ELEVATED INTRAOCULAR PRESSURE FOLLOWING PENETRATING KERATOPLASTY*

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INTRODUCTION

UNCONTROLLED GLAUCOMA IS A SERIOUS COMPLICATION OF PENETRATING keratoplasty (PK) with adverse consequences for graft survival.[1-4] Polack[5] found no clear grafts at 10 years in glaucomatous eyes, with most failing before the fifth postoperative year. There have been many recent modifications in PK technique, including the use of oversize donor grafts, change in suturing methods, employment of iridoplasty, peripheral anterior synechiolysis (PAS lysis), and viscoelastics intraoperatively, as well as the routine use of steroids, and new glaucoma drugs postoperatively.[6,7] In addition, the use of intraocular lenses (IOLs) has raised questions as to the potential relation of pseudophakia to postoperative pressure elevation.

Many authors have identified a bimodal distribution of intraocular pressure (IOP) rise; the first in the immediate postoperative period (days to weeks) with IOP returning to normal in most cases[8] and a second in the late postoperative period (weeks to months).[9] This study was undertaken to reevaluate potential risk factors influencing chronic elevated IOP in the late postoperative period following PK using current surgical techniques and postoperative care.

MATERIALS AND METHODS

A total of 229 consecutive eyes in 222 patients undergoing PK performed by one surgeon (KRK) between January 1979 and January 1986 were

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studied retrospectively. Patients under the age of 18 years were excluded, as were those with less than 3 months of follow-up. Table I depicts pertinent demographic information. A wide range of diagnostic categories were represented (Table II) with most cases having some form of corneal edema (52.4%), and the majority of these having either aphakic or pseudophakic bullous keratopathy (34.9% overall). Only 20 eyes (8.7%) had previous keratoplasties. Fifty-one percent of the sample had visual acuities of 20/300 or worse. Sixty-three (27.5%) had preexisting glaucoma, and the majority (77.8%) of these cases had controlled IOP (< 24 mm Hg) preoperatively (Table III). Table IV depicts the preoperative lens status of the sample; notably only 18.1% of phakic eyes had glaucoma *vs* 48.5% and 22.2% of aphakic and pseudophakic eyes, respectively.

Data was collected during outpatient office visits at approximately 1 week, 1 month, 3 months, 6 months, 1 year, and the most recent visit recorded thereafter. IOP elevation in the immediate postoperative period was not investigated. The mean follow-up period was 83.3 weeks (standard error of the mean [SEM], 4.2), with 59.1% of the sample having a

TABLE I: DEMOGRAPHIC PROFILE OF THE SAMPLE

Eyes (no.)	229
Patients (no.)	222
Age	
Mean ± SEM	60.5 ± 1.3
Range	18–92
Sex	
Male	123 (53.7%)
Female	106 (46.3%)

TABLE II: PREOPERATIVE CORNEAL DIAGNOSES

	NO.	%	NO.	%
Corneal edema			120	52.4
ABK	40	17.5		
ABK and Fuchs' dystrophy	8	3.5		
PBK	28	12.2		
PBK and Fuchs' dystrophy	4	1.7		
Fuchs' dystrophy (as primary diagnosis)	32	14.0		
Other edema	8	3.5		
Scar			62	27.1
Keratoconus			40	17.5
Other diagnosis			7	3.1
Total			229	100.0

ABK, aphakic bullous keratopathy; PBK, pseudophakic bullous keratopathy.

TABLE III: PREOPERATIVE GLAUCOMA

	NO.	%
Controlled IOP	49	21.4
Elevated IOP	14	6.1
Total preexisting glaucoma	63	27.5

TABLE IV: PREOPERATIVE LENS STATUS

		TOTAL	WITH GLAUCOMA	WITHOUT GLAUCOMA
Phakic		127 (55.5%)	23 (18.1%)	104 (81.9%)
Aphakic		66 (28.8%)	32 (48.5%)	34 (51.5%)
Pseudophakic		36 (15.7%)	8 (22.2%)	28 (77.8%)
PC IOL	n = 3			
AC IOL	n = 29			
Iris clip	n = 4			

follow-up of 1 year or more. IOP was measured by either Goldmann applanation tonometry or pneumotonometry. When tactile tensions were estimated, no numerical values were recorded in our database.

SURGICAL TECHNIQUE

Nearly all cases were performed under modified Van Lint facial block and retrobulbar anesthesia, followed by digital or Honan balloon massage and/or intravenous mannitol (50 ml, 25%). Using a disposable trephine, the corneal graft was punched from the endothelial surface. The host corneal bed was prepared using a motorized trephine (H. Geuder). The donor button was almost always cut 0.5 mm larger than the recipient bed. Variations from this pattern included the use of same size grafts (n = 20), primarily in keratoconus patients, prior to 1983. After July 1982, 0.2 or 0.5 mm oversize grafts were used in all but one case, and therefore, the data is not appropriate for evaluation of graft size in relation to IOP rise.

Where necessary, an anterior vitrectomy was performed with either automated vitrector (n = 60), cellulose sponge (n = 13), pars plana (n = 2), or vitreous aspiration (n = 2) techniques. PAS lysis was undertaken utilizing dry cellulose sponges in those eyes judged to have significant angle closure due to synechia (n = 23). When a large iris defect was present, an iridoplasty was performed using 10-0 nylon or polypropylene suture (n = 62) Thirty-one cases had both vitrectomy and iridoplasty, and 10 cases had all three procedures performed.

In 14 aphakic eyes, flexible open-loop anterior chamber (AC) IOLs were implanted at the time of keratoplasty (13 AC placements, 1 posterior chamber [PC] placement). In 7 pseudophakic eyes (5 closed-loop AC IOL, 2 iris clip IOL) the IOL was removed with out replacement, while in 15 pseudophakic eyes (3 PC IOL, 12 AC IOL) the original IOL was retained. In 26 eyes, a triple procedure (ie, keratoplasty, extracapsular cataract extraction [ECCE], and PC IOL) was performed. One additional eye had an intracapsular cataract extraction (ICCE) with AC IOL placement. One hundred phakic eyes remained phakic and 52 aphakic eyes remained aphakic.

Sodium hyaluronate (Healon) was used in every phakic or pseudophakic case to cover the anterior surface of the crystalline lens or IOL. No attempt was made to remove Healon from the eye. The corneal button was secured in place with eight equidistant interrupted 10-0 nylon sutures, followed by a 16 bite running 10-0 nylon suture.

STATISTICAL ANALYSIS

Postoperative elevated IOP (defined by pressure $\geq$ 24 mm Hg, continuing for more than 2 weeks postoperatively or developing after 2 weeks) was used as the dependent variable. The term, "glaucoma" was deliberately *not* used as inadequate data were available concerning gonioscopy, visual fields, and optic nerve head cupping; this information is often difficult to attain after PK. Independent variables included pertinent demographics, diagnostic categories, and surgical procedures. All categorical data were assessed using chi-square analyses, with Yates correction when appropriate. Continuous variables (eg, age, number of previous surgeries) were analyzed using independent *t*-tests. Preoperative to postoperative change in visual acuity was assessed using Wilcoxon match-pairs signed-ranks test. Because of the large number of analyses performed, a conservative alpha level ($P < .01$) was used so as to reduce the possibility of obtaining significant results by chance alone.

RESULTS

Chronically elevated IOP was found in 77 (33.6%) cases. The pressures in most eyes were ultimately controlled, although 30 (13.1%) had elevated pressures at the time of their last follow-up visit, and 31 (15.6%) cases with normal IOP were on a greater number of glaucoma medications than preoperatively.

The mean length of time from surgery to first IOP elevation for all eyes was 24.3 weeks (SEM, 4.1). Among eyes with preexisting glaucoma, the

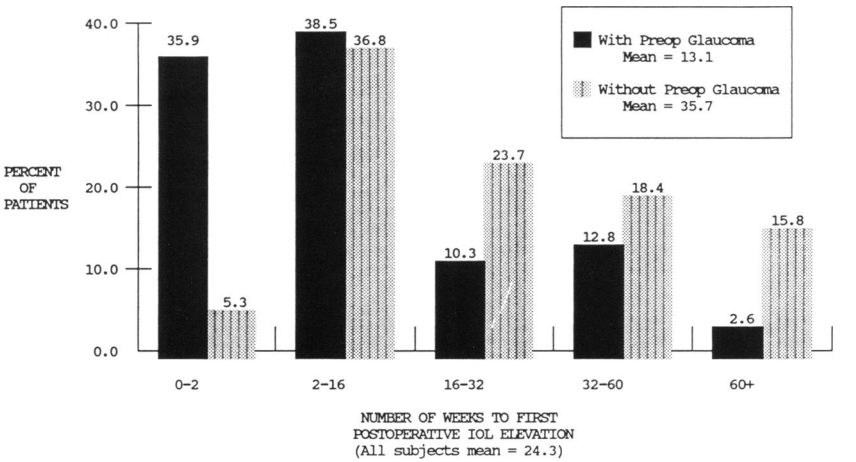

FIGURE 1
Length of time to first IOP elevation for eyes with and without preoperative glaucoma.

mean time to first IOP rise was 13.1 weeks (SEM, 2.9). When excluding cases with preexisting glaucoma, this mean increased to 35.7 weeks (SEM, 7.4). Fig 1 depicts the rates of elevated IOP at different follow-up periods.

Of all 77 eyes with postoperative IOP rise, 39 (50.6%) had preoperative diagnoses of glaucoma. Twenty-one (33.3%) of the 63 cases with preexisting glaucoma required an increase in medications during the follow-up period, while 20 (31.8%) had a decrease in their glaucoma medications. Eighteen (28.6%) of the eyes with preexisting glaucoma were found to have uncontrolled IOP at the time of their last follow-up visit.

Of all subjects without preoperative glaucoma, 38 (22.9%) developed elevated IOP postoperatively. Twelve (7.2%) of these cases had elevated IOP at the time of their last visit. The majority of cases without preoperative glaucoma and with postoperative IOP rise did not require glaucoma medications at the time of their last visit (n = 21; 55.3%). Of those requiring medication, 13 (34.2%) required one glaucoma medication and 4 (10.5%) required two medications.

Five complicated cases with preexisting glaucoma were unable to be controlled medically and required glaucoma surgery; four of these grafts, however, remained clear. Two of these patients had iridocorneal endothelial syndrome, extensive preoperative PAS, and glaucoma; both were filtered, one successfully with a clear graft and normal IOP, the other unsuccessfully with an edematous graft and uncontrolled IOP. A third

TABLE V: RELATIONSHIP BETWEEN PREOPERATIVE GLAUCOMA AND POSTOPERATIVE ELEVATED IOP

	NO PREOPERATIVE GLAUCOMA	PREOPERATIVE GLAUCOMA
Postoperative IOP not elevated	128 (77.1%)	24 (38.1%)
Postoperative IOP elevated	38 (22.9%)	39 (61.9%)
	$P<.001$	

patient had congenital glaucoma with multiple filtering procedures and cryosurgery preoperatively; following PK and subsequent ECCE, cyclocryotherapy resulted in IOP control. A fourth patient with Marfan's syndrome, a failed PK and extensive PAS, with angle-closure glaucoma, required cryosurgery following repeat PK for adequate IOP control. The fifth patient had uncontrolled aphakic glaucoma despite four glaucoma medications prior to PK and required laser trabeculoplasty 1 year postoperatively.

Graft failure, usually due to rejection, occurred in 14 eyes (6.1%), and five repeat keratoplasties were performed. Of those cases (n = 214) with reported visual acuities at most recent follow-up visit, 160(75.1%) showed improvement, as median acuity preoperatively of counting fingers rose postoperatively to 20/100 ($P < .001$). Overall, 23.7% had an acuity of 20/300 or worse postoperatively, compared with 51.1% preoperatively. This significant improvement in visual acuity was found in eyes with and without preexisting glaucoma.

FACTORS ASSOCIATED WITH ELEVATED IOP

The variable most significantly associated with postoperative elevated IOP was a preexisting diagnosis of glaucoma ($P < .001$; Table V). Preexisting elevated IOP (ie, uncontrolled glaucoma), however, was not significantly related to postoperative IOP elevation ($P > .1$). When evaluating all subjects, aphakic eyes were significantly more at risk compared with phakic eyes ($P < .01$), though pseudophakic eyes were not significantly different from either aphakic or pseudophakic eyes (both $P > .1$). Due to the large percentage of aphakic eyes with preoperative glaucoma, the data were reanalyzed to account for this factor. When subjects with preexisting glaucoma were excluded from the analysis, there were no longer any significant between group differences ($P > .1$; Fig 2). Having had previous keratoplasties was not significantly associated with IOP elevation ($P > .1$).

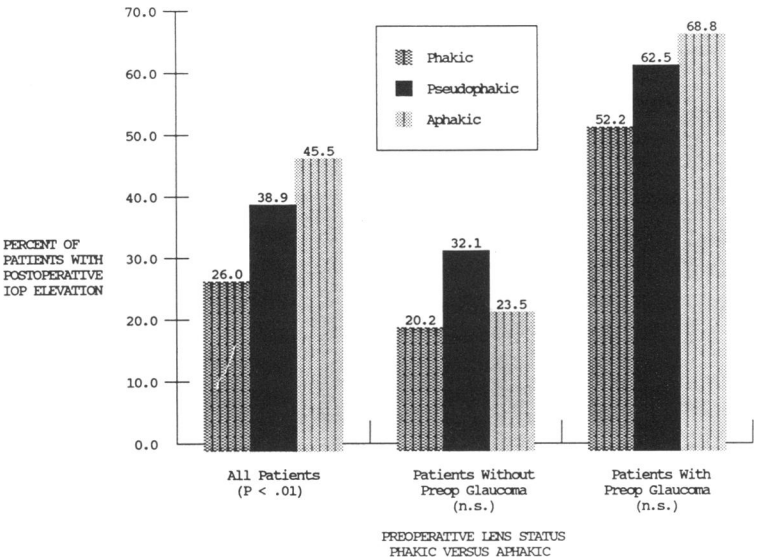

FIGURE 2
Relationshp between preoperative lens status and postoperative IOP elevation. Among all patients: phakic *vs* aphakic, $P < .01$; pseudophakic *vs* either phakic or aphakic, both NS.

When patients with pre-keratoplasty glaucoma were excluded, the relationship between total number of ocular surgeries and postoperative IOP elevation was insignificant ($P > .1$).

Patients with keratoconus had a significantly lower incidence of postoperative IOP risė than patients with either corneal edema ($P < .001$) or scar ($P < .01$); patients with the latter two diagnoses were not significantly different from one another ($P > .1$; Fig 3). Among the specific corneal edema diagnoses, a lower percentage of patients with Fuchs' dystrophy developed postoperative elevated IOP (31.3%) than did patients with either aphakic bullous keratopathy (ABK) or phakic bullous keratopathy (PBK) (50.0%), but this difference was not statistically significant ($P > .05$). The relationship between age and IOP rise approached significance ($P < .05$), with older patients being more at risk than younger patients. When keratoconus patients were excluded from the analysis, however, age was no longer associated ($P > .1$).

Performing cataract surgery during keratoplasty was not significantly associated with postoperative IOP rise ($P > .1$), nor was performing a triple procedure ($P > .1$). Among those cases with IOL implantations at the time

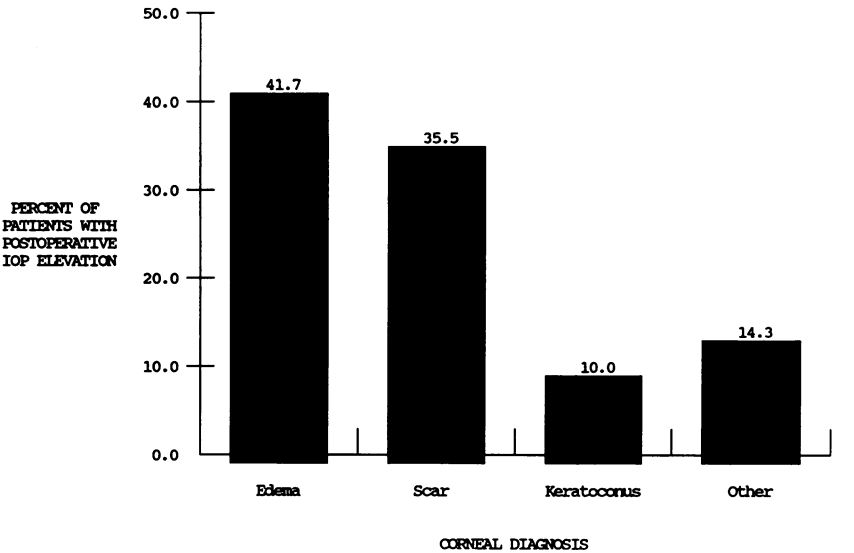

FIGURE 3
Relationship between preoperative corneal diagnoses and postoperative IOP elevation.

TABLE VI: RELATIONSHIP BETWEEN IOL MANIPULATION AND POSTOPERATIVE IOP ELEVATION*

	IOL MANIPULATION		
	RETAINED	EXCHANGED	REMOVED
IOP not elevated	13 (86.7%)	8 (57.1%)	1 (14.3%)
IOP elevated	2 (13.3%)	6 (42.9%)	6 (85.7%)

*Retained *vs* removed, $P < .01$; retained *vs* exchanged, NS; exchanged *vs* removed, NS.

of PK, there was no difference between AC or PC placement with regard to postoperative IOP increase ($P > .1$). Of pseudophakic eyes there was a significant difference between those in which IOLs were retained and those in which IOLs were removed ($P < .01$), with those eyes undergoing IOL removal having a greater percentage of postoperative IOP rise. Eyes in which IOLs were exchanged during PK were not significantly different from either those in which the IOLs were removed or retained (both, $P > .1$; Table VI) Of note is that, of the six eyes with IOP elevation following IOL removal, only one had preoperative glaucoma. Finally, for those eyes which were either aphakic or pseudophakic (preoperatively or during

keratoplasty), neither PAS lysis, vitrectomy, nor iridoplasty were significantly associated with postoperative IOP elevation (all, $P > .1$). It is difficult to ascertain unequivocally, however, the beneficial effects of PAS lysis and iridoplasty, as cases undergoing these anterior segment reconstructive procedures did not demonstrate a significant decrease in postoperative IOP. Specifically, among eyes with PAS preoperatively which underwent PAS lysis, six eyes maintained no PAS postoperatively whereas four eyes again developed PAS. Overall, only 19 eyes in the entire series demonstrated postoperative PAS, and among these, 8 displayed significant PAS with elevated IOP, with only 2 of these requiring glaucoma surgery.

DISCUSSION

The overall incidence of chronically elevated IOP postoperatively was 33.6%, suggesting that IOP rise remains a frequent occurrence following PK. Recent studies of IOP following PK show a range of between 18% and 35% for chronic postoperative IOP elevation.[10-13] When comparing the present study specifically with three recent investigations that also used predominantly oversized grafts, the incidence of elevated IOP is similar to that found by Karesh and Nirankari[11] (23/80; 29%), and by Kirkness and Moshegov[12] (91/305; 30%) but higher than that found by Foulks[13] (91/502; 18%). In Foulks[13] study 15% of the cases had preexisting glaucoma compared with 27.5% in this study; 22% of Foulks' cases had a diagnosis of keratoconus *vs* 18% in this study. The results of the present study suggest both that preexisting glaucoma is significantly associated with postoperative IOP rise, and that keratoconus patients were found to fare significantly better than patients with other corneal diagnoses. It is possible, therefore, that our higher incidence of elevated IOP is due, at least in part, to the higher frequency of preoperative glaucoma and lower proportion of keratoconus patients. In further contrast, however, is the apparently greater severity of glaucoma in Foulks' series with 22 eyes (4.4%) requiring surgical intervention, predominantly cyclocryotherapy, *vs* 5 eyes (2.2%) in this series. Possibly the latter observation is the result of anterior segment reconstructive techniques[6,14] in an effort to reduce postoperative angle closure.

Preexisting glaucoma was the factor most closely associated with postoperative chronic elevated IOP. Over 60% of patients with preoperative glaucoma had an elevated IOP postoperatively. Of those 30 patients whose IOP remained uncontrolled at their last follow-up, over half had preexisting glaucoma and a fourfold greater relative risk (28.6% *vs* 7.2%) than subjects without preexisting glaucoma. All five patients who required

glaucoma surgery also had preexisting glaucoma. These results, though different than those obtained in early studies,[8,15] are consistent with more recent reports which have also found preoperative glaucoma to be a highly significant risk factor for postoperative elevation of IOP.[11-13] Preoperatively uncontrolled IOP in the glaucoma group was not significantly associated with postoperative IOP elevation. This is consistent with work by Olson and Kaufman.[9] It is, therefore, apparently not the immediate preoperative elevated IOP which is critical in subsequent IOP rise, but rather, the predisposition of the eye to glaucoma.

Postoperative elevated IOP was significantly more likely in aphakic eyes than in phakic or pseudophakic eyes, with chronic IOP rise in 46% of aphakic eyes. However, as Fig 2 indicates, when eyes with preoperative glaucoma were excluded, there was no longer an association between aphakia and IOP rise. Since the advent of disparate graft sizing there has been controversy as to whether aphakia remains an independent risk factor for post-keratoplasty IOP elevation. The results of the present study are consistent with reports by Polack[16] and by Goldberg et al[10] in finding that preoperative glaucoma accounts for the high incidence of elevated IOP in aphakic eyes postoperatively. These results differ, however, from Foulks[13] who found that aphakia remained an independent risk factor after controlling for preexisting glaucoma. Further investigation, utilizing a prospective design with a large sample, would be valuable in clarifying the relationship between aphakia and post-keratoplasty glaucoma.

The incidence of postoperative IOP rise in patients with IOLs was intermediate between that of phakic and aphakic patients, though not significantly different from either (Fig 2). When cases with preoperative glaucoma were excluded there were still no significant differences. Schanzlin and colleagues[17] also found no difference between eyes with PBK and ABK with regard to postoperative glaucoma. Polack,[16] on the other hand, found a higher incidence of post-keratoplasty IOP rise in pseudophakic eyes compared with aphakic eyes (25% and 20%, respectively). The results of the present study cannot be directly compared to Polack's, however, because the percentage of patients with preexisting glaucoma was not reported.

Six of seven patients with IOL removal at the time of PK developed increased IOP although only one eye had preoperative glaucoma. Although the numbers of cases are small, this finding raises the question of whether IOL removal during keratoplasty is intrinsically risky or whether the results are confounded by the clinical situations in which the IOLs were removed but not replaced, eg, eyes with significant AC abnormality. The latter seems more likely, as our current surgical convention is to

replace IOLs whenever IOL removal is indicated.[14] Polack,[16] however, found no difference in IOP among cases with IOL retained, exchanged, or removed.

There was no increased risk of postoperative elevated IOP in patients who had a triple procedure (PK plus ECCE and PC-IOL). Though literature on the results and complications of the triple procedure is limited, these results are consistent with previous investigations.[18,19]

Consistent with previous studies,[9,11,20] intraoperative vitrectomy was not a significant factor in this population for patients with either aphakic or pseudophakic eyes preoperatively. Both iridoplasty and PAS lysis were also not associated with postoperative elevated IOP. This is especially noteworthy as one might expect these cases to do significantly worse given that they represent eyes with the most extensive preoperative anterior segment abnormality. Though these procedures were also not associated with decreased incidence of postoperative IOP elevation, the present findings support the belief of Cohen et al[6] and more recently of Waring et al[14] that iridoplasty and PAS lysis are safe procedures and should be used in those difficult situations where they are especially applicable.

Preoperative corneal diagnosis was found to be a significant factor, with keratoconus patients having the lowest incidence of IOP rise and patients with corneal edema or scar having the highest. Among patients with corneal edema diagnoses, those with Fuchs' dystrophy appeared to be at lower risk when compared with patients with ABK and PBK, though this difference was not statistically significant. These findings are similar to Polack[16] and to Wood and colleagues,[8] who found that elevated IOP is rarely a problem for keratoconus patients. Goldberg and colleagues,[10] also found that patients with keratoconus, Fuchs' or stromal dystrophy were all at low risk. Eyes having had previous PKs were not at significantly increased risk of elevated IOP postoperatively. This adds further support to the recommendation that repeat grafting can be performed in the absence of uncontrolled glaucoma.[21]

The average time to the first recorded IOP increase was approximately 6 months. When patients with preoperative glaucoma were excluded from the analysis, the mean time to first IOP rise was over 8 months. This underscores the importance of frequent measurements of IOP over a long follow-up period in all PK patients, particularly those with preoperative glaucoma. The mechanism of this late IOP elevation is not completely understood. Various processes have been suggested including steroid response, iritis, damage to outflow mechanism, loss of angle support following cataract removal combined with PK, and angle closure due to

PAS.[4,8,15] It has also been suggested that glaucomatous eyes already have compromised outflow systems and tolerate the stress of intraocular surgery poorly.[22]

The overall success of PK in this unselected group of patients is indicated by the substantially improved visual acuity as well as the relatively acceptable rate of graft failure (6.1%) during the follow-up period. Nonetheless, there is a high incidence of delayed IOP rise in the postoperative period which appeared late and was often long-lasting. Postoperative elevated IOP remains a particularly significant concern for patients with preexisting glaucoma, and especially for those with both aphakia and glaucoma. Careful and ongoing observation of IOP throughout the prolonged follow-up period is recommended for all PK patients, with prompt attention to IOP treatment as indicated.

SUMMARY

IOP was retrospectively studied in 229 consecutive cases of PK, with a mean follow-up period of 84 weeks. Twenty-seven percent of the cases had preoperative glaucoma, three-fourths of which were medically controlled prior to keratoplasty. Following PK, 34% of the total sample developed sustained elevated IOP but only five (2%) patients required surgical treatment for glaucoma. The mean time from PK to first IOP rise was 24 weeks. Variables which were significantly associated with IOP rise included preoperative glaucoma ($P < .001$), aphakia ($P < .01$), and IOL removal ($P < .01$). When eyes with preoperative diagnoses of glaucoma were excluded from the analysis, aphakia was no longer associated with postoperative IOP rise. Keratoconus patients were at significantly less risk than patients with other corneal diagnoses. Factors not associated with IOP rise included previous PKs, pseudophakia, intraoperative vitrectomy, PAS lysis, iridoplasty, secondary IOL placement, and concomitant cataract extraction with IOL (triple procedure).

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DISCUSSION

Dr M. Bruce Shields. In this retrospective study, Doctor Simmons and coworkers underscore the significance of glaucoma as a postoperative complication of penetrating keratoplasty (PK). They also provide some information that should help surgeons in reducing the frequency of this problem.

The prevention of blindness from glaucoma following PK requires an understanding of many aspects of the condition, including: (1) predisposing factors; (2) mechanisms of intraocular pressure (IOP) elevation; (3) intraoperative and postoperative measures to avoid these mechanisms; and (4) therapeutic approaches when the problem does occur. The present study deals primarily with the first of these considerations, but provides some information regarding the other three.

As with previous studies, preoperative glaucoma was a major predisposing factor. It is surprising, however, that uncontrolled glaucoma before PK, which was present in 14 cases, was not significantly related to postoperative IOP elevation.

Despite this observation, I am sure the authors agree that in general, every effort should be made to bring the glaucoma under control before proceeding with a PK.

Aphakia has also been cited in most studies as a risk factor for post-keratoplasty glaucoma. The fact that it was no longer significantly associated with postoperative IOP elevation in the present study when subjects with preexisting glaucoma were excluded from the analysis does not necessarily mean that it is not involved in the glaucoma mechanism. If all aphakic eyes were eliminated from the analysis, would preoperative glaucoma no longer be a significant predisposing factor? It is quite likely that both preexisting glaucoma and aphakia play a role, to variable degrees, in the presence of glaucoma after PK. As the authors point out, further study is needed regarding the significance of these and other predisposing factors.

The apparent mechanisms of IOP elevation following PK in the present study were not specifically mentioned in the paper. We know from other reports that there are several possible mechanisms. Some of these are common to any anterior segment intraocular operation, such as inflammation, hemorrhage, pigment dispersion, the use of viscoelastic substances, and pupillary block in the early postoperative period and steroid-induced glaucoma and epithelial ingrowth in the later course. In addition, there may be other mechanisms that are more specific to the keratoplasty procedure, such as angle closure and trabecular meshwork collapse in the early phase after surgery and a progressive, chronic angle closure in the last postoperative period. Considering the average time of 6 months to the first recorded IOP rise, it is likely that the late onset mechanisms played the principal role in the present study. It would be of interest to know, however, what the gonioscopic course was in these patients.

As the authors point out, it was not possible in their study to evaluate the efficacy of surgical techniques to avoid these glaucoma mechanisms, since they were used in the majority of cases, with no control group for comparison. Oversized grafts were used in most eyes and iridoplasty was employed in all cases with large iris defects. Previous reports suggest that both of these techniques may reduce the incidence of angle closure, especially in aphakic eyes. It is interesting to note in the present study that neither iridoplasty, vitrectomy nor the lysis of peripheral anterior synechia with dry cellulose sponges were associated with IOP elevation after the surgery, suggesting that these measures may have minimized postoperative glaucoma in the high risk eyes.

Regarding the management of glaucoma following PK, most cases in the present study were controlled medically, with only five eyes requiring surgical intervention. Two of these received filtering surgery, of which one failed, two were controlled with cyclocryotherapy, and one underwent laser trabeculoplasty. While these numbers are too small to allow any conclusions, they are consistent with the general impression in the literature that cyclodestructive surgery is the procedure of choice for uncontrolled post-keratoplasty glaucoma. For the past 2 years, we have been using neodymium:YAG transscleral cyclophotocoagulation as the cyclodestructive procedure for these eyes, especially the aphakic cases. While our preliminary experience with this has been encouraging, further study is needed to determine whether this or other operations will be the surgical procedure of

choice in eyes with uncontrolled glaucoma after PK.

I wish to congratulate Doctor Simmons and her associates on a very thorough study of a large patient population, which provides additional help in our effort to prevent blindness from glaucoma following PK.

DR ROBERT A. HYNDIUK. I, too, enjoyed the paper. I think there is another reason for your excellent results with regard to postoperatively minimizing pressure problems. I'd like to suggest that part of the pressure lowering effect was because of your careful management of vitreous on the iris face and angle, therefore minimizing the progressive anterior synechiae that is seen in many of these patients; and your careful total anterior vitrectomy. Thank you Doctor Kenyon for a valuable contribution to our knowledge in these potentially difficult problems.

DR KENNETH R. KENYON. Thank you Doctor Shields for your generous discussion. We can always count on a Southern gentleman for kind remarks. In closing, I want to reiterate that an elevation following keratoplasty remains a prevalent problem, affecting 30% of cases in our series, so we must be vigalent to diagnose and treat it appropriately. On the other hand, it is encouraging that with current keratoplasty techniques, despite involving IOL exchanges, secondary implantations and/or anterior segment reconstruction, we are doing somewhat better in avoiding the major disasters of post-keratoplasty glaucoma in which progressive peripheral and anterior synechia formation and angle closure are the ultimate complications of corneal transplantation. This observation is also reinforced by the recent paper by Doctor Waring and myself, "Results of anterior segment reconstruction for aphakic and pseudophakic corneal edema" (*Ophthalmology* 1988; 95:836-841.). So, on one hand, we still have a significantly prevalent problem. On the other hand, however, most cases can be successfully managed by medical means with only few cases requiring surgery and with only very few cases remaining uncontrolled. In this regard, I concur with the view of Doctor Hyndiuk in attributing the techniques of anterior segment reconstructive surgery as being most likely responsible for avoiding the major complications of progressive angle closure glaucoma following keratoplasty.