

Vitreous and peripheral retinal conditions

Investigation of flashes & floaters; PVD; retinal tears; retinal detachment, retinal tufts; chorioretinal degenerations; pigmented lesions

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Introduction

'Vitreous floaters' is one of the most common presenting symptoms reported to optometrists by patients attending for an eye examination. The challenge for the optometrist is to correctly differentially diagnose, as far as is possible, the cause of the floaters and also any associated symptoms such as 'flashes of light' so that appropriate management decisions may be taken.

The cause of floaters varies from a benign age related vitreous degeneration and uncomplicated posterior vitreous detachment (PVD) to a complicated PVD with a retinal tear and a retinal detachment.

Adequate investigation and management is essential because although vitreous degeneration and PVD are both common and commonly uncomplicated, it is also frequently associated with retinal breaks some of which lead to retinal detachment.

This lecture will

- discuss techniques for examining the vitreous and retina
- review the anatomy of the vitreous and retina
- discuss the pathophysiology of vitreous degeneration, PVD, retinal tears and retinal detachment and
- advise on strategies for the optometric management of patients with these conditions

Techniques for examining the vitreous and retina

Direct ophthalmoscopy is not adequate to view the peripheral retina either with or without a dilated pupil. As the vast majority of retinal breaks occur beyond the retinal equator it is essential to dilate the pupil and use indirect ophthalmoscopy whenever a patient presents with flashes or floaters.

Mydriasis

Mydriasis is obtained by instilling an anti-muscarinic such as Tropicamide 0.5% or 1%. This will not only dilate the pupil but also abolish the light pupil reflex, essential when using bright sources of light to view the fundus. Enhance dilation is obtained by also instilling a sympathomimetic such as phenylephrine 2.5% which acts on the dilator muscle. Tropicamide is available to all optometrists as a POM and phenylephrine is available as a P Medicine (use 2.5%).

Precautions to be taken prior to dilation include a routine evaluation of the anterior chamber angle. Acute glaucoma is rare but one need to proceed with caution if the angle is so narrow as to predict an attack of acute glaucoma if the pupil is dilated. It is

advisable anyway to always to screen all adults for narrow angles using a simple method such as Van Herrick.

Sympathomimetics must be used with caution in any patient with a history of cardiovascular disease, with pregnant women, with patients with advanced diabetic disease and with patients taking medication such as MAOI. Ask women of childbearing age if they are pregnant and record their answer. It is advisable to check the systemic blood pressure of any patient over the age of 40 years before instilling phenylephrine.

Instrumentation

As previously discussed, most tears occur anterior to the equator (Jones, 1998). Direct ophthalmoscopy will only provide a view of the fundus up to the equator **with a dilated pupil** (Elliott, 1997). Thus, most retinal tears will not be visible with a direct ophthalmoscope even if the pupil is dilated.

The optimum method of examining the peripheral retina is with the head set BIO with scleral depressor. Head set BIO takes a lot of practise and according to the College of Optometrists *Clinical Practice Survey* (2001) only 15% of respondent optometrists used the Head set BIO in practice. Scleral depression or indentation further enhances the view of the peripheral retina and aids detection and analysis of retinal breaks. However, this takes further skill and it is likely that even fewer optometrists in the UK use this techniques.

Another useful method of viewing the peripheral retina is with a slit lamp microscope with a mirrored contact lens. Once more this is a technique that is probably currently not used by many optometrists in the UK. Indeed, the College of Optometrists survey did not even enquire after its use.

Whilst not as adequate as a head set BIO with scleral depression, the slit lamp biomicroscope in conjunction with BIO lens such as the Volk 90 D Superfield affords vastly better views of the peripheral retina than direct ophthalmoscopy. Further, according to the College of Optometrists *Clinical Practice Survey* (2001), 80% of respondent optometrists stated that they use slit lamp BIO in practice.

Anatomy of the vitreous and peripheral fundus

The retina composed of inner neural or sensory layers and outer pigment epithelium.

The normal retina is transparent except for pigment in the blood. The sensory retina thin and weak and is susceptible to full thickness breaks.

The retinal pigment epithelium (RPE) is a uni-layer of polygonal cells, uniform in size and pigment except at macular and vitreous base (vitreoretinal symphysis). The inner surface has microvilli giving loose attachment or bond to sensory retina.

The RPE is attached to the basement membrane which forms the innermost layer of basal lamina (Bruch's membrane). Bruch's membrane bonds to the choroid.

The equator of the fundus is situated approximately 14 mm from the limbus and is located ophthalmoscopically by finding vortex veins which drain blood from the region.

The ora serrata demarcates the anterior limit of neural retina and has a scalloped appearance. It is 2mm wide nasally and 1 mm temporally and is situated 7-8 mm from the limbus. Rounded extensions of the pars plana into ora are called ora bays.

The vitreous is 98% water, fills 2/3rds of the globe and provides structural and metabolic support for retina. Collagen supplies the structural frame and hyaluronan gives it its viscoelasticity. There is a depression posterior to lens called the patellar fossa and Cloquet's canal traverses anterior/posterior.

The vitreous base is a 3-4 mm zone straddling the pars plana.

There are vitreoretinal adhesions in the normal eye. The cortical vitreous is loosely attached to the inner limiting membrane of the sensory retina, strongly attached to the vitreous base, fairly strongly attached to the optic disc margin, weakly attached around the fovea, and weakly attached along peripheral blood vessels.

As the vitreous body slowly degrades with age it undergoes liquefaction (*synchisis*) and shrinkage (*syneresis*) and these can be readily seen in middle age and older. As this aging occurs what any intact vitreous structure increasingly makes rotational movements as the eyes are moved. These centrifugal and centripetal forces can then lead to a separation of the vitreous from the retina (a PVD) and, in the presence of excessive vitreous traction on the retina, may lead to a retinal tear.

Vitreous floaters

Jones (2007) describes a vitreous floater is any part of the vitreous that is dense enough to be detected by the patient (symptom) or to be seen during ophthalmoscopy by the optometrist (sign). Floaters commonly derive from material naturally found within the eye but can potentially come from external sources such as intravitreal foreign bodies. Vitreous floaters can be the result of natural age changes, from hereditary vitreoretinal degenerations or secondary to inflammatory disease or from unknown causes (e.g., asteroid hyalosis).

Vitreous floaters come in many different shapes and sizes from the most tiniest of dots to long filaments, amoeboid discs or rings. As the vitreous becomes more liquefied with

age, any floaters present can become more mobile. Typically, a patient with floaters in the presence of a fairly intact vitreous gel, will report that the floater will move with the eye. In the presence of a more liquefied vitreous or a PVD the motion of floaters will be more considerable. The colour will vary from grey to white, red in the case of erythrocytes or brown if melanin granules.

A common larger sized floater is caused by the ring of glial tissue that surrounds the optic nerve and which can come away as a PVD occurs. Such a floater can appear as a ring and is known as a Weiss's ring.

Floaters are usually produced by vitreous shrinkage (*syneresis*), fibrillar degeneration or posterior vitreous detachment (PVD). They are usually most noticeable and troublesome to the patient when they first develop but often become less annoying with time. For some patients floaters continue you to be psychologically debilitating to the extent that they may enquire about possible treatments to remove them. Photodestruction with neodymium-YAG laser has been advocated.

Anterior vitreous floaters, that is. behind the lens, may be vitreous fibrillar strands, erythrocytes, leucocytes, pigment cells and asteroid hyalosis. Vitreous strands become more apparent after a PVD because their greater movement causes them to be intertwined. Erythrocytes appear as tiny red dots and occur because of bleeding into the vitreous. Leucocytes appear white and occur with uveitis. Pigment cells appear as brown dots and result from the release of pigment from the RPE and, in this context, are known as *tobacco dust* or *Shafer's sign*. The presence of *Shafer's sign* indicates the possibility of a retinal break and demands a careful dilated fundus examination and advisedly, referral for an ophthalmological opinion. Other causes of pigment cells in the vitreous include malignant melanomas.

Posterior vitreous floaters may be the result of posterior vitreous condensation, fibrillar strands, erythrocytes, inflammation such as chorioretinitis, and PVD. As the vitreous degenerates it can liquefy (*synchysis*) and condense to form small semi-transparent floaters which are the most common reported by patients. Asteroid hyalosis occurs as anything from a few to a multitude of whitish bodies made of a complex combination of compounds including calcium salts. The cause of asteroid hyalosis is not known but may be more common amongst diabetics. It is usually unilateral. A similar, but less common condition is synchysis scintillans which is similar to asteroid hyalosis except that the bodies are cholesterol crystals which are freely moving because of the synchysis.

PVD

PVD is the condition in which the vitreous cortex separates from posterior retina and optic disc. If it extends to ora serrata it is termed a complete PVD and if only posterior region it is termed an incomplete or partial PVD.

Prevalence of PVD

Statistics for prevalence vary considerably and are discussed by Jones (1998; 2007). One examples of prevalence for adults of all ages is that 2% exhibit an incomplete PVD and 12% a complete PVD. The prevalence for over 65 years of age is 3% for incomplete and 31% complete. If there is PVD in one eye then PVD is likely to develop in the fellow eye within a few years.

The risk of PVD increases with high myopia probably because of the increase in axial length which adds to the stresses at the vitreoretinal interface. For myopes higher than 3 D, PVD occurs 10 years earlier than in emmetropes or hypermetropes.

.It is reportedly more common in females and it has been suggested that there may be a correlation with postmenopausal changes in the vitreous. It is also more prevalent amongst aphakes.

Trauma to the eye increases the incidence of PVD and certain sports such as boxing are risk factors. Interestingly, there have reportedly been two cases of PVD following non-contact tonometry.

Clinical appearance

Ophthalmoscopically it can be seen as very thin and transparent membrane and can sometimes be seen with the direct ophthalmoscope. With biomicroscope it appears as a more dense transparent membrane in central portion of vitreous cavity. The posterior face of the vitreous cortex contains particulates which scatter light and these move on eye movements.

The presence of an avulsed pre-papillary gliotic ring (Weiss) is pathognomonic of PVD. It may appear as a complete annulus or be broken. Because it is close to visual axis it is frequently seen by patient. Glial strands may be attached and seen by patients as spikes, threads or spider web. If the patient is asked to move his eye around then the glial tissue will move especially in the presence of synchisis. This is sometimes known as the ascension-descension phenomenon.

Other signs associated with PVD include "tobacco dust" (Shafer's sign) which consist of tiny brown dots which are pigment cells from the RPE. In the presence of tobacco dust one must assume retinal tear until proven otherwise.

Pathophysiology

PVD usually commences over posterior pole. Synchisis leads to formation of pockets of fluid called lacunae, syneresis occurs and the vitreous cortex may collapse inferiorly.

Symptoms of PVD

Floater

Floater are most common and are variously described as cobwebs; hairnet; strings and come in many shapes and sizes. As discussed earlier, floaters may be due to condensation of vitreous fibrils in cortex, glial tissue torn from epipapillary region or intravitreal blood from superficial vessels. They move about freely with the detached vitreous. One or two longstanding floaters indicate vitreous condensation or a PVD annulus. These are generally benign symptom with no significant associated risk.

However, the report of a sudden onset of floaters calls for a dilated fundus examination without undue delay. 95% of patients over 50 years with a sudden onset of floaters have a PVD. Even a single floater may indicate a PVD annulus with a small possibility of a retinal tear.

Photopsia

Photopsia is also a frequent symptom of PVD and occur because of mechanical stimulation of the retina by the traction produced by detaching vitreous cortex tugging at the retina. They are usually perceived by the patient as bright white flashes or sparks, usually occur with significant traction, for example on movement of the eyes, and usually occur during active process of vitreous detachment. Such flashes of light persist if areas of vitreoretinal traction remain.

An arc of light is usually associated with traction at the vitreous base whereas a flash-bulb type of photopsia repeatedly in the same position indicates localised traction.

Although photopsia is a symptom of vitreoretinal traction, patients with photopsia have no higher incidence of retinal tears than those without the symptom.

Very few eyes with asymptomatic PVD have gone on to produce a retinal tear.

Complications of acute PVD

Retinal haemorrhage

Tiny dot haemorrhages in the extreme retinal periphery can be associated with PVD and indicate an area of traction but not necessarily a retinal tear. Patients with such haemorrhages who do not have symptoms should be warned of the symptoms of PVD and monitored as if they are undergoing a PVD.

Retinal tear

Various published papers have suggested that between 8 – 46% of eyes with PVD develop retinal tears due to traction at sites of strong vitreoretinal adhesions. Although they usually develop at time of PVD but can occur weeks or months later.

Tears are usually, but not always, symptomatic (photopsia and/ **or floaters**).

There are various predisposing factors for PVD producing tears including the presence of lattice and snail track degenerations.

Premacular or epiretinal membranes

There is a strong correlation between PVD and the formation of epiretinal membranes which are best viewed with optical coherence tomography (OCT). Epiretinal membranes will only be treated surgically if causing a significant reduction in vision. Referral for epiretinal membrane is usually not urgent unless associated with a partial thickness macula hole.

Flap or Horseshoe Retinal Tear

A flap tear results from vitreous traction that is significant enough to tear a semi-circular flap of sensory retina. The edge of the flap almost always remains attached at the anterior margin of the break. This gives the flap a horseshoe shape appearance. Because the vitreous usually remains attached to the edge of tear it will, particularly on eye movements, continually cause traction at that point leading to a heightened risk of the tear increasing in size.

Whilst most flap tears are associated with symptoms, even asymptomatic flap tears have been reported to have a 25% -90% incidence of progressing to a detachment. Therefore, essentially all flap tears are treated.

Operculated tear

In this case traction causes the removal of a plug of sensory retina (operculum). The commonest cause of an operculated tear is PVD. The operculum shrinks with time to 20% of its original size and therefore, the size of this plug of retina compared to its associated hole will give an indication of how recent the lesion occurred.

There is a 10 -20% risk of retinal detachment with an operculated tear. Scleral depression enhances the view. Generally, operculated tears are not treated unless there is significant RD.

Sometimes there may be white collar around the hole that represents a very localized detachment (less than 1 DD from the edge of the break).

These tears are most often found between ora serrata and equator where the retina is thinner than in posterior region.

Retinal detachment (RD)

There are three broad groups of RD.

Rhegmatogenous detachment. This name is derived from *rhegma* meaning 'break' is most commonly associated with PVD or trauma.

Tractional retinal detachment is associated with conditions such as proliferative diabetic retinopathy, Eales disease and central retinal vein (CRV) occlusion.

Exudative retinal detachment is associated with, for example, choroidal tumours.

A retinal detachment (RD) is the separation of the sensory retina from the RPE (outer segments of the photoreceptors from the microvilli of the RPE).

The appearance of a fresh RD is a white membrane, with tiny folds and blood vessels, in the vitreous cavity that moves (undulates) on eye movements. Because the RD is separated from the RPE by a fluid reservoir, the underlying choroidal detail will be obscured (as it also is for a retinoschisis).

The detached retina will be mobile and to differentiate it from retinoschisis, the patient should be asked to move his eye and then re-fixate. The appearance of the folds in an RD will be different on each occasion. In contrast to this, a retinoschisis will appear unchanged.

Retinoschisis

Splitting within the sensory retina. Very common. Usually semi-circular with convex curve towards posterior pole – usually bullous and transparent. Looks like a “blister”. No movements on eye movement. Absolute scotoma (compare with relative scotoma with retinal detachment. Generally asymptomatic.

Retinal tufts

Also known as granular tissue. Grey/white lesions in the peripheral retina. Small, static and do not increase in size with age. Usually asymptomatic but can cause photopsia due to significant vitreoretinal traction Can cause a retinal break (e.g., operculated hole)

and 10% of rhegmatogenous retinal detachment caused by tuft. however, very few retinal tufts lead to problems. Check patient every 12 months.

Lattice degeneration

A vitreo-retinal degeneration found in about 7% of the population. Develops at an early age and incidence does not increase with age. Usually seen as small, elongated patches of inner retinal thinning running parallel to but with a distinct separation from the ora serrata. Degenerative retina is often criss-crossed by white lines (ghost-vessels).

Slowly progressive leading to thinning of the retina, cysts and breaks. 25% lead to retinal breaks. About 30% of retinal detachments are associated with lattice degenerative breaks.

Cobblestone degeneration

Present in approx 25% of normal eyes (Kanski, 1989; Jones, 2007). Majority just posterior to ora serrata. No treatment necessary

Pigmented lesions

CHRPE 1% of population. Caution if multiple CHRPE like lesions = ? POFL

Choroidal Naevi 10% of population. Monitor annually

Malignant melanoma Rare but the most common intraocular tumour. 21/million per year. Various shapes but will usually be elevated. Look out for yellow mottling – lipofuscin.

Other choroidal disturbances

e.g, toxoplasmosis, toxocara, histoplasmosis. Inflammatory activity gives white cells in vitreous. Pigment denotes age.

Strategies for the optometric management of ‘at risk’ patients

The College of Optometrists offers guidance for the optometrist with regard to patients reporting flashes of light or floaters.

It is always necessary for the optometrist to bear in mind that, when examining a patient who complains of flashes and/or floaters, the optometrist should conduct an examination appropriate to the patient's needs. In the presence of relatively new or changed symptoms or in the presence of risk factors such as myopia or recent trauma, this invariably means carrying out an indirect ophthalmoscopy under mydriasis.

The optometrist must then first make a decision as to whether to examine patients who make an appointment because of flashes or floaters. If the optometrist feels unable or is unwilling to do this they must refer the patient to someone who is able to perform an adequate examination. This could be another optometrist or an ophthalmologist.

The College of Optometrists guidelines state that once you have decided to examine the patient you are committed to continuing until you detect a problem and can make a diagnosis or you accrue sufficient evidence to make a considered decision on what action to take next,

If there is a local protocol in place for seeing these patients, this should be followed.

Telephone conversations

Patients often telephone the practice and report symptoms. They will not always get to speak to the optometrist, so it is crucial that the support staff is instructed on how to deal with such a patient. This may be determined by local protocols or by the optometrist training his/her staff as to how to advise such patients.

Patients should be told that a diagnosis cannot be made over the phone and that he/she should be seen as soon as possible, preferably that same day. Any advice must be recorded and, for example, even someone not known to the practice who is advised to go to casualty, should have their details taken and notes recorded about the telephone conversation and advice given. Receptionists should be trained to make notes in the record recording what the patient complained of e.g., *“patient has flashing lights; advised to see optometrist today; appointment booked”*.

Examination routine

If a patient presents with new symptoms indicating a possible PVD, retinal break, retinal tear or retinal detachment, the examination should include:

- 1) History and symptoms including onset and to enquire of possible risk factors, e.g., recent trauma.
- 2) Unaided vision/visual acuities (pinhole if necessary).
- 3) Slit lamp examination of the anterior vitreous to look for pigment cells.
- 4) Examination of the retinas by indirect fundoscopy under mydriasis. It is advisable to examine both eyes even if the patient only reports symptoms in one eye.
- 5) Giving appropriate advice to the patient (ideally supplemented by a written information sheet).
- 6) A visual field examination may also be useful to detect shallow detachments that may otherwise be difficult to detect with ophthalmoscopy. It should be noted that a retinal tear in the periphery will not show up in a central visual field assessment such as a Humphrey C40 or a SITA test but, in the absence of visible signs on

the retina or in the vitreous, a normal visual field can provide supplementary evidence suggesting normality.

Patient management

Emergency referral

For any patient reporting flashes and/or floaters:

- 1) If pigment cells (Shaffer's sign; 'tobacco dust') are observed in the anterior vitreous then it should be assumed that a retinal tear is present unless proved otherwise by a retinal specialist. An emergency referral is indicated.
- 2) If a flap or horseshoe tear is detected, whether or not accompanied by symptoms, an emergency referral to a casualty eye department or to a retinal ophthalmologist should be made immediately. There is a very high risk of a flap tear developing into a retinal detachment.
- 3) There may be more discretion available regarding an operculated tear that is old, especially if previously investigated, and if the floaters are longstanding. However, a new operculated hole accompanied by symptoms carries a risk of retinal detachment and should also be treated as an emergency with a same day visit to an ophthalmologist.
- 4) A retinal detachment demands an immediate emergency referral
- 5) It is advisable to refer for an opinion a patient complaining of flashes and/or floaters in the presence of lattice degeneration even in the absence of an observable tear.

In all cases of same day emergency referral, the patient should be given a letter to take to the ophthalmologist outlining the pertinent findings including symptoms, history, VAs and examination findings. It may be helpful to the ophthalmologist to include a note of the name, concentration and time of instillation of the mydriatic drop to avoid any confusion with regard to the cause of the fixed dilated pupil.

Uncomplicated PVD

With regard to a patient with an uncomplicated PVD, whether partial or apparently complete, management protocols vary between optometrists depending on training and experience.

Having examined a patient under mydriasis and found no evidence of complications it is perfectly acceptable to refer all new PVDs requesting an ophthalmologist's opinion *without undue delay*.

Alternatively it is also acceptable for some optometrists to manage the patient in-practice whilst following a management protocol.

An example of such a protocol might be thus:

- 1) The patient should be examined again under mydriasis at a follow-up appointment looking again for vitreous and retinal signs of disease
 - a. If the symptoms are of recent onset, a 6-week follow-up is indicated
 - b. If the symptoms have been present for 3-months or more, then review in six months and
 - c. If the symptoms have been present for 12-months or more, then review in 12 to 24 months.
- 2) At the initial consultation the optometrist should carefully explain to the patient the importance of looking out for any change or worsening of the symptoms such as an increase in floaters, more flashes or a veil or curtain coming across the vision.
- 3) The patient should be given an information sheet reinforcing what was explained verbally. You may also consider providing the patient with your out of hour's mobile number for emergency use only.
- 4) Care and caution should be taken with any patient who reports symptoms since sustaining trauma to the eye. A retinal dialysis may take many months or even a year or two before progressing to a full blown retinal detachment

Records

Optometrists are reminded to keep full and accurate records of all patient encounters. As discussed above, this includes when the patient is spoken to on the telephone (by the optometrist or another member of staff) as well as when they are in the consulting room. All advice that is given to the patient should be carefully noted, together with any information that was given to the patient in writing such as a referral letter or information leaflet(s). Negative as well as positive findings should be noted (e.g. 'no retinal tears or breaks seen').

Medico-legal defence

If a patient detaches sometime after having had a thorough dilated examination with an indirect method, the defence would be that the condition was not ordinarily apparent at the time of examination. The complainant would have to prove that the condition was definitely present and that the optometrist was incompetent at undertaking the indirect technique used.

If the patient detaches at any time after having had an examination with a direct instrument whether dilated or not, the defence would be more difficult as it could be argued that the examination technique was not adequate.

Example reports and referral letter

Part 1

Mrs A
W Road
London N

3rd November 2010

Dear Mrs A

It was a pleasure to see you today for your routine eye examination. This is a routine report for your records. I note your history of laser refractive surgery in London in 2005, that you take thyroxin, Prempak and various vitamins, and that there is no family history of ocular disease.

Your unaided vision is R. 6/6+ L. 6/6. Refraction gave R. -0.25 /-0.25 X90 =6/5+ L. +0.00DS = 6/6 Add +1.00DS for reading = N4 at 40 cm. Zeiss i-Profiler Aberrometry shows significant high order aberrations (including coma) in both eyes. Central corneal thicknesses (CCT) are 480 μ in each eye.

External eyes, pupillary reflexes, ocular motility and ocular motor balance are normal. Anterior chambers are wide and intraocular pressures are R. 9 L. 10 mmHg by Pascal DCT at 09.00. Visual fields were full to C40 Humphrey screening.

There is grade 1 corneal epithelial down-growth in each eye. Both eyes show partial uncomplicated posterior vitreous detachment (PVD). The ocular media are otherwise clear. The ocular fundi are normal apart from some minor peripheral sensory retinal changes in the extreme periphery of each eye.

We discussed that due to the PVD and your previous moderate myopia, if you ever notice flashes of light or "floaters" (spots) in your vision, you are to seek attention straight away.

I would suggest a routine examination in twelve months.
I have attached copies of your various scans for your records.

Regards

Dr Simon Barnard PhD FCOptom FAAO DipCL DipClinOptom

Part 2

Mr Riaz Asaria
Consultant Vitreo-Retinal Surgeon
Wellington Hospital
NW8

23rd August 2011

Dear Riaz

Re: Mrs A DOB 1959

Thank you for seeing Mrs A tomorrow (Friday) at 15.30 at the Wellington (Platinum).

She had telephoned me this morning reporting seeing a black spot floating in her vision and I asked her to come in to see me today and I saw her at 15.00 hours.

Unaided vision is R & L 6/6.

There is a small flap tear superior nasal (2 o'clock) flap tear right eye. There are peripheral retinal changes (white without pressure; PSRD) elsewhere in both eyes but I could not see any other tears.

By way of a history, I have attached a copy of a report I wrote in 2010.

With kindest regards

Simon Barnard

This patient provided her own description . Go to

www.barnardlevit.co.uk/about-us/testimonials/

Acknowledgement

I am particularly indebted to Bill Jones from whose books referenced below are a great source of information

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