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The Effect of Phacoemulsification Surgery on Corneal Endothelium

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Abstract

At birth, the endothelial monolayer is approximately 10 µm thick that cover the entire posterior corneal surface and fuses with the cells of the trabecular meshwork. Similarly, the Descemet membrane becomes continuous and fusing peripherally with the trabecular beams. The human corneal endothelial cells either from the center or the periphery of the cornea retain potential proliferative capacity when isolated and cultured in vitro, although they are non-proliferative in vivo. Recent studies have focused on the methods of isolation and cultivation of the endothelial cells derived from the donor's cornea then transplantation into the recipient in cases of corneal endothelial dysfunction. Cataract continue to be an important cause of blindness. The only treatment for cataract is surgical removal to restore the transparency of the visual axis. Elderly, narrow pupil, hard cataract, high phaco power, and high flow rate are considered the main causes of endothelial cell loss during cataract surgery concluding that hard cataract is the main cause which is mainly due to endothelial trauma by nuclear pieces. Postoperative endothelial cell changes and increased corneal thickness occur after cataract extraction with or without lens implantation. These changes occur mainly in the superior cornea in the first week and include increase cell size and decrease the percent of hexagonal cells.

Keywords: Phacoemulsification, Cataract, Corneal Endothelium.

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1. Introduction

The endothelial layer of the cornea maintains corneal clarity by keeping it in a relatively deturgescence state. Endothelial cell density and topography continue to change throughout life. From the second to eighth decades, the cell density declines from 3000-4000 cells/mm^2 to around 2600 cells/mm^2, and the percentage of hexagonal cells declines from approximately 75% to approximately 60% [1]. The central endothelial cell density (ECD) decreases at an average rate of 0.6% per year in normal corneas (Bourne et al, 1997). Endothelial cells do not regenerate; however, it has a significant reserve at birth, with cell density (CD) about 3500 cells/mm^2. This number decreases with time at approximately 0.6% per year. Eyes with ECD below 500 cells/mm^2 may develop corneal edema. Endothelial cell morphology also plays a role in pump function. An increase in cell size (polymegathism) and an increase in variation of cell shape (pleomorphism) result in decreased ability of the endothelial cells to dehydrate the stroma [2]. The use of specular microscopy is limited to healthy corneas without significant edema, scarring, deposits, or opacities. Clear media is required to avoid distortion of light and allows a clear view of the endothelium [3]A minimum of 50 cells per image should be detected to obtain maximum accuracy of ECD results [4].

1.1 Anatomy of the Cornea

The cornea is a transparent avascular tissue covering the front part of the eye. It is one of the most highly innervated tissues in the body. In the average adult, the horizontal diameter of the cornea is 11.5 to 12.0 mm and about 1.0 mm longer than the vertical diameter. It is approximately 0.5 mm thick at the center and gradually increases in thickness toward the periphery. The shape of the cornea is prolate (flatter in the periphery and steeper centrally), which creates an aspheric optical system. The human cornea consists of 6 recognized layers epithelium, Bowman membrane, stroma, Dua layer, Descemet membrane, and endothelium [5].

1.1.1 The Corneal Endothelium &Descemet Membrane

The endothelial layer is a monolayer, which appears as a honeycomb-like mosaic when viewed from the posterior side. In early embryogenesis, the posterior cornea is lined with a neural crest-derived monolayer of orderly arranged cuboidal cells [6] Over time, these cells flatten and become tightly adherent to one another. Immediately

anterior to the flattened layer is a discontinuous homogeneous acellular layer, which in time becomes the Descemet membrane [7].

1.1.2 Descemet Membrane

Beginning in utero at the 8-week stage, endothelial cells continuously secrete the Descemet membrane. it can accumulate up to 10 µm in thickness with age.

1.1.3 Physiology of Corneal Endothelium & Pathophysiology of corneal edema

The corneal endothelium acts as incomplete barrier which prevents free flow of water into the cornea. This effect is opposed by the intraocular pressure which tends to draw water into the cornea and the positive imbibition pressure of the stromal proteoglycans (60 mmHg). This dehydration is activated by the osmotic gradient from a relatively hypo-osmotic stroma toward a relatively hypertonic aqueous humor. It is a passive movement and energy is required for transporting ions to generate the osmotic gradient. The two essential ion transport systems are the membrane-bound Na+ and K+ -ATPase sites and the intracellular carbonic anhydrase pathway. Activity of these pathways results in partially dehydrated stroma with water content of about 78% [8]The endothelial cells decrease with age, trauma, inflammation, and other disease processes (Fuchs dystrophy). However, the remaining cells stretch to cover the area of degenerated cells. Accordingly, the remaining cells enlarge (polymegathism) and lose their hexagonal shape (pleomorphism). Hypertonicity of the tear film due to evaporation of its water content at the corneal surface helps also to draw water out of the cornea. Thus, corneas are slightly thicker in the morning due to low evaporation under closed eyelids also due to the decrease in metabolic activity of endothelial cells. Such corneal hydration is increased in persons with damaged endothelium. This usually results in blurring of vision after awakening which gradually resolves with time [19].

1.1.4 Corneal endothelial regeneration

Corneal endothelial cells have little or no ability to regenerate after birth. However, some studies suggest that endothelial cells keep some degree of proliferative potential even in adult life [10].

1.1.5 Corneal thickness

Central corneal thickness (CCT) is an important indicator of corneal endothelial cell function. It is about 0.5 mm centrally which increases gradually towards the periphery to reach about 0.7 mm. It can be measured by contact and non-contact methods. Contact methods as ultrasound pachymetry and contact specular microscopy. Non-contact methods as pentacam, non-contact specular microscopy, ultrasound biomicroscopy (UBM), anterior Segment-Optical coherence tomography (AS-OCT)[11] .

1.1.6 Corneal Endothelium Examination

Assessment of corneal endothelium can be done using slit-lamp biomicroscopy, specular microscopes and confocal microscopes. Slit-lamp specular examination has some difficulties as low magnification, difficult visualization by scattered light and small area of examination. Noncontact specular microscope represents a method of choice for clinical evaluation of the endothelium. It is easy, convenient to the patient, with a clear imaging of the endothelial cells. Confocal microscope provides a wide-field corneal imaging and can be used with corneal edema when visualization is difficult by specular microscope [12].

1.1.7 Specular Microscopy

Specular microscopy (SM) is a tool to study layers of the cornea under high magnification (100 times that of slit-lamp). It is mainly used to evaluate corneal endothelium regarding its count, shape, and distribution. SM is essentially used to assess the endothelial functional reserve, especially before intraocular surgeries or to evaluate the donor cornea in penetrating keratoplasty [13].

1.1.8 Optics and types of specular microscopes

Only 0.02% of the incident light is reflected at the endothelium-aqueous interface due to the small difference in refractive indices in-between. This reflected light from the endothelium can be photographed and analyzed [14] However, the reflected light at the epithelial surface interfere with a good endothelial view. To overcome such interference, two methods are used: Using a contact lens, with a coupling fluid whose refractive index is like that of the cornea. It has the advantage of higher magnification and checking of eye movement, yet patient intolerance is an obstacle and non-contact method: The incident light is adjusted by so that the reflected light from the epithelial surface is moved away of the specular endothelial reflection [15]. A light slit is projected onto the corneal endothelium, and the specularly reflected light is captured by a camera positioned at the same angle of incidence of the projected light. The illuminated portion by the slit is the capture area.

1.1.9 Evaluation criteria

Analysis of endothelial cells includes: The mean cell area (μm^2), cell density (CD): normally $\geq$ 2500 cells/mm^2, coefficient of variation (CV); cell size variability: Normally $\leq$ 30 (ranges from .22 to 0.31). CV values from 0.32 to 0.40 are elevated, and above 0.40 are abnormal. CV=SD/ mean cell area (SD of the mean cell area). Standard deviation (SD) $\leq$ 120, percent of hexagonal cells (normally above 60%), degree of pleomorphism and polymegathism. Polymegathism is defined by CV value and average cell size = 200-500 μ^3 [16].

1.1.10 Characteristics of an abnormal endothelium

ECD < 1500 cells/ mm^2, Severe polymegathism: increase in the variation of individual cell area. Pleomorphism: abnormal cell morphology with decrease in the percentage of hexagonal cells (<50%). Pleomorphism may affect the fluid barrier function of the endothelium. Those patients are at risk of developing postoperative corneal edema, presence of corneal guttata, abnormal single-cell defects, extensive areas of severe edema and presence of inflammatory cells on the endothelium.

1.1.10.1 Cataract and Phacoemulsification

Cases of blindness increased from 12.3 million in 1990 to 20 million in 2010, worldwide. These cases represent 12.7 % of blind patients in North America to 42 % in Southeast Asia [17].

1.1.10.2 Risk Factors

Several risk factors for cataract have been identified such as Age, Smoking, Sunlight exposure, Poor lifestyle habits, Metabolic syndrome, Diabetes mellitus, HIV/AIDS, and Systemic corticosteroid use [18]**.**

1.1.10.3 Clinical Presentation

Most cases occur in patients over age 60. Younger individuals with Cataract are likely to have risk factors. Patients of all ages are subject to cataract due to significant eye trauma. The clinical presentation of age-related and non-age-related Cataract is the same. In many patients, there is "myopic shift," and is caused by an increase in the refractive index of the lens[19]**.**

1.1.10.4 Diagnosis

The diagnosis of Cataract is based on characteristic findings of lens opacity. Cataract typically have one of three components: nuclear opacity, cortical spoking, and posterior subcapsular haze. Each affects a different anatomical part and has different symptoms and progression. Most patients have a combination of components. Cataract are also classified according to their maturity. A mature cataract degrades vision to the 3/60 level or worse. A hyper mature cataract is one in which the cortex of the lens has liquefied [19]**.**

1.1.11 Classification

Cataract are classified as having nuclear, cortical, or posterior subcapsular components, evident on examination. Most patients have a combination of components. Nuclear cataract: It dulls colors and white significantly. It progresses very slowly. Distance vision is affected much more than near vision. Cortical cataract: It does not degrade vision very much, although, it is quite evident on exam. It may appear suddenly after trauma but tend to progress slowly. Posterior subcapsular cataract: It tends to cause disabling glare in bright sunlight and from headlights, even if visual acuity is affected slightly. Typically, distance and near vision are affected equally. It tends to progress more quickly than nuclear cataract. Steroid use and diabetes are associated with the formation of this type of cataract [19]**.**

1.1.11.1 LOCS III classification of cataract

The Lens opacities classification system III (LOCS III) contains an expanded set of standards that were selected from the Longitudinal Study of Cataract slide library at the center for Clinical Cataract Research, Boston, Mass. It consists of six slit-lamp images for grading nuclear color (NC) and nuclear opalescence (NO), five retro- illumination images for grading cortical cataract (C), and five retro-illumination images for grading posterior subcapsular (P) cataract. Cataract severity is graded on a decimal scale, and the standards have regularly spaced intervals on a decimal scale [20]**.**

1.1.12 Indications for Surgery and Timing

Surgery is indicated when it interferes with the patient's ability to perform his needs of daily life; there are no criteria related to visual acuity level [21]**.**

1.1.13 Surgical techniques

The current technique for cataract extraction is Phacoemulsification. The lens is removed through a small (2.4-3-mm) incision. It is not feasible in mature Cataract with very hard nucleus. The small incision is self-sealing, of more rapid visual recovery and the decreased likelihood of suture-induced astigmatism [22].

1.1.14 Corneal Endothelial Cell Loss during cataract surgery

Corneal endothelial cell loss (ECL) occurs during anterior segment operations due to manipulation, whirling cortical or nuclear lens fragments, flow of fluid in the anterior chamber and intra-cameral pharmacological agents used during surgery. Phaco generates also heat and turbulent flow in the anterior chamber [23]**.** ECL may result in stromal edema that ranges from striate keratopathy to deep corneal edema, clouding and decompensation. Mild striate keratopathy needs lubricants and topical steroids and resolves within two to three weeks. However deep stromal edema usually needs hyperosmotic agents with topical steroids [24]**.**

1.1.15 Power modulation protects the corneal endothelium

The total amount of phaco energy is the most important indicator of postoperative visual acuity and corneal clarity. It was reported that the effective phaco time (EPT) is directly proportional to the degree of nuclear hardness and the amount of ECL. The highest cell loss (13.35%) was reported in mature Cataract and the lowest (6.76%) was in PSC [25]**.**

1.1.16 Torsional phacoemulsification

It produces side to side movement of the phaco tip (instead of forward and backward movement) which allows proper flowability of the nuclear matter with less repulsion at the phaco tip caused by conventional longitudinal US. Liu et al showed significant reduction in endothelial cell loss by torsional U/S (12.5%) compared to 19% with conventional phaco [26]**.**

1.1.17 Cataract surgery in eyes with compromised endothelium

Symptomatic corneal edema may or may not be present at the time of surgery. Specular microscopy and pachymetry should be performed preoperative for all patients with suspected endothelial shortage or defect corneal edema. Trauma during surgery were cause more endothelial loss [27-9]**.**

1.1.18 Surgical Technique

Careful pre-operative evaluation and intimate surgical techniques are necessary to minimize the endothelial cell injury during surgery and improve the postoperative results and visual outcome . [27]Other factors which may aggravate ECL in compromised corneas include long phaco time, short axial length and posterior capsular rupture [29-28]The more posterior scleral tunnel approach is preferred than the temporal clear corneal one. Jagani et al found that the mean ECL by clear corneal incision was about 13.6%, while in scleral tunnel, it was about 11% at 1week, which is considered statistically significant, but there is no statistically significant difference in ECL between both techniques at 6weeks, 3months postoperatively.

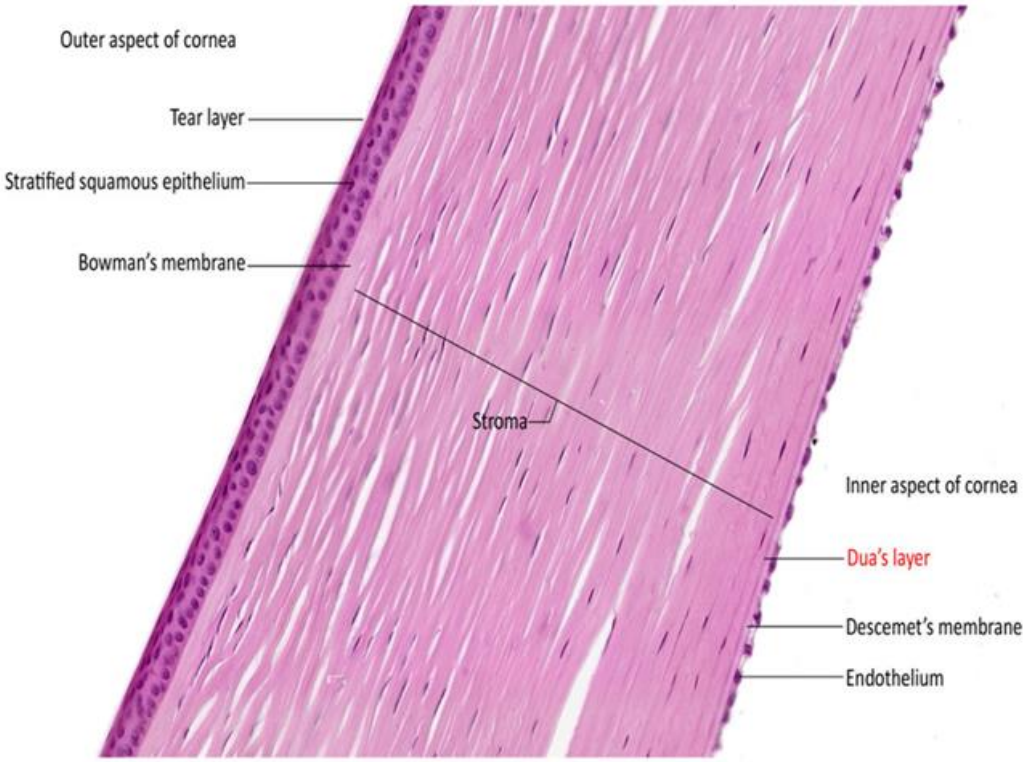

Figure 1: Anatomy of the cornea

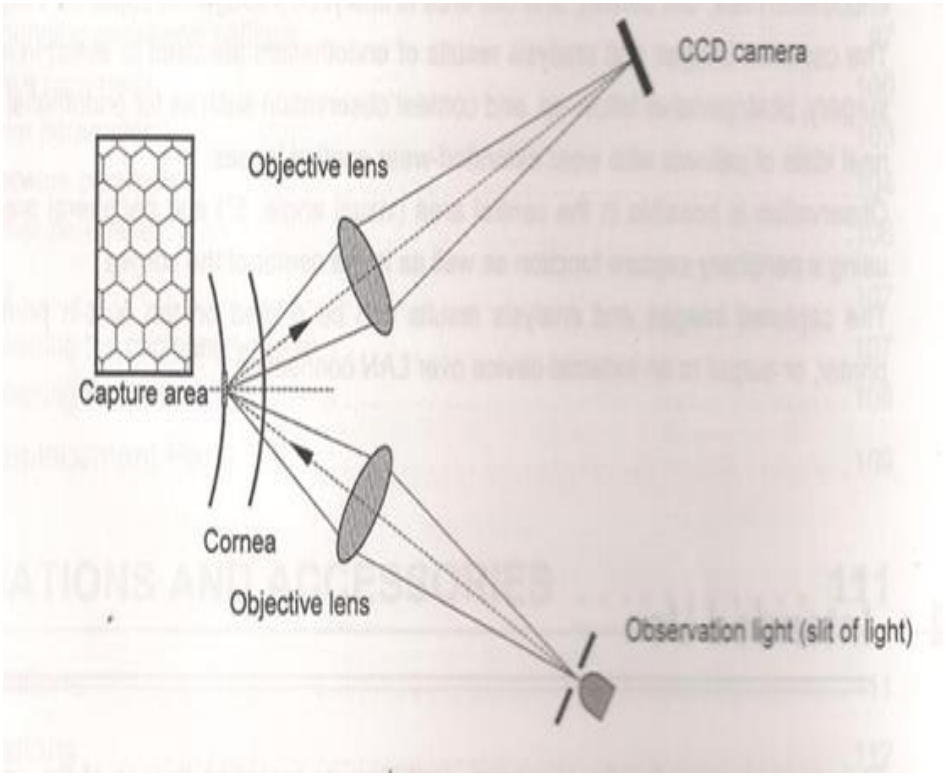

Figure 2: Optical Konan noncontact specular microscope (Class-1 Japan)

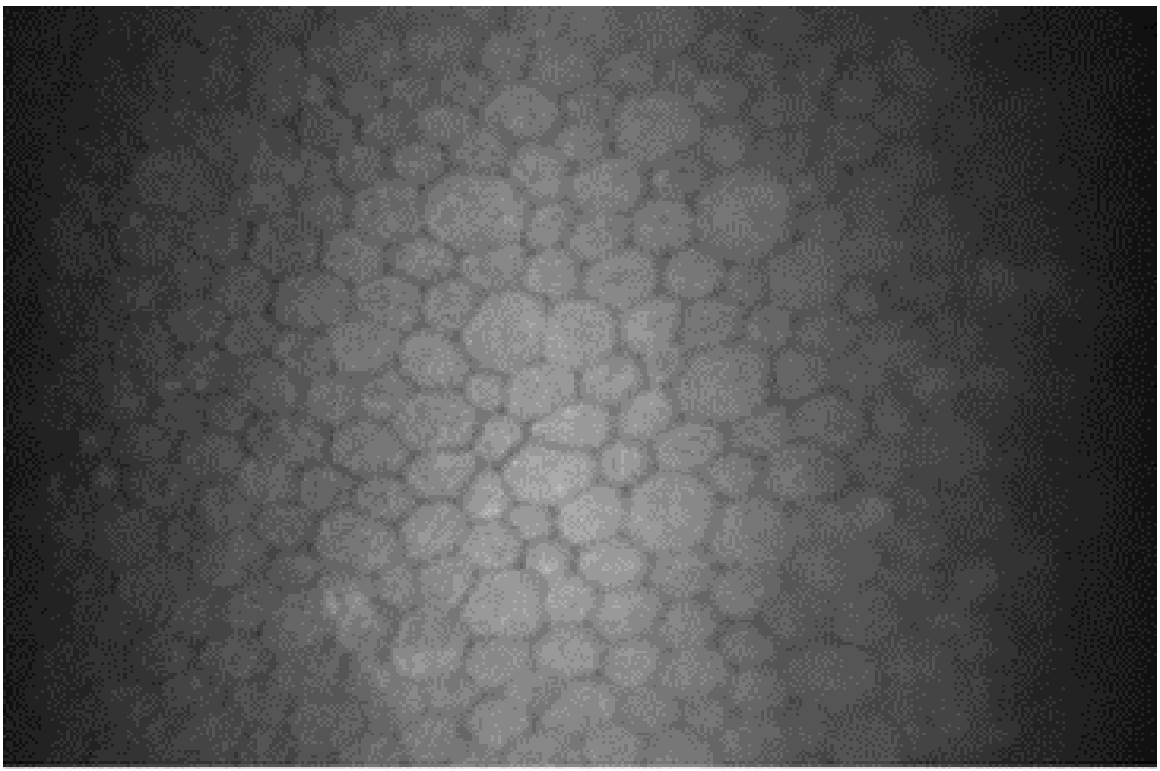

Figure 3: Cornea with polymegathism and pleomorphism

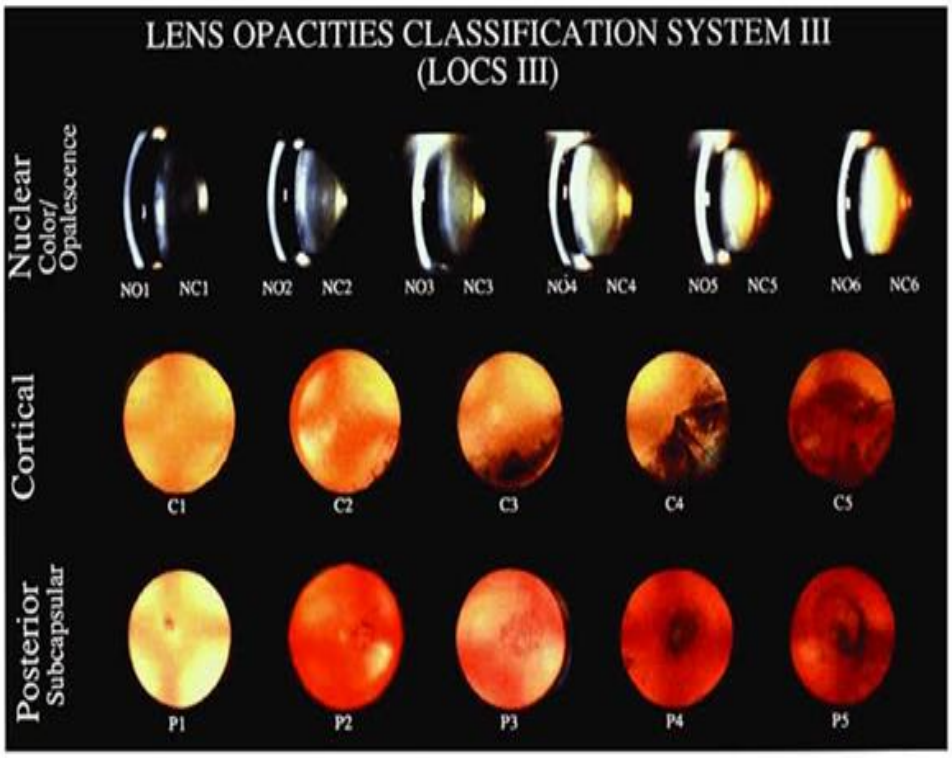

Figure 4: Lens opacities classification system III (LOCS III)

The Lower viscosity dispersive OVDs (Viscoat) are more protective than cohesive ones as they coat the corneal endothelium and protect it from the turbulence produced by phaco [30]Soft shell technique (SST) is first described by Arshinoff and Norman [31]and it allows the use of both dispersive and cohesive types of OVDs in the same operation to take the benefits of both and avoid their drawbacks. SST is safer than Healon alone in protection of the corneal endothelium which is evidenced by several studies as it causes less increase in the CCT and less ECL and in cases of Fuchs endothelial dystrophy and dense cataract [32]**.** Tri-shell technique: in which dispersive OVD is used with visco-adaptive one instead of cohesive OVD with the balanced salt solution (BSS) used as inferior shell below the viscoadaptive. This technique preserves maximal corneal protection together with the function of viscoadaptives over cohesives in anterior chamber (AC) maintenance and stabilization. Viscoadaptive OVDs (Healon 5) change its nature and behavior according to the degree of fluid turbulence in the anterior chamber. They act as high viscous, cohesive OVD at low flow rate and have pseudo-dispersive properties at higher flow rate [31]Extra-capsular cataract extraction (ECCE) is safer than phacoemulsification in patients with hard cataract with the mean ECL was 18.9% in phaco group versus 11.8% in ECCE group [33]**.**

1.1.19 Regional changes

Regional differences in endothelial cell density ECD after cataract surgery may be significant. Muller et al reported regional differences in ECD after PC IOL implantation with great changes by 1 month, the loss was about 11, 20, and 5% in the central, superior, inferior corneal regions respectively [34]**.**

1.1.20 Other factors affecting corneal endothelium

Irrigating fluid: To protect the endothelium, the irrigating solutions must have appropriate osmolarity and PH around 306 milliosmoles and 7.4 PH respectively, and that calcium, glutathione and bicarbonate are present. Calcium protects the corneal endothelium as calcium free solutions damage the junctions between endothelial cells and affect the barrier function. Glutathione is a reducing agent that might protect the endothelium from the oxidative effects. A bicarbonate buffer system is preferable because bicarbonate is the normal aqueous human buffer. The addition of glucose and adenosine (as a source of energy) improves the pump function of the endothelium [16]**.**

1.1.20.1 Air Bubbles

Kim et al studied the effect of air bubbles on the corneal endothelium. They found that air bubbles in the intraocular fluids with high surface tension could cause a ring-shaped pattern of damage to the endothelium. The mechanism by which this pattern has appeared seems to be a surface tension phenomenon [35]**.**

1.1.21 Intraoperative complications

Difficulties encountered during surgery as those caused by shallow anterior chamber (as in glaucoma), narrow pupil, weak zonules (as in pseudoexfoliation), or even actual lens subluxation, may carry great risk to the corneal endothelium. Other complications as posterior capsular rupture, vitreous loss, and dislocated lens fragments may also add to the corneal risk. Jaffe et al reported that the risk of ECL is increased in patients exposed to intraoperative capsular rupture or vitreous loss, ECL was ≥ 15% which was considered as severe cell loss in the study [36]**.**

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