

Articles

A mathematical description of causative factors and prevention of elevated intraocular pressure after keratoplasty

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In keratoplasty with grafts the same size as the recipient bed, tight sutures and thick recipient corneal periphery distort the angle and may collapse the filtering meshwork. This can cause very high postoperative pressures, which can be avoided by the use of donor grafts larger than the recipient bed. These relationships can be mathematically predicted.

Key words: keratoplasty, trabecular collapse, angle distortion, elevated pressure, aphakia.

With the advent of electronic applanation tonometry and its proved efficacy in the face of edematous or irregular corneas,¹⁻³ it was discovered that most patients undergoing aphakic keratoplasty and even more in keratoplasty with cataract extraction would have elevated pressures postoperatively. Irvine and Kaufman⁴ had a mean maximum pressure rise of 40 mm. Hg in aphakic transplants and of 50 mm. Hg in combined transplants and cataract extraction. This was not related to preoperative glaucoma, and on gonioscopy, angle

closure was not seen. Phakic grafts, on the other hand, did not have a postoperative pressure problem.

Wood et al.⁵ further showed that this pressure rise would usually return to normal levels over a period of days to a few weeks and found little change in the pressure with acetazolamide treatment. Although this is usually the case, it has been our experience that a significant percentage of patients go on to have severe pressure problems that can be resistant to all modes of medical treatment and eventually require cyclocryotherapy.

Zimmerman et al.⁶ have shown that in phakic transplants done in eyebank eyes, there is no outflow facility change with keratoplasty. Such was not the case with aphakic keratoplasty in eyebank eyes, where perfusion studies showed an average of 37 percent decrease in outflow facility compared to the control after keratoplasty where the donor and recipient trephines were the same

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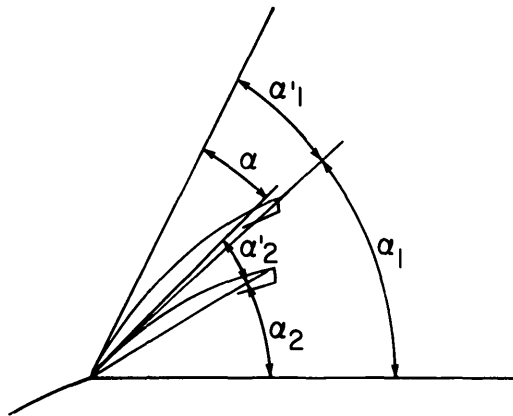


Fig. 1. Angle relationships before (α_1 and α_1') and after (α_2 and α_2') suturing in keratoplasty.

size. Further work with an 8.0 mm. donor in a 7.5 mm. bed blocked this decrease in outflow.⁷

At the University of Florida and Washington University, St. Louis, a randomized study⁹ has just been completed that clearly showed a decreased postoperative intraocular pressure in aphakic and combined-procedure keratoplasties where a donor 0.5 mm. larger than the recipient bed was used. This improvement was not so great when an 8.5 mm. donor in an 8.0 mm. recipient was used as compared to an 8.0 mm. donor in a 7.5 mm. recipient.

This paper is a mathematical presentation of factors altering the angle after keratoplasty in an attempt to explain what is happening in postkeratoplasty elevated intraocular pressure.

Mathematical derivation

Keratoplasty in cross-section leaves two recipient corneal arms with a certain thickness (C_1) and length from the limbus (D_1). For the purpose of this paper we will consider a keratoplasty that is perfectly centered. The central wound (W_1) has a diameter equal to the trephine diameter. The corneal diameter limbus to limbus (L_1) is also a measurable item. After suturing of the donor cornea all these relationships can change by the effect of tissue compression and shortening by the suture as well as a possibly larger or smaller donor cornea compared to the original wound size (W_1). We will call these postsuturing relationships D_2 , W_2 , and L_2 , and all of these can be measured.

The peripheral cornea has a certain radius of curvature (R_1), and suturing could change this by pulling and flattening the recipient cornea (R_2). The only other definitions we need to begin our derivation is the definition of five angles (Fig. 1). α_1 we will define as the angle from the limbal plane to the chord of the recipient cornea before suturing. α_2 is the same angle after suturing. α_1' is the angle between the chord of the recipient cornea and the tangent to the cornea at the limbus, whereas α_2' is the same relationship to α_2 after suturing. We will start out by trying to explain the change in the angle of the tangent to the cornea at the limbus from a fresh and untouched eye, to that same angle after keratoplasty and call this change α .

$$\alpha = \alpha_1 + \alpha_1' - \alpha_2 - \alpha_2'$$

We have defined alpha in terms of four angles, and now we will see if we can define each of the angles in terms of L_1 , L_2 , W_1 , W_2 , D_1 , D_2 , R_1 , and R_2 (Fig. 2).

$$\cos \alpha_1 = \frac{1/2 (L_1 - W_1)}{D_1}$$

and

$$\cos \alpha_2 = \frac{1/2 (L_2 - W_2)}{D_2}$$

The derivation of $\alpha_1' + \alpha_2'$ is a little more difficult. The angle between the radius and the perpendicular dropped from the chord D_1 to the center of the corneal curve is equal to α_1' because its opposing acute angle and α_1' make up a right angle (the tangent is always perpendicular —i.e., 90 degrees to the radius of a circle at its point of contact) (Fig. 3). Our perpendicular to the chord through the center of curvature bisects the chord because the chord plus two radii through the point of intersection of the chord and the circle make up an isosceles triangle. Now we can define α_1' in terms of things we know.

$$\sin \alpha_1' = \frac{1/2 \text{ chord length}}{R_1} = \frac{1/2 D_1}{R_1}$$

It then follows that:

$$\sin \alpha_2' = \frac{1/2 D_2}{R_2}$$

It is also apparent that a cross-section of the limbal plane is a chord that intersects the cornea at the point of contact of the limbal tangent to the cornea. From our foregoing analysis, then, the angle between the limbal chord and the limbal tangent which we called $\alpha_1 + \alpha_1'$ is equal to the angle whose sine is half the chord length over the radius.

Therefore:

$$\alpha_1 + \alpha_1' = \sin^{-1} \frac{L_1}{2R_1}$$

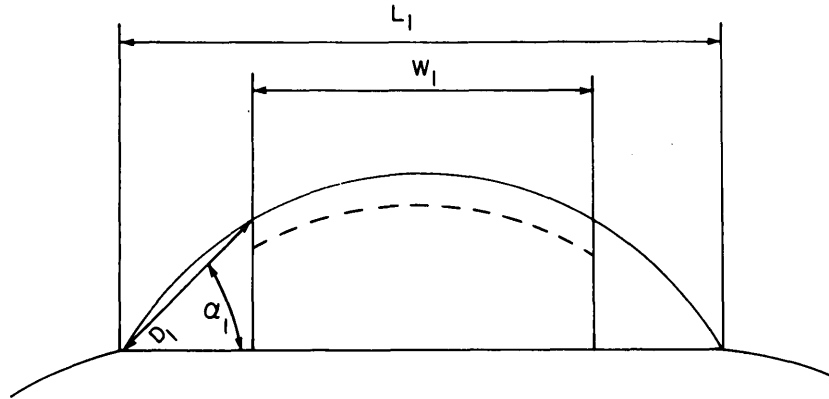


Fig. 2. Cornea in cross-section.

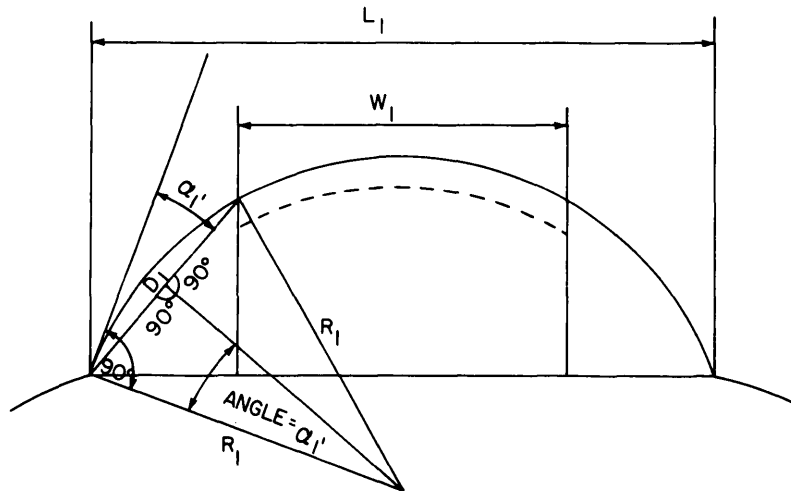


Fig. 3. Cornea in cross-section. R_1 , Peripheral corneal radius of curvature.

This statement is only truly accurate for a perfectly regular corneal radius which the cornea is not, but in the discussion and the use of experimental data this will be an easy way to check for gross errors in technique.

One assumption made is that the peripheral cornea is part of a true circle. This should be a fairly accurate assumption, since D_1 and D_2 are small lengths in reference to R_1 and R_2 and also that $\alpha_1' + \alpha_2'$ are small angles and will tend to cancel each other out.

Our angle α or the change seen at the angle caused by suturing is:

$$\alpha = \text{Cos}^{-1} \left(\frac{L_1 - W_1}{2D_1} \right) + \text{Sin}^{-1} \left(\frac{D_1}{2R_1} \right) - \text{Cos}^{-1} \left(\frac{L_2 - W_1}{2D_2} \right) - \text{Sin}^{-1} \left(\frac{D_2}{2R_2} \right)$$

This change in angle α will have a real effect

on the trabecular area because the cornea is not a thin line but has a definite thickness (C_t). We have calculated what happens externally only. Internally the story is different, and it is internally that the trabecular meshwork is. The anterior surface will act as a fixed surface because our measurements will be taken there. The interior surface will necessarily be compressed, and this compression will have some relationship to our angle α .

We will treat a thin slice of peripheral cornea fixed at the limbus as a rectangle that after suturing rotates through an angle already defined as α (Fig. 4). The triangle so formed is a good approximation of the amount of tissue compressed. The internal dimension of this triangle we will call B.

$$\text{Tan } \alpha = \frac{B}{C_t}$$

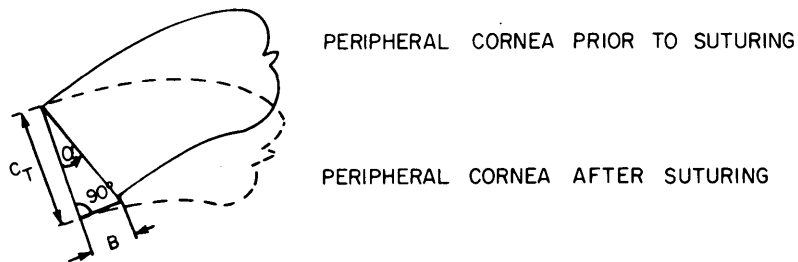


Fig. 4. Corneal cross-section showing the tissue compressed in the angle with keratoplasty.

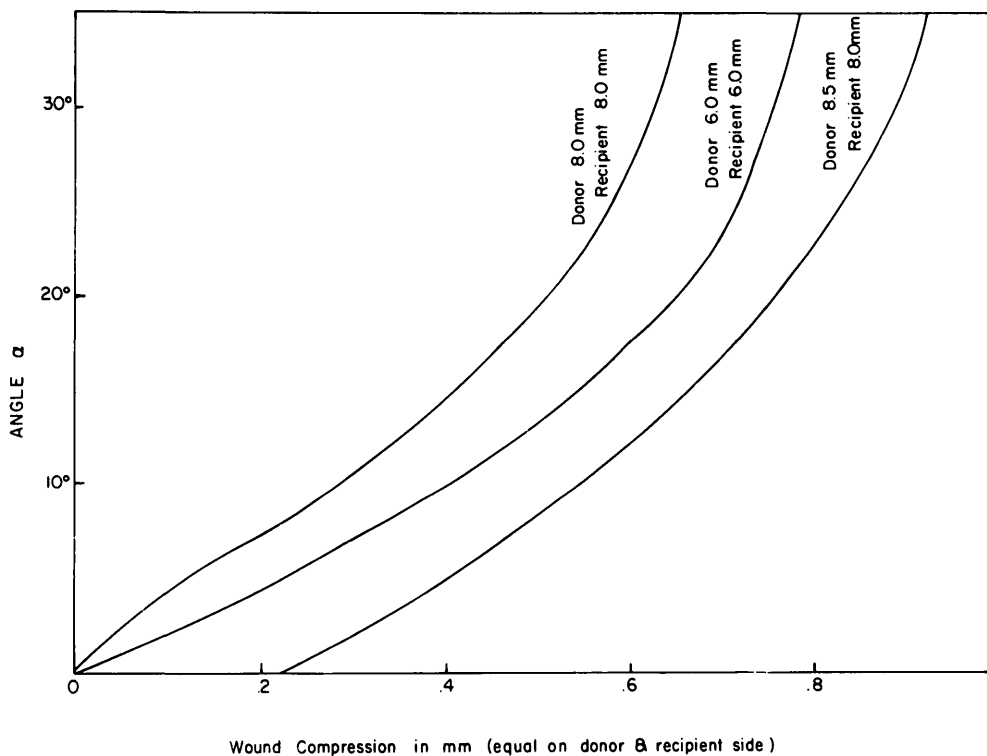


Fig. 5. Relationship of wound compression and angle α (12.00 mm. corneal diameter).

so that

$$B = \tan \alpha C_t$$

The area of this triangle would be:

$$\frac{B \times C_t}{2} = \frac{\tan \alpha C_t^2}{2}$$

How trabecular resistance (R) is related to the dimension of B or the area of the compressed triangle is unclear. Definitive evidence of such a relationship does not exist, but an angle change is probably occurring. We will look at B as having a linear relationship with R and also as having an exponential relationship, which is much more likely. An exponential relationship would say that for the first increment of compression internally we would not expect much change

in R but that later as critical levels of compression and distortion occur, there would be a greater incremental increase in resistance for each increment of tissue distortion. Plotting resistance as the Y axis and internal tissue change (B) as the X axis we have

1. Linear relationship: $R = AL + E = A C_t \tan \alpha + E$
2. Exponential relationship: $\ln R = A C_t \tan \alpha + E$

The Y intercept will have to be the initial resistance (R_0) because no change in the corneal curve will not cause a change in resistance. Constant A, or the slope of our relationship between tissue compression and resistance, is not known at the present time.

Now we can look at changes in intraocular

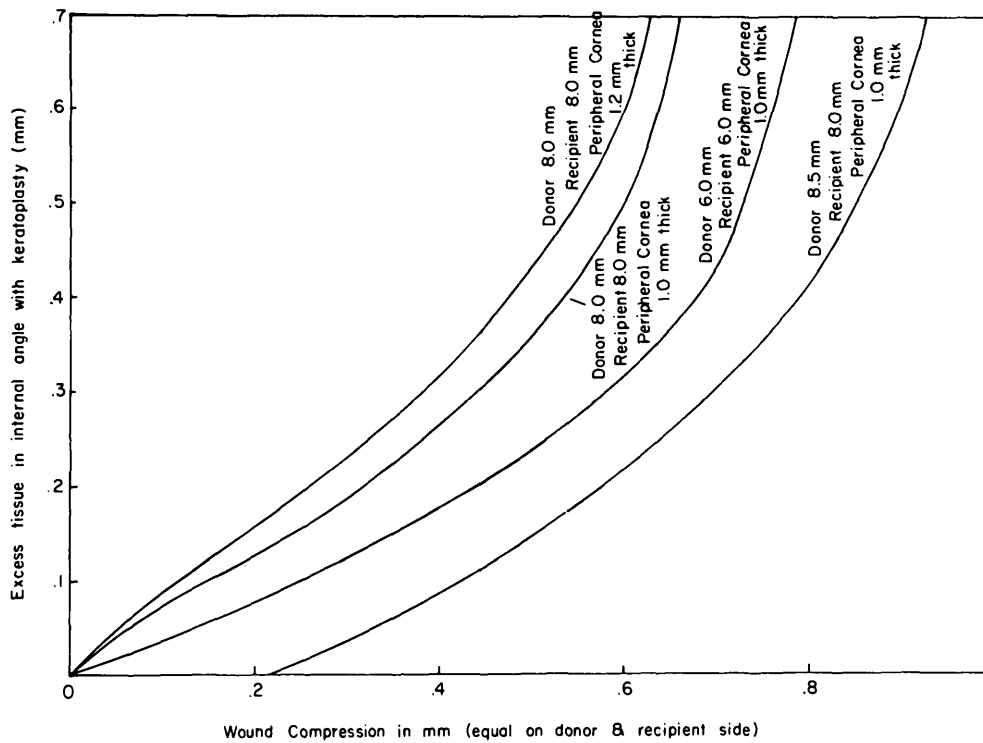


Fig. 6. Relationship of wound compression and internal angle relaxation (12.0 mm. corneal diameter).

pressure as related to angle α in this experimental model.

1. For a linear relationship: $P_o = FAC_t \tan \alpha + P_v$
2. For an exponential relationship: $P_o = Fe^{A\alpha} \tan \alpha R_o + P_v$

Where P_o is intraocular pressure, P_v is scleral venous pressure, and F is aqueous flow.

It should be noted that resistance and intraocular pressure could be related to the area of the compressed triangle. This would change the two equations by simply squaring C_t .

Discussion

This model accurately predicts which factors will increase the angle distortion and which factors might reduce it. Tight suturing and long bits with more compressed tissue, larger trephine sizes, smaller total recipient corneal diameter, and increased peripheral corneal thickness will aggravate the problem. Less tight wounds, smaller trephines, donor corneas larger than the recipient, thinner corneas, and larger over-all corneal diameter, by the

same token, will all tend to alleviate this problem.

The relationship is also interesting in that it is not linear for any of the alleviating factors mentioned except possibly corneal thickness. The ramifications of that statement are great, let us consider a few of them. With the angle plotted as a function of wound compression (Fig. 5), we see a gentle slope becoming quite steep, but the steep range depends on all the other factors mentioned. We have plotted several donor recipient combinations and see that the worst combination is the 8.0 mm. recipient with the same donor size. The 6.0 mm. trephine has a curve that needs greater compression for the same change in angle α , with the 8.5 mm. donor with the 8.0 mm. recipient even better.

When internal angle compression is considered, the same picture occurs but is even more accentuated. Fig. 6 shows this for several combinations of donor-recipient size with one example of a peripheral cor-

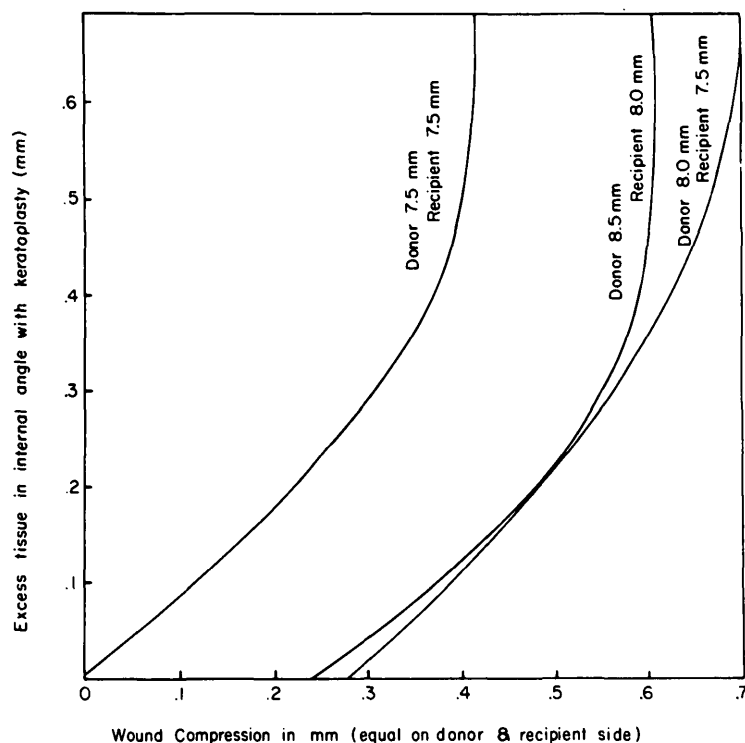


Fig. 7. Relationship of wound compression and internal angle relaxation (11.0 mm. corneal diameter).

nea 0.2 mm. thicker than the others. This moves the curve into an even more critical range, as we have already predicted. The relationship would be even more exaggerated if C_t^2 is the more accurate predictor. Fig. 7 shows these relationships in a host cornea 1.0 mm. smaller in diameter which makes the 7.5-7.5 mm. combination cause more angle change per unit of wound compression than the 8.0-8.0 mm. combination did with a 12 mm. host corneal diameter. Also seen here is the difference that only 0.5 mm. makes, where a 8.5-8.0 mm. combination is decidedly worse than 8.0-7.5 mm. combination in this particular host corneal diameter. So change in corneal diameter and graft size causes an exponential increase in angle distortion that can become critical for small changes with the larger trephines or smaller corneas.

The real question is how the angle distortion causes glaucoma. Two theories can be brought forth. All theories predict a roll of excess compressed tissue in the angle.

In theory one, it is suggested that this excess tissue, through distortion, edema, etc., at a critical level, by itself affects the function of the trabecular meshwork.

There is one major problem with this distortion theory, and that is the phakic eye. Remember that this is really only an aphakic problem, and Zimmerman et al.⁶ have shown in eyebank eye perfusion studies that keratoplasty does not seriously effect the phakic eye. The compression would be equal in phakic as well as aphakic eyes, so that this makes the distortion theory alone less acceptable.

The second theory, offered by Zimmerman et al.,⁶ is the idea of trabecular collapse. They postulate that the trabeculum needs posterior fixation afforded by the ciliary body-lens support system and an anterior support afforded by Descemet's membrane. In aphakia the posterior support is relaxed but not critically. With keratoplasty this loose roll of tissue in same-size transplants would relax the an-

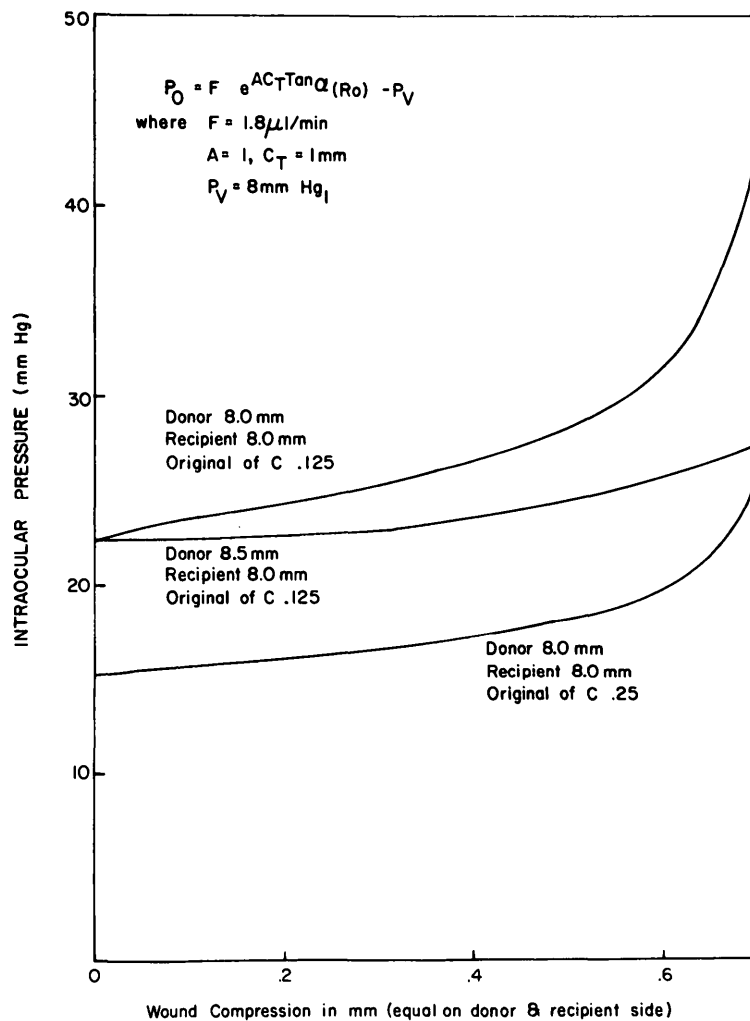


Fig. 8. Hypothetical relationship of wound compression and intraocular pressure.

terior support as well and lead to some trabecular collapse. This theory then would nicely explain the difference seen between phakic and aphakic intraocular pressures after keratoplasty. It would also explain how a larger donor cornea than recipient, which would cause less interior relaxation and crowding, could alleviate trabecular collapse. There is one further piece of supporting evidence for this theory. Through-and-through sutures, which pull on Descemet's membrane by stretching it in the roll of tissue incorporated in the suture bites, could give anterior support even in the face of a large excess of angle tissue. Zimmerman et al.⁸ have shown exactly this

in perfusion studies of eye bank eyes with through-and-through suturing.

The most likely possibility is that some combination of the two theories is occurring. For instance, the compressed tissue could critically affect Schlemm's canal and the trabecular meshwork only when some posterior support as in aphakia is lacking.

Whatever the relationship between our internal angle change and resistance, it is probably not a linear one. If it is linear, a graph plot of resistance as a function of wound compression would look like Figs. 6 or 7. An exponential relationship would look like Fig. 8, in which the slope is very flat until a critical range is reached and

then sudden very sharp rises in resistance and pressure occur. This certainly seems to be the clinical situation. Either you have pressure problems or you don't. If the pressure is over 30 mm. Hg, then it probably will be over 50 mm. Hg. What evidence is there to support this mathematical concept? First of all, the problem is a real one and clinically fits the all-or-nothing phenomenon as predicted. Second, larger donor corneas than recipients have greatly alleviated pressure problems as would be predicted. The most interesting evidence, though, is the fact that an 8.5-8.0 mm. combination did not do nearly as well as a 8.0-7.5 mm. combination in our randomized study (exactly as would be predicted). Laboratory studies are underway to better define the relationship between wound compression and resistance, and we hope to have soon a number for our constant A. With this we could accurately predict the change in baseline resistance for any set of trephine-host corneal sizes.

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