

Retinal Break and Rhegmatogenous Retinal Detachment: The Current Management

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Almost 90 years ago, in 1919, the first successful procedure for a previously untreatable retinal detachment that could cause blindness was reported by Jules Gonin in Swiss Ophthalmologic Society. Until 10 years later, in 1930, his first publication in English reached the world in the Archives of Ophthalmology.¹ Gonin's principles of retinal detachment surgery based upon 3 steps: find the retinal break, treat the retinal break, and close the retinal break. Even though many changes have occurred in the management of retinal detachment since then, the 3 essential steps for successful surgery still remain.

Currently, there are many ways to manage retinal break and detachment, the decision for optimal treatment depends upon a lot of factors including disease factors (the type of retinal break and detachment), surgeon factors (training, bias, age), equipment and operating room availability, cost considerations, and patient factors (age, compliance, underlying disease, patient's decision). In this article we aim to discuss and describe causes and the treatment options of retinal break and detachment.

A rhegmatogenous retinal detachment (RRD) (Greek rhegma= rent, tear) is a separation of the sensory retina from the retinal pigment epithelium caused by liquefied

vitreous entering the retinal break into the subretinal space. (Fig 1)

The vitreous is a transparent gel-like structure, occupying the posterior segment of the eye. In a normal young eye, the vitreous is attached to the whole surface of the retina, the head of the optic nerve, ora serrata, pars plana, the ciliary body and posterior lens surface. Vitreous gel-like structure changes to a more liquid state (liquefaction) with aging and in certain ocular conditions (such as high myopia, intra-ocular inflammation, ocular trauma or ocular surgery). After the increase of vitreous liquefaction, collagen fibrils will eventually collapse together and form bundles of collagen fibrils condensations that became more opacity and visible.

The shadows of the opacities in the vitreous cavity that cast onto the retina were described by patients as "floaters" (varies in shapes and sizes, such as dots, lines, strings, cobwebs, veils, etc.; move with the eye's movement) and were usually observed easier in the area of bright illumination such as when looking at the sky or plain light color background. Floaters may be caused by other vitreous opacities such as blood, epipapillary glial tissue torn from the optic nerve head or the area adjacent to the optic nerve head.

"Flashing" is an important visual sensation symptom of bright light flashes caused by stimulation of the retina



Fig 1. A rhegmatogenous retinal detachment (RRD) is a separation of the sensory retina from the retinal pigment epithelium caused by liquefied vitreous entering the retinal break into the subretinal space



Fig 2. Prophylactic laser treatment of breaks to reduce the risk but not eliminate the risk of new tears or RRD.

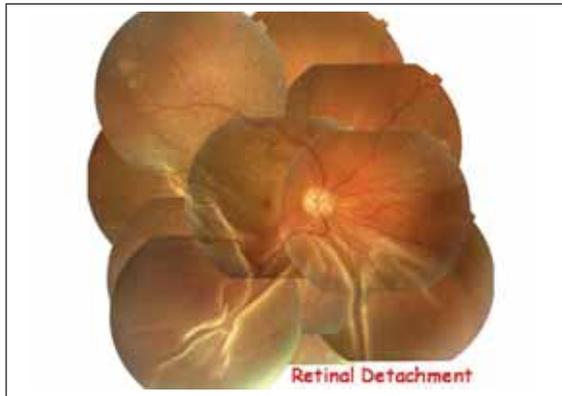


Fig 3. Retinal detachment characteristics : opalescent detached retina, corrugated retinal surface, and decrease the view of the underlying choroidal vessel.

due to its mechanical traction on vitreoretinal adhesion site from the collapse of vitreous gel. The vitreous is firmly attached to the retina around the disc, at the macula to superficial major retinal vessel, to the vitreous base at ora serrata and pars plana, and to vitreoretinal degenerative lesions such as the edge of lattice degeneration, chorioretinal scar.

In most ordinary cases after increasing vitreous collapse, the posterior vitreous detachment (PVD), a separation of the posterior vitreous cortex from the internal limiting lamina of the retina will occur without causing retinal damage. A symptom of sudden increase floaters may be caused by PVD only or with rupture of retinal vessels with hemorrhage into the vitreous cavity. But in some cases, vitreous traction at the sites of significant vitreoretinal adhesion is responsible for retinal breaks that lead to RRD. Prophylactic treatment of breaks may be considered to reduce the risk of RRD but it does not eliminate the risk of new tears or detachment. (Fig 2)

“Progressive visual field loss” is another important visual symptom that is caused by RRD due to liquefied vitreous enters through a retinal break into a subretinal potential space and separate photoreceptor cells from the retinal pigment epithelium. The detached retina becomes opalescent because of intraretinal edema, detached retinal surface may appear corrugated, and the view of the underlying choroidal vessel decreases. (Fig 3) Visual field loss usually progresses from periphery to central because a break usually occurs in the peripheral retina at the vitreous base. Almost all patients with a symptomatic RRD will progressively lose vision unless the detachment is repaired. Spontaneous reattachment is extremely rare. Currently, more than 95% of RRDs can be successfully repaired, although more than one procedure may be required.

Prevention or early diagnosis is very important in RRD management because the rate of successful reattachment is higher and the visual results are better if RRD does not involve the macula. By knowing the natural history of precursors to RRD (that includes PVD, retinal breaks, and lattice degeneration), epidemiology, risk factors of RRD will help us find the optimal managements to prevent RRD and to detect early RRD in the patients.

Precursors to RRD²⁻⁴

Posterior Vitreous Detachment

The symptoms of PVD include flashing of light and floaters, and patients with these symptoms are at significant risk of retinal detachment.

Approximately, 15% of patients with acute symptoms of PVD have a retinal tear at the time of initial examination and 50-70% of PVD with vitreous hemorrhage have retinal tears. However, patients with acute PVD who have no retinal breaks on presentation have a 2% to 5% chance of developing them in the weeks that follow. In patients who present with substantial vitreous hemorrhage, 67% were found to have at least one break, with 31% having more than one break and 88% of the breaks occurred in the superior quadrants. Currently, there are no effective methods of preventing vitreous changes that lead to RRD.

Retinal Breaks

Not all retinal breaks progress to RRD; however, retinal break should be treated if any significant risk of the progression to RRD. Retinal breaks may be classified as

Flap, horseshoe tear is a strip of retina that was pulled anteriorly by vitreoretinal traction, often due to a posterior vitreous detachment or trauma; it has increased risk of RRD.

Giant retinal tear is a tear that extends 90° or more circumferentially.

Operculated hole is a tear with a piece of retina completely free from the adjacent retinal surface, occurs when traction is sufficient.

Dialyses are circumferential, linear breaks that occur along the ora serrata, commonly as a consequence of blunt trauma.

Atrophic hole is generally not associated with vitreoretinal traction and has not been associated with an increased risk of RRD.

Macular hole is a defect in the vitreoretinal interface to full-thickness defect in the retina at the macular area.

Symptomatic Retinal Breaks

A symptomatic retinal break is defined as a break caused by vitreoretinal traction in a patient with a new PVD or a break associated with a significant increase in flashes and floaters.

Half of untreated symptomatic retinal breaks with persistent vitreoretinal traction (horseshoe or flap tears) will cause a clinical RRD unless treatment is applied. Prompt treatment by creation of a chorioretinal adhesion around these symptomatic tears decreases the chances of RRD to less than 5%.

Asymptomatic Retinal Breaks

Asymptomatic operculated holes and atrophic round holes very rarely lead to RRD. Atrophic retinal breaks unrelated to vitreoretinal traction may be preexisting in eyes with acute symptomatic PVD and no evidence of a benefit of prophylactic therapy has been proven.

Lattice Degeneration

Lattice degeneration is a peripheral retinal degeneration that present in 6% to 10% of the general population. It occurs more commonly in myopic eyes and is bilateral in 30% to 50%. Because of atrophy of the inner layers of retina, overlying pocket of liquefied vitreous with condensation and firm adherence of the vitreous at the margin of lattice. It may predispose retinal breaks and detachment. It is found in 20% to 30% of all eyes with RRDs.

Atrophic round holes within lattice lesions usually do not require prophylaxis treatment. Younger myopic patients who have lattice degeneration with holes need regular follow-up visits because a small localized RRDs may occasionally develop and slowly enlarge to become clinical retinal detachments. Treatment should be considered if the detachments are documented to increase significantly in size.

Retinal detachment does not occur in eyes that have lattice degeneration without holes unless PVD causes a horseshoe tear. When PVD occurs in an eye that has lattice degeneration with holes, new tears may develop but RRD rarely occurs from the old ones.

A study with 11-year follow-up showed that RRD occurred in approximately 1% of untreated lattice degeneration patients with no symptomatic tears so lattice with or without atrophic holes generally does not require prophylaxis in the absence of other risk factors. Prophylactic treatment may be suggested in cases of lattice with these risk factors: presence of flap tears, high myopia, presence of RRD in the fellow eye, and aphakia.

A retrospective study found that the risk of RRD in the fellow eye of patients with phakic lattice RRDs was reduced over 7 years of follow-up from 5.1% in untreated eyes to 1.8% in those with full treatment of their lattice. However, the incidence of RRD was low in both groups. Therefore, prophylaxis is not universally recommended.

Epidemiology of RRD²⁻⁴

The annual incidence of RRD is 10 to 15 per 100,000 persons. Multiple studies have shown that males are more frequently affected than females by a ratio of 1.3 - 1.5 to 1. Well-established risk factors include older age, myopia, cataract surgery, and trauma. Racial differences have also been described. Asian population was found to have an increased incidence of RRD, due to a higher prevalence of myopia.

Risk factor for RRD²⁻⁴

Risk factors for RRD include retinal breaks, lattice degeneration, myopia, cataract surgery, trauma, and a history of RRD in the other eye. Combinations of these factors in a single eye increase the risk of RRD.

Myopia

More than half of nontraumatic RRDs occur in myopic eyes. The risk of RRD increases with the increase of axial length. Low myopes (1 to 3 diopters) have a 4-fold risk and higher myopes (>3 diopters) have a 10-fold risk compared with that of emmetropes.

Cataract Surgery

The overall risk of RRD after cataract surgery is approximately 1%. Vitreous loss, increased axial length, lattice degeneration, Nd:YAG laser capsulotomy, Caucasian race, and younger age have been reported to increase the risk of RRD after cataract surgery. Two large studies found that the risk of RRD after cataract surgery is 6 to 7 times greater than that of phakic control groups.

Trauma

Blunt or penetrating ocular injuries cause changes in the structure of the vitreous or retina and increase risk of RRD. The risk of RRD by blunt trauma in younger age group is decreasing because of a formed (not liquefied) vitreous. The detachment may not be symptomatic for years even though nearly all breaks caused by a blunt trauma may occur at the time of the injury.

Rhegmatogenous Retinal Detachment in the Fellow Eye

Because pathologic vitreoretinal changes are frequently bilateral, approximately 10% of phakic patients who have RRD in one eye and 20%-36% of aphakic patients with RRD in one eye develop RRD in their second eye. A pseudophakic RRD is not necessarily caused by cataract surgery alone. The fellow eye in a patient with pseudophakic RRD is also at higher risk for developing a RRD, whether the fellow eye is phakic or pseudophakic. Phakic fellow eyes in patients with pseudophakic RRD have about 7%

risk of RRD. This indicates that all risk of RRD cannot be attributed to cataract surgery alone.

Other Risk Factors

Other risk factors that have been reported include prior retinopathy of prematurity and Stickler syndrome.

PREVENTION AND EARLY DETECTION²⁻⁴

Patients at high risk should be educated about the symptoms of PVD and RRD as well as the value of periodic follow-up examinations.

Peripheral fundus examination in patients with factors that increased the risk of RRD should be done with indirect ophthalmoscopy combined with scleral depression or with slit-lamp biomicroscopy combined with a 3-mirror or wide angle contact lens.

B-scan ultrasonography should be performed to search for retinal tears or detachment and other causes of vitreous hemorrhage, if vitreous hemorrhage is dense and obscure the posterior pole. If no abnormalities are found, frequent follow-up examinations are recommended. There is insufficient evidence to guide management recommendations for patients with dense vitreous hemorrhage but the treatment options includes observation with or without bed rest, bilateral patching or early vitrectomy.

TREATMENT FOR RETINAL BREAK²⁻⁴

The goal of prophylactic treatment for lesions (break, lattice) at risk of developing RRD is to create a firm chorioretinal adhesion with laser photocoagulation or cryotherapy in the attached retina around the retinal lesion or the focal subretinal fluid associated with the lesion.

Cryotherapy is a transcleral procedure using cryoprobe which creates cold energy to freeze the surrounding retina around the break. The inflammation in the frozen areas will result in chorioretinal scars around the break. This procedure may be uncomfortable due to indentation of the eye wall by the cryoprobe and may cause conjunctival swollen. Cryotherapy may increase the risk of proliferative vitreoretinopathy due to RPE cell liberation into vitreous cavity. Laser treatment is performed with laser machine at slit lamp (Fig 2) or with indirect ophthalmoscope laser to create chorioretinal scar around the break. Patients may feel uncomfortable from a bright flash of light and pinprick sensations as the laser is applied. Complications of laser include accidental foveal burns, Bruch's membrane rupture, hemorrhage, fibrous proliferation. In case of peripheral horseshoe flap tears treatment should be extended into the vitreous base and if possible to ora serrata because continued vitreous base traction can extend the flap tear out of the treated area and cause RRD from fluid leak through tears into the subretinal space. Evidence-based recommendations for management of these conditions exist only for acute, symptomatic horseshoe tears. For other vitreoretinal abnormalities, the relative risks, benefits and alternatives to surgery should be discussed with the patient.

The treatment can be performed using a variety of anesthesia techniques that include local (regional) anesthesia (e.g., retrobulbar, peribulbar, periocular, sub-Tenon's injection, or topical) in the majority of the cases and also general anesthesia. Complications of periocular injection of anesthesia include hemorrhage and globe perforation while retrobulbar anesthesia has additional complications including strabismus, retrobulbar hemorrhage, and macular infarction.

After treatments, epiretinal membrane has been observed with extensive treatment but its association is uncertain. RRD may occur after appropriate therapy. In large breaks

or breaks with bridging retinal blood vessels, the persistent traction may pull the tear-off the treated area because the treatment adhesion is not complete for up to 1 month. During long-term follow-ups, 10% to 16% of the patients develop additional breaks. Pseudophakic patients are more likely to develop new breaks which require retreatments.

The chorioretinal scar from treatment should be evaluated after treatment by indirect ophthalmoscope and scleral depression or contact lens at 2 or more weeks. Additional treatment should be considered if break or subretinal fluid is not completely surrounded by scars. Patients should be advised to contact their ophthalmologist promptly if they have a significant change in their symptoms and also should have routine follow-up in the absence of additional symptoms. Patients with any degree of vitreous hemorrhage or visible vitreoretinal traction should be asked to return for a second examination within 6 weeks following the onset of the symptoms. If vitreous hemorrhages obscure the fundus, B-scan ultrasonography should be followed periodically and in the eyes with suspected retinal tear, a repeat ultrasonographic study should be performed within approximately 4 weeks of the initial evaluation.

Rhegmatogenous Retinal Detachment: The Current Management¹⁻⁵

The treatment of primary RRD has been controversy over the last 10 to 15 years, as the availability of many techniques increases. The procedures that are used to surgically treat RRDs include the following: Pneumatic retinopexy (PR), Scleral buckling (SB) procedure, Pars plana vitrectomy (PPV). The overall rate of anatomical reattachment with these current techniques is more than 90%. Postoperative visual acuity depends on the status of the macula, whether it was detached as well as the duration of detachment. Almost 90% of RRDs sparing the macula recover the visual acuity of 20/50 or better compared with 30-50% of RRDs with detached macula. Among macular detachment patients, 75% will obtain a final visual acuity of 20/70 or better if the macular detachment duration is less than 1 week, as opposed to 50% with 1-8 weeks' duration. Even though RRDs with macula attached preoperatively were successfully repaired, visual acuity may not return to preoperative level in 10-15% of patients due to macular edema, epiretinal membrane. The most common cause of failure to repair RRD is proliferative vitreoretinopathy (PVR), occurring in approximately 7% of eyes after retinal reattachment surgery. PVR is caused by retinal pigment epithelial, glia, and other cells grow on both inner and outer retina surfaces and on the vitreous face forming membranes that may causes retinal contraction, retinal fold, detachment of pars plana, tractional detachment, new breaks, and reopening of causative breaks.

Numerous studies have compared therapies for RRD under various indications, to date there have been few large, randomized, controlled trials that have demonstrated the superiority of a single technique. Because the results have been unable to consistently demonstrate the superiority of a single method, the careful selection of the right management strategy is necessary due to different in risk and benefit of each management. Gonin's principles of retinal detachment surgery, which based upon 3 steps: find the retinal break, treat the retinal break and close the retinal break, are still important.

Finding the retinal breaks

More than 90% of RRDs, the causative break can be found by indirect ophthalmoscope with scleral indentation or slit lamp with 3-mirror or wide-field viewing contact lens. Finding all retinal breaks primary to the repair of

RRDs is very important for pneumatic retinopexy (PR) and scleral buckling (SB) procedures but these need may change. Now many retinal detachment surgeons do not feel that all retinal breaks need to be identified before surgery because they only need a binocular stereoscopic operating microscope to identify all retinal breaks in the operating room when they use vitrectomy techniques for the repair of RRDs.

Treating and closing the retinal breaks

Pneumatic retinopexy (PR) (Fig 4) uses a gas bubble injected into the vitreous to seal the break. This procedure requires the patient to stay in a special head position or posture to adjust the bubble position on the break. After the break is sealed, the subretinal fluid will be absorbed by retinal pigment epithelium cells pumping. Once the retinal around the retinal break is reattached then the chorioretinal adhesion can be made around the break by laser or cryotherapy. The gas bubble will be reabsorbed within a period of time depending on the type of gas. This is an out-patient procedure and can be done under local anesthesia.

PR has traditionally been indicated for uncomplicated cases of RRD, including superior retinal breaks smaller than 1 clock hour or multiple breaks not extended for more than 1 clock hour. Its contraindications include proliferative vitreoretinopathy (PVR), media opacities, uncontrolled glaucoma and aphakic status. Advantages of PR are, minimally invasion, reduced postoperative morbidity and recovery time. The serious complication of PR reflects the nature of the procedure. Because PR does not relieve vitreoretinal traction, as SB or PPV do, new breaks may form, and previously unidentified breaks may be recognized, or the original break may reopen. Thus, careful selection of candidates for the procedure is warranted.

Scleral buckling (SB) procedure (Fig 5) is a suturing of the scleral buckle material to the eyewall to create an indentation of eye from outside so that the eyewall (sclera, choroids and retinal pigment epithelium) is indent closer to the retinal break. The indentation relieves existing gel traction on the retina and closes the retina break. The chorioretinal adhesion can be made around the break by cryotherapy or laser. Subretinal fluid will be absorbed by

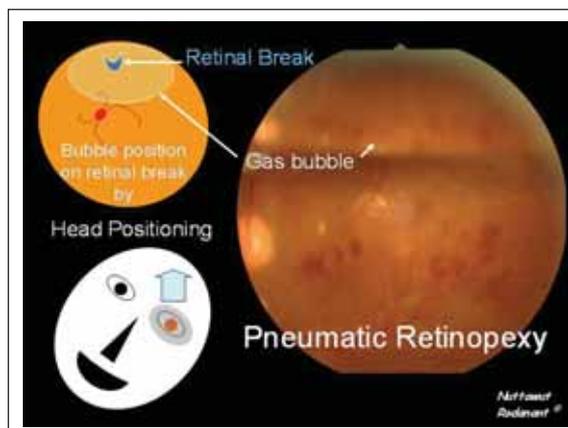


Fig 4. Pneumatic retinopexy (PR) is the use of a gas bubble injected into the vitreous to seal the break. This procedure requires the patient to stay in a special head position or posture to adjust the bubble position on the break. After the break is sealed, the subretinal fluid will be absorbed by retinal pigment epithelium cells pumping. Once the retinal around the retinal break is reattach then the chorioretinal adhesion can be made around the break by laser or cryotherapy.

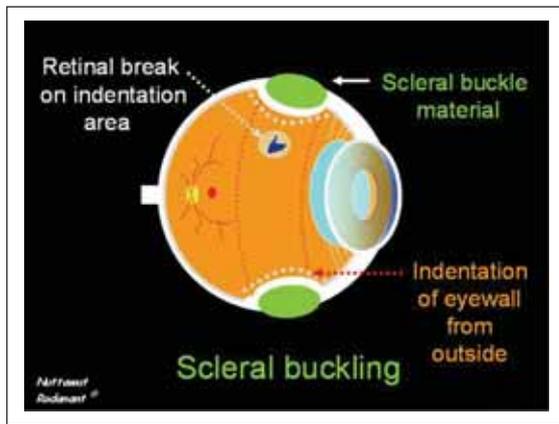


Fig 5. Scleral buckling (SB) procedure is the suturing of the scleral buckle material to the eyeball to create an indentation of eye from outside so that the eyeball (sclera, choroids and retinal pigment epithelium) is indent closer to the retinal break.

retinal pigment epithelium cells pumping. External subretinal fluid drainage via sclerotomy may be considered if the subretinal fluid is enormous. SB procedure can be done under local anesthesia and also can be an out-patient procedure.

SB surgery has been considered the standard of care for uncomplicated cases of RRD. Final anatomic success rates are greater than 94% after SB surgery have consistently been reported. The disadvantages of SB surgery include intraoperative hemorrhage or retinal incarceration during drainage, change in refraction with increased myopia, postoperative floaters, potential buckle extrusion with resultant infection, and prolonged recovery time with pain and ocular motility disturbances during the postoperative period. Failure of scleral buckling may associate with the presence of multiple retinal breaks, breaks larger than 3 quadrants or 3 disc diameters, detachments lasting longer than 1 week, poor visual outcome and macular detachment.

Pars plana vitrectomy (PPV) is an intraocular procedure that attempts to cut the vitreous by using vitreous cutter and other intraocular microsurgical instruments. In RRD, PPV is performed in order to remove vitreous gel and traction at the retina and break then replace vitreous with vitreous substitutes (e.g., gas, silicone) to close the break and tamponade the retina. After retina break is attached, chorioretinal adhesion is created around the break by laser or cryotherapy intraoperatively. Postoperative positioning is needed for a period of time depending on the type of RRDs and vitreous substitute. PPV can be done under local anesthesia and also can be an out-patient procedure. In the past, pars plana vitrectomy (PPV) was used as a primary surgical intervention in complicated RRDs such as RRD with proliferative vitreoretinopathy (PVR), vitreous hemorrhage, giant break, or breaks at the posterior pole. Later, PPV has become increasingly used as primary option in RRD repair because of the advantages in visualization all retinal breaks, removing opacities, removing retinal tractions, and it also has favorable rates of reattachment with low intraoperative complication rates. The disadvantages of PPV include the requirement for postoperative positioning, avoidance of flying or driving to high altitudes with the gas bubble in the eye, and the prolonged recovery time after placement of long acting gases. Other complications after PPV include postoperative cataract progression, lens trauma, epimacular membrane and endophthalmitis.

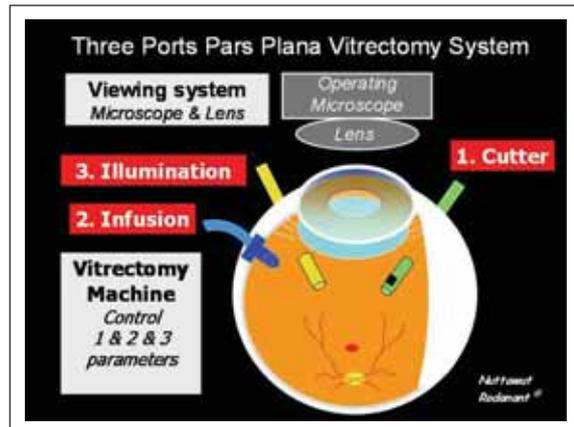
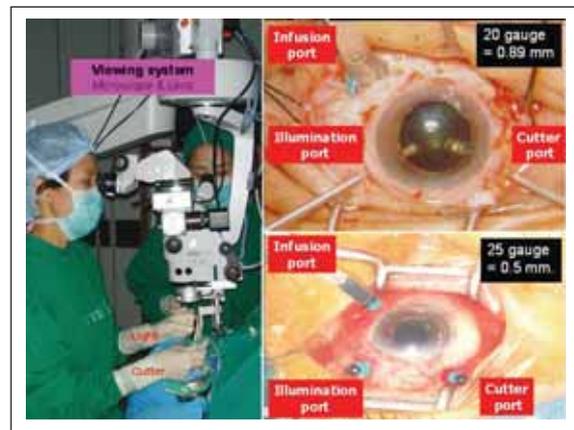


Fig 6. The standard three ports Pars plana vitrectomy (PPV) is depend on four essential systems: The vitreous cutter and other intraocular instruments, the infusion system, the illumination system, and the viewing system

The standard three ports PPV depends on four essential systems: vitreous cutter and other intraocular instruments, infusion system, illumination system, and viewing system. (Fig 6) A recent advancement in vitreous surgery is that it is a minimal invasive sutureless transconjunctival vitrectomy system with a high speed cutter. This 25 and 23 gauge vitrectomy instruments do not require suture closer as the standard 19 and 20 gauge instruments.

A vitreous cutting device aspirates small amount of the vitreous gel into an opening near an end of the device, and cut it off into small pieces by a rotating or guillotine blade at high speed (up to 2500 cut per minute) to reduce the traction force when the vitreous is removed from the retina. The cutter speed and the aspirate pressure are controlled by vitrectomy machine. The intraocular micro instruments include scissor (manual or automated), forceps, pick, fragmatome (for lensectomy), diathermy probe, laser probe, aspiration tips, magnet which also includes multifunction instruments such as combined light with forceps or laser, combined laser with extrusion, etc.

The infusion cannula is used to infuse a physiologically fluid (e.g., balanced salt solution, silicone oil, heavy silicone oil and perfluorocarbon liquid), air or gas (e.g., SF₆, C₃F₈) to replace the vitreous that was removed by vitreous cutter and maintaing the eye in its normal contour. Varieties of the vitreoretinal conditions need different type of vitreous substitutes for retinal tamponade. The infusion port was sutured to the sclera. The intraocular pressure is regulated by a pump in the vitrectomy machine or by height of solution container.

The illumination system is fiber optic instrument that deliver the light from the light source in vitrectomy machine or separated light source, and emits light from the end of fiber optic to create visualization of vitreous and retina during vitrectomy via an operating microscope and lens system. Later, the illumination system combined with infusion port and also a self retained illumination port as the 4th port were developed so that the illumination probe didn't need to be held by one hand as in standard 3 ports vitrectomy system. This allowed surgeon to perform bimanual vitrectomy.

Using operating microscope in conjunction with lens viewing systems, the vitrectomy can be performed. Many types of lens systems are developed for viewing of retina and vitreous in direct or indirect (wide-angle) view by contact or non contact system. The direct viewing systems allow greater magnification and enhanced stereopsis but allow smaller field of view compared with the indirect viewing system. The advantages of indirect visualization include a wider field of view, better visualization through miotic pupils, gas-filled eyes and media opacities.

Vitrectomy can be performed in conjunction with ophthalmic endoscopy and has value in two broad areas, first in visualization of internal view of the eye despite anterior segment opacities (cornea, anterior chamber, iris, lens), second in visualization of inaccessible or difficult to access regions of the eye (area behind iris, ciliary body, pars plana, ora serata, peripheral retina).

Even though there are many types of endoscopes and their combinations, The most commonly used form in ophthalmology is the fiberoptic type of endoscope. Ophthalmic endoscopy is combined with 2 set of components. The first component is the endoscope probe or handpiece that connected with the second component includes the illumination source, laser sources, and video endoscopy systems. The surgeon monitors the course of procedure on the video monitor without stereopsis instead

of the operating microscope. The most commonly used field of view is 110 degree with the depth of field from 0.75 to 4 mm. that created by the objective lens on the distal tip of the fiberoptic image probe. The fiberoptic laser endoscope is most commonly used form of laser in ophthalmic endoscope. The laser fiber is built into the endoscope. Laser sources used in ophthalmology can be combined with endoscopy, including argon, frequency-double Nd:YAG, and 810 nm diode laser.

The new instruments and treatments are researched and developed. One of the research projects is to develop materials to liquefy the vitreous so that the vitreoretinal traction will be released without the need for scleral buckling or vitrectomy. This pharmacologic vitrectomy is a logical treatment but will not be easy to develop the safe dissolution of the collagen fibers within the vitreous and will take time to achieve. Another important research is how to prevent PVR which is a major cause of failure following RRD surgery. By knowing the RRD treatment options and also the advantage and disadvantage of each treatment will help us find the optimal management for patients to gain better anatomic and visual results combined with fewer complications.

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