological. This link has never been demonstrated.

The belief that cataract surgery is a risk factor for retinal detachment has existed since 1929 when Bauermann reported a 2% incidence after extracapsular surgery with a 10% detachment rate if vitreous was lost. Benson in 1968 showed that the risk to the fellow eye following aphakic retinal detachment was 7% before cataract surgery and 14% afterwards, which appeared to show that the risk doubled if cataract surgery was performed. In 1989 I looked at the whole problem of retinal detachment in patients affected by cataract and found that the risk of retinal detachment seemed to be the same before as after cataract surgery, especially in the presence of nuclear cataract in younger people.

As far as YAG laser capsulotomy is concerned, Richard Sheard has carried out a prospective study on patients treated by laser capsulotomy looking particularly at the association with posterior vitreous detachment. He has demonstrated that the incidence of PVD increases after cataract surgery but that there is no statistically significant increase in the risk of retinal tearing or detachment following YAG laser capsulotomy.

So if surgery itself is not the cause what then is the reason for the apparent association between cataract and retinal detachment? The answer must lie in the nature of posterior vitreous detachment, and what distinguishes pathological from non-pathological PVD.

It is remarkable how little has been written on the

subject of PVD. There have been some studies concerning the significance of symptoms, and a few have looked at the management of symptomatic PVD. Other than this there has been very little until Martin Snead joined my team 10 years ago.

Martin described his clinico-pathological study, the results of which suggested that the posterior hyaloid membrane consists largely of the separated internal limiting membrane (ILM). This was a significant step forward in our own understanding of PVD. However it still did not answer the dilemma as to why the ILM separates from the retina in this way and on a certain day in a person's life.

So is the separation of the internal limiting membrane from the retina a passive event, due perhaps to some degenerative process taking place in the adhesion between the ILM and underlying cells?

Martin Snead has shown that the classical Weiss ring results from an opacification of the posterior hyaloid membrane due to a dense concentration of cells that is associated with the separation of the ILM from the optic disc.

These appear to be part of a population of similar cells, which lie throughout the posterior hyaloid membrane. They are found within, as well as on the surface of the membrane. The cell bodies are few and far between except at the disc, but their cytoplasm may extend across wide areas, perhaps throughout the ILM and over the entire retinal surface. We have called these cells 'laminocytes' although how they relate to what have been called hyalocytes by Balaazs or glial cells found after membrane separation following surgery for macular holes, remains to be seen.

The second observation is the appearance of the membrane itself on the slit lamp. It usually shows fine wrinkling and folding suggesting that some sort of contraction has taken place. Cell bodies can often be seen associated with the membrane and these may well be the nuclei of laminocytes spread more thinly over the peripheral areas of the membrane.

The third observation is that when the membrane separates incompletely from the retina, the remaining attachment at the macula leads to cellophane maculopathy in which the surface membrane contracts distorting the retina, often affecting vision.

I am suggesting that posterior vitreous detachment is not a passive degenerative process at all, but the result of an active contraction of laminocytes associated with the membrane.

PVD seems to begin posteriorly and spread peripherally. Whether a Weiss ring forms is dependent on whether the ILM separates cleanly from the edges of the disc and subsequently rolls up and opacifies or whether the tear in the ILM occurs peripheral to the disc leaving membrane attached to the disc and surrounding retina. If the latter occurs then the hole in the membrane is large and does not opacify so readily.

What does seem to be certain is that PVD is often a violent event within the eye, and sufficiently so to tear the tough internal limiting membrane at the posterior pole. How rapidly the membrane cells contract may determine whether symptoms such as the flashing light occur. The latter is however not an indication of retinal pathology.

This then perhaps answers the question as to what happens as the ILM separates from the retina to form posterior vitreous detachment. The question it does not answer is why these cells should contract in this way en masse. Neither does it answer the all-important question as to why most vitreous detachments are non-pathological and only a few lead to retinal tearing. There are however a few observations which may be significant.

The first is that horseshoe tears form only at the peripheral attachment of the posterior hyaloid membrane to the retina, at what is normally called the equator. It is very likely that this is related to the reason why the membrane does not normally separate anterior to this location, and to the behaviour of laminocytes.

The second observation comes from the appearance of the tear itself and particularly its many variations. We need to ask ourselves the basic question as to why most PVD-related tears are in fact horseshoe-shaped, and indeed why they are always orientated with the flap lying anteriorly.

It is interesting to note the variety of presentation of retinal tearing. Many are horseshoe-shaped, some have a tendency to be more linear, illustrated very well in drawings by Gonin. When the tear has a curved edge, these are usually retracted and the flap is often distorted, and may separate completely in a very similar way to a macular hole. Frequently there are secondary tears within and around the primary tear. We even occasionally see a horseshoe tear within the flap of a larger tear.

All this strongly suggests to me that the edges of these tears are under the influence of tangential radial retraction.

Peripheral PVD-related tears may be very large and ragged and these are usually associated with rolling of the posterior edge. When such tears develop they always show early retraction of their edges.

I believe that the causation of horseshoe-shaped and allied retinal tears is related to a localised cellular contractual process at the boundary of attached and detached ILM and is associated with the contraction of cells which themselves cause PVD. In this respect it is

interesting to note that if vitreous detachment is pathological and symptomatic, most of the retinal tearing occurs in a very close time relationship to this event.

It could well be that it is the distribution and concentration of laminocytes, together with the nature of the adhesion between the ILM and underlying cells, which determines whether or not the retina tears in PVD. The risk factors that determine whether the retina tears or not may be bound up with the distribution and behaviour of these cells.

An understanding of the process of retinal tear formation is perhaps one of the most important factors towards improving results. After we have eliminated avoidable surgical inaccuracy and complications, we are still left with a number of cases where further breaks are discovered after the first procedure. Some of these may be missed breaks but many are undoubtedly new ones. To understand this problem we must know the exact relationship between PVD and break formation.

For example, is PVD a continuous process or may it proceed in phases in some people? Do most retinal breaks occur during the initial contractual period, so that once complete the risk recedes? I think it is very probable that most tears occur at the time of PVD and that new tears occur as a result of further activity of laminocytes. If this is the case then we need to know a lot more about the time scale over which PVD occurs, since it may have considerable relevance to the timing of primary surgery if this is to be carried out after all the tears that are going to occur have done so.

So where does all this speculation lead us? It is this. If PVD-related retinal breaks are the result of an active contraction of an identifiable group of cells then it might be possible to prevent this contraction pharmacologically. And herein lies the possibility of truly preventing retinal detachment as well as other complications of PVD.

I do not believe that the future should lie in the search for more sophisticated vitrectomy instruments, nor in the investigation of anti-proliferative substances to treat recurrent retinal detachment. Rather we should first be looking towards the improvement of our understanding and approach to primary retinal detachment.

There is a huge variation in the presentation of retinal detachment, from the apparently simplest single break with a localised detachment to an only eye affected by a 360° giant retinal tear. Some cases may have huge numbers of breaks, even in both eyes at once.

There is no one single technique that can deal with all these problems. There is however an approach which says that the goal has to be to get it right first time most effectively, with no risk of recurrence and least risk of significant complication. We may dispute the method by which this may be achieved, but I doubt whether any one of us would argue with the intention.

It would be good to be able to live up to Weve's prediction in 1930 that the coming generation would see a 100% success rate. These aspirations can only be achieved if we accept the fact that failure of primary surgery is rarely the result of factors which are beyond our control.

Although this presentation has been my personal view of present and future perspectives in retinal detachment surgery we should not forget the debt we all owe to Jules Gonin. His vast experience and knowledge of pathology remains as relevant to today's practice as it did 70 years ago. His struggle to convince a sceptical ophthalmic world that what he was doing was right has always been an inspiration to me. He worked alone with the support of a very small number of colleagues who shared his expertise, in the knowledge that nobody else had a solution for what had previously been an untreatable condition.

References

1 Chignell AH, Fison LG, Davies EW, Hartley RE, Gundry MF. Failure in retinal detachment surgery. *Br J Ophthalmol* 1973; **57**: 525–530.
2 Sullivan PM, Luff AJ, Aylward GW. *Eye* 1997; **11**: 869–871.
3 Scott JD. Prevention and perspective in retinal detachment. *Eye* 1989; **3**: 491–515.