Early postoperative capsular block syndrome related to saccadic-eye-movement-induced fluid flow into the capsular bag

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ABSTRACT

Purpose: To determine the relationship between saccadic eye movements and the development of early postoperative capsular block syndrome.

Setting: Fundacion Oftalmologica, Los Andes, Santiago, Chile.

Methods: An artificial experimental setup was designed to reproduce the anatomical, hydrodynamic, and kinetic conditions of an early postoperative pseudophakic eye with a capsulorhexis and intracapsular intraocular lens (IOL) implantation. An electromechanical transducer driven by a digital-to-analog converter was used to mimic saccadic eye movements at physiological speeds and accelerations. Position information and differential pressure measurements between the intracapsular space and the extracapsular space were digitized. Various anterior capsule–IOL configurations were tested including partial adhesion of the anterior capsule to the lens.

Results: Saccadic movements increased intracapsular pressure by displacing fluid into the capsular bag. This finding was inconstant and only observed when the capsular rim adhered to the IOL optic by more than 70%. Development of positive intracapsular pressure was noted above 4 mm Hg.

Conclusion: A valve-like mechanism formed by the capsulorhexis rim partially adhered to the IOL optic can occur postoperatively. Under these conditions, saccadic eye movements can increase intracapsular pressure by a unidirectional inertial displacement of fluid into the capsular bag. *J Cataract Refract Surg 2000; 26:415–419 © 2000 ASCRS and ESCRS*

Early postoperative capsular block syndrome is considered a complication of capsulorhexis.[1–3] Typical findings of this condition appear between 1 day and 2 weeks after surgery[1] and comprise slight to moderate myopia, a shallow anterior chamber, and a potential intraocular pressure (IOP) rise. The underlying phenomenon that promotes these signs is hyperexpansion of the capsular bag by fluid. Slitlamp examination reveals a forward displacement of the anterior capsule and the intraocular lens (IOL) optic and a backward displacement of the posterior capsule.

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The origin of the fluid trapped in the capsular bag remains unclear. It has been suggested that retained viscoelastic substance or endocapsular epithelial cell products produce an osmotic gradient that drives water into the capsular bag.[2,4] That hyperexpanded capsular bags can be detected as soon as 24 hours after surgery and that the osmotic forces potentially involved are considered too weak to move fluid across the lens capsule[2] have led to a complementary theory to explain the pathogenesis of early postoperative capsular block syndrome.

I propose that saccadic eye movements may have a role in the introduction of fluid into the capsular bag. It is accepted that saccadic eye movements can reach rotational speeds up to 1000 degrees per second in normal patients. This is equivalent to a linear displacement of approximately 17 cm per second at the capsulolenticular plane of a normal eye. The high accelerations and decelerations involved in this rapid movement can promote a net flow of aqueous humor into the capsular bag.

This inflow seems to require particular anatomic conditions and elevation of a portion of the capsular rim above the anterior IOL surface. This partial elevation can usually be seen at the end of surgery and soon after (Figure 1, *A*). A valve-like structure must exist between the capsulorhexis rim and the IOL's anterior surface to retain the inflow inside the capsular bag (Figure 1, *B*). A glue-like tissue at the capsular rim adhering the capsule to the IOL surface has also been described in the early postoperative period.[4] This tissue may help create this proposed valve (Figure 2). Aqueous humor could then be pumped into the capsular bag by rapid eye movements that produce an inertial unidirectional displacement of fluid toward the capsular bag.

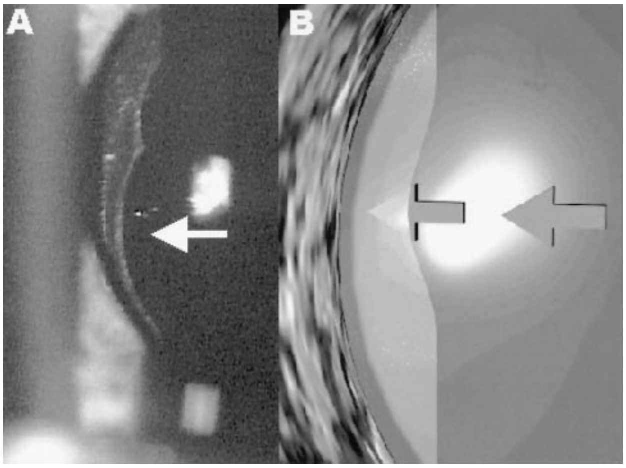

Figure 1. (Zacharias) *A*: Slitlamp videophotograph taken on the first postoperative day in a patient with capsulorhexis and an intracapsular IOL. The capsular rim is partially elevated above the IOL surface (*arrow*). *B*: A schematic representation shows the suggested unidirectional path of fluid between a partially elevated capsular rim and an underlying IOL optic (*arrows*) in response to inertial fluid displacements produced by saccadic eye movements.

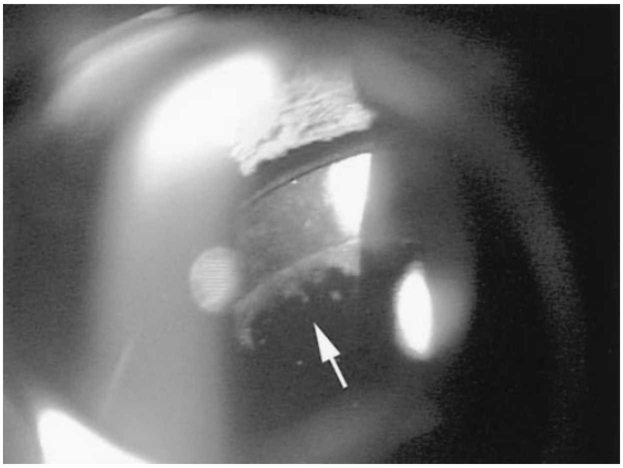

Figure 2. (Zacharias) A slitlamp videophotograph shows adherence between the capsular rim and the IOL optic occurring soon after surgery. This adherence is produced by a glue-like tissue (*arrow*).

Materials and Methods

An in vitro setup was created to simulate the anatomic and kinetic conditions in the early period after cataract surgery. This setup consisted of a reproduction of the globe, an extracapsular space, and an intracapsular space, separated by a capsulolenticular array (Figure 3). The extent of the adhesion between the perimeter of the capsule and the IOL surface was varied by applying a plastic adhesive between the capsular rim and the underlying IOL optic to different extents. The purpose was to reproduce the conditions that may cause early postoperative capsular block syndrome.

The globe was mounted with the rotation center parallel to the capsulolenticular plane. The extracapsular and the intracapsular spaces were separately connected to a differential pressure transducer (NovaSensor 410 0050D3L). Tubing exiting the globe was conducted through the axis of rotation to avoid centrifugal forces that would interfere with the experiments. A constant IOP of 20 mm Hg was applied during the extracapsular chamber experiments. A 3-way stopcock controlled the flow of balanced salt solution to the extracapsular space

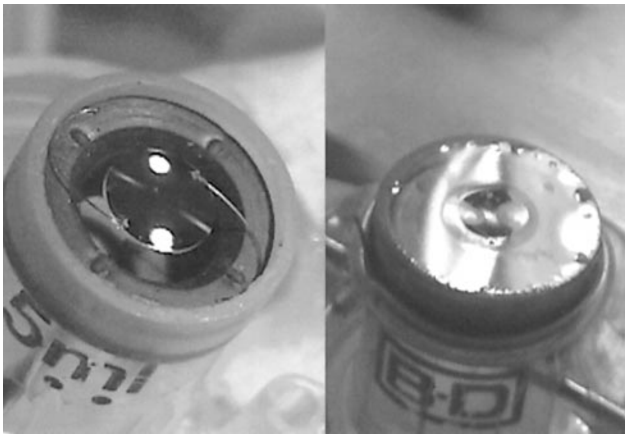

Figure 3. (Zacharias) An acrylic IOL positioned in a special holder inside the uncovered artificial globe before placement of the plastic membrane that simulates the anterior capsule (*left*). An artificial capsule with a capsulorhexis has been placed on top of the IOL optic to simulate the naturally occurring capsulolenticular relation. Different proportions of the capsulorhexis perimeter were adhered to the IOL optic with a water-resistant adhesive. Tangential force was applied while attempts were made to elevate portions of the capsule in the nonadhered part of the circumference (*right*).

alone or transiently connected it with the intracapsular space to equalize pressures and reset the gradient to zero. Motion was applied to the eye model with a computer-controlled electromechanical transducer.

The perimeter of adhesion between the IOL and the capsular rim was always set symmetrical to the plane of rotation of the globe, promoting a free overlapping rim zone that would always travel parallel to the direction of movement. Experiments were divided in 4 groups: Group A, no capsular rim adhesion; Group B, 30% to 50% adhesion; Group C, 50% to 70% adhesion; Group D, 70% to 90% adhesion (Figure 4). The capsular setup was repeated 3 times in Groups A, B, and C and 8 times in Group D. After motion was applied with the nonadhered capsular rim lying flat over the IOL surface, the free capsular rim was manipulated with a blunt forceps to create a degree of elongation, avoiding flat contact with the underlying IOL and approximating the capsule configuration in Figure 1, *B*. Several experiments were performed with each capsule–IOL array.

The movement pattern applied to the artificial globe followed a waveform that simulated saccadic eye movements. This waveform was extracted from an actual digitized electro-oculogram (EOG) recording of a normal eye (Figure 5) and fed through a digital-to-analog converter to the electromechanical transducer. A motion sensor was used to monitor the actual waveform of the globe's rotation. A drawing and a photograph of the experimental setup are shown in Figures 6 and 7, respectively.

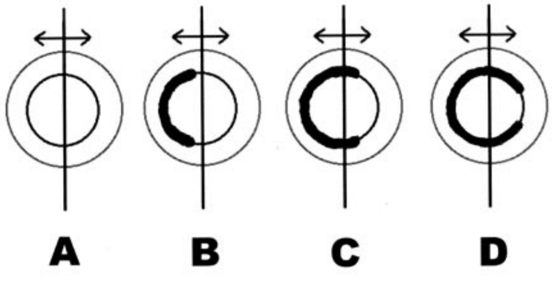

Figure 4. (Zacharias) Drawing of the artificial capsule setup over the IOL optic. The outer circle represents the IOL optic and the inner circle, the overlying capsular rim. The thick line in the inner circle corresponds to the adhered portion of capsular rim to the IOL surface, achieved using an elastic adhesive. The vertical line represents the axis of rotation. The arrow points in the direction of saccadic oscillations. Group A (*A*) shows no capsular rim adhesion; Group B (*B*), 30% to 50% adhesion; Group C (*C*), 50% to 70% adhesion; Group D (*D*), 70% to 90% adhesion.

Results

No pressure rise was observed in Groups A, B, and C with or without free capsular rim deformation. Group

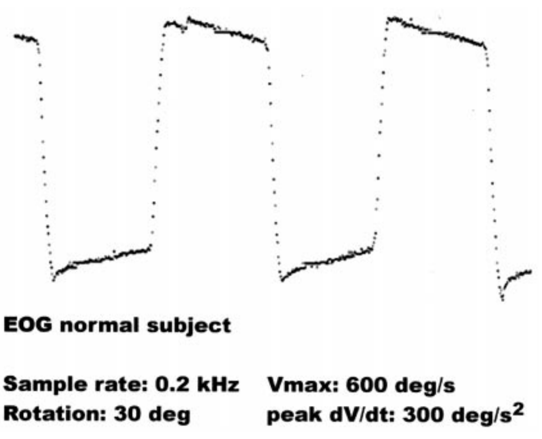

Figure 5. (Zacharias) Actual EOG recording digitized from a normal eye and used to apply motion in a similar pattern to the artificial globe to simulate saccadic eye movements.

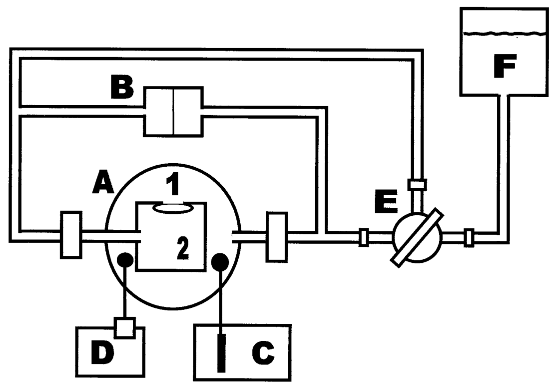

Figure 6. (Zacharias) The interconnection of elements composing the experimental setup used to test the development of a positive intracapsular pressure in an artificial globe exposed to saccadic movements. *A*: Artificial globe containing an extracapsular chamber (A1) and an intracapsular chamber (A2) separated by a capsulolenticular array. *B*: Differential pressure transducer. *C*: Electromechanical transducer used to apply movement to the artificial globe. *D*: Motion sensor used to detect the actual movement of the globe. *E*: Three-way stopcock used to apply IOP and equalize pressure differences between the intracapsular and extracapsular chambers before and after experimental runs. *F*: Fluid reservoir leveled 27 cm above the artificial globe to provide a constant IOP of 20 mm Hg.

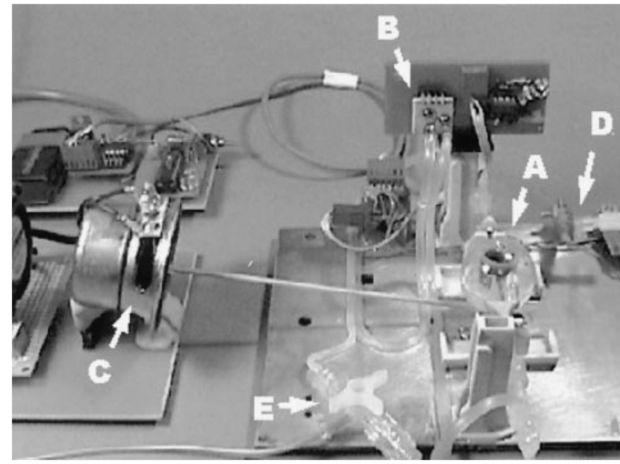

Figure 7. (Zacharias) The experimental setup. *A*: An artificial globe containing an extracapsular and intracapsular chamber separated by a capsulolenticular array. *B*: Differential pressure transducer. *C*: Electromechanical transducer to apply movement to the artificial globe. *D*: Motion sensor to detect the actual globe movement. *E*: Three-way stopcock to apply IOP and to equalize pressure differences between the intracapsular and extracapsular chambers before and after experimental runs.

D showed different degrees of motion-related progressive intracapsular pressure development. A pressure gradient was measured in 4 of the 8 capsule setups in Group D and only after deformation was applied to the free border, avoiding flat contact with the underlying IOL.

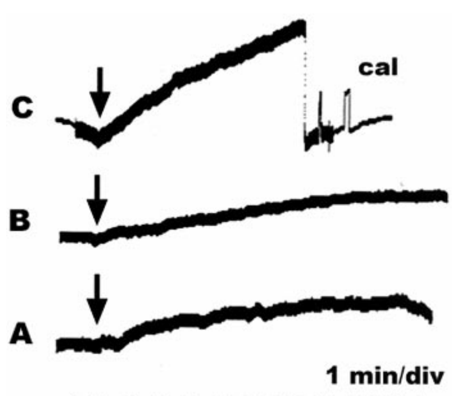

Figure 8. (Zacharias) Tracings A, B, and C correspond to different experiments performed with a capsular rim adhesion of 85% to the IOL optic. The arrows mark the moment when the communication between the intracapsular and extracapsular spaces was closed. The artificial globe was already being subjected to motion. A gradual increase in intracapsular pressure can be observed. Tracing C returns to baseline after communication between both chambers opens, showing that the pressure difference is real and not a baseline drift. Calibration mark (cal) = 1 mm Hg.

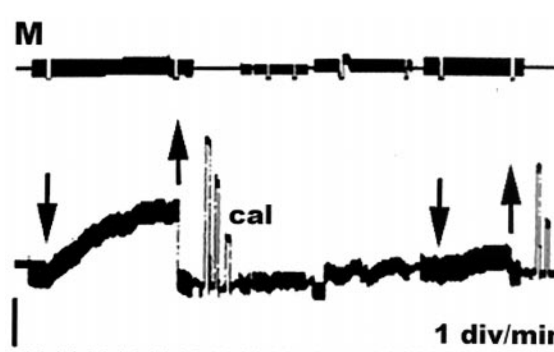

Figure 9. (Zacharias) Upper tracing (M) shows the motion sensor recording of a long experimental run in which motion patterns were varied between EOG (*left*), sine wave (*middle left*), sine-wave double amplitude (*middle right*), and EOG (*right*). The lower tracing shows the corresponding differential pressure measurements with a single capsulolenticular setup. The arrows pointing down show the closure of the external communication between the intracapsular and extracapsular spaces. Arrows pointing up mark the opening of the external interchamber. The experiments shown in the left and right corners were performed under the same motion pattern and capsule setup, but pressure buildup was different between the experiments. Calibration marks (cal) = 5 to 4 to 2 mm Hg.

Figure 8 shows pressure-gradient measurements from 3 experiments in Group D.

Pressure rise reached a plateau of stabilized differential pressure after approximately 10 minutes of permanent saccadic movements. Different maximum pressure gradients extending above 4 mm Hg occurred in the intracapsular space (Figure 8, *C*). The pressure rise was inconstant, and repeated runs with the same capsulolenticular setup yielded different amounts of pressure buildup (Figure 9).

Discussion

An artificial model was used to test the hypothesis that saccadic eye movements can force fluid into the capsular bag in the early postoperative period. Adhesion between the capsular rim and the IOL surface and some amount of capsular rim deformation were necessary to create a motion-dependent pressure gradient. Early adherence of the capsular rim to the IOL is commonly observed in vivo. The capsule's disposition over the IOL is variable and depends on the capsulorhexis shape and size, IOL position, progressive rim cell proliferation adhering to the IOL optic, and capsule characteristics of individual patients, among other factors.

It is possible that a valve-like mechanism can be formed between the anterior capsule and the underlying IOL when a combination of these factors occurs. Rapid eye movements can then act as a motor that pumps fluid inside the capsular bag, potentially reaching pressures able to distend the capsular bag. There are recent reports of capsular block syndrome occurring with sulcus-implanted IOLs. In this situation, the IOL optic was overlying, not underlying, the capsular rim. An adhering tissue between the capsulorhexis margin and the IOL optic is a constant observation.[5] Early postoperative capsular block syndrome has also been reported after cataract surgery when no viscoelastic substance was used.[5] These findings confirm that at least in some cases, the theory that retained viscoelastic substance is responsible for capsular block syndrome cannot be sustained.

Single saccadic eye movements in a proper direction can force the entrance to the capsular bag of small amounts of fluid from the extracapsular space to be retained inside the capsular bag by the valve-like nature of a favorable capsulolenticular relationship. A constant factor in the production of capsular block syndrome appears to be the adhesion of the anterior capsule to the IOL.

Repetition of rapid eye movements would keep fluid flowing into the capsular bag until a pressure equilibrium is reached. At this moment, the glue-like tissue grows to completely seal the capsular opening, keeping the pressurized fluid entrapped within the capsular bag, tending to chronicity. Thus, later changes in fluid composition may occur.

This study shows that rapid eye movements can produce a unidirectional fluid flow into the capsular bag in the presence of a mixture of favorable anatomic conditions. It cannot be stated from these experimental results that early postoperative capsular block syndrome is fully dependent on saccadic eye movements. What is suggested is that saccadic eye movements can promote aqueous humor flow into the capsular bag and that this inflow can be retained by the valve-like nature of the capsule–IOL relation initiating a capsular block condition. Other factors such as osmotic gradient or cellular by-products could also participate in the genesis, maintenance, and aggravation of the condition.

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