Topographic and keratometric astigmatism up to 1 year following small flap trabeculectomy (microtrabeculectomy)

S A Vernon, H J Zambarakji, F Potgieter, J Evans, P B Chell

Abstract

Aim—To determine the induced corneal astigmatism by measuring the changes in manual keratometry and computerised corneal videokeratography up to 1 year following small flap trabeculectomy (microtrabeculectomy).

Method—A prospective study of a case series of small flap trabeculectomy procedures performed at the 90 degree meridian on 16 eyes of 16 patients, all followed to 1 year postoperatively. Changes in manual keratometry and computerised videokeratography (Eyesys) readings were analysed by vector analysis and vector decomposition techniques.

Results—By vector analysis, the mean surgically induced refractive change (SIRC) cylinder power vectors induced at 1, 3, 6, and 12 months as measured by manual keratometry were 0.68, 0.38, 0.52, and 0.55 dioptres, and by keratography 0.75, 0.66, 0.59, and 0.64 dioptres. Vector decomposition on the induced vector cylinders on manual keratometry resulted in a “with the rule” mean vector of 0.52 and 0.22 dioptres at 1 and 3 months and an “against the rule” mean vector of 0.16 and 0.16 dioptres at the same time points (p=0.03 and 0.28 respectively). Vector decomposition at 6 and 12 months revealed no significant with the rule change induced. Similar analysis on the videokeratography results revealed significant induced with the rule astigmatism until 3 months, but not at 6 and 12 months postoperatively.

Conclusion—Small flap trabeculectomy (microtrabeculectomy) produces smaller changes in corneal curvature that resolve sooner than previous reports of larger flap techniques. (Br J Ophthalmol 1999;83:779–782)

Trabeculectomy, since its introduction in 1968, has become the gold standard surgical procedure for progressive open angle glaucoma.1 In the 1990s a number of authors have studied the refractive changes induced in the cornea as a result of the procedure.2,3 Several surgical modifications have been described,4 each of which may theoretically influence the changes noted in the cornea following surgery. In 1995 we reported the intraocular pressure results following microtrabeculectomy, a small flap modification of the original design which we have utilised routinely since 1991.5 The scleral trapdoor at 2 × 2 mm is approximately one sixth of the area of the Cairns’ procedure, and the 0.75 × 0.75 mm internal opening is just one seventh of the area of the original osteum. As a smaller sized operation theoretically should result in less surgical trauma, we performed a prospective study to investigate the changes in corneal curvature following microtrabeculectomy. We utilised vector analysis and vector decomposition techniques to determine the “with the rule” and “against the rule” changes induced in the cornea.
TX, USA) running software version 3.0, was performed before and at 1, 3, 6, and 12 months postoperatively. Manual keratometry was carried out by an experienced optometrist (JE) with the same calibrated keratometer, the operator being masked with respect to previous data. Corneal videokeratoscopy was performed by one of two operators (HJZ and FP) within 1 hour of keratometry with care taken only to accept good quality images. Keratometric measurements of both types were made before instillation of eyedrops where possible. The Eyesys operator was also masked to previous data when performing the scans. The simulated “k” power and meridian readings produced automatically by the machine were then used in the data analysis (these utilise all data points within the central 3 mm rings on the videokeratoscope to calculate the readings displayed on the screen).

Vector analysis\(^{12}\) was performed on the data using a computerised method of calculating the surgically induced refractive change (SIRC) for each eye at every time point postoperatively. All changes were compared with the preoperative data set and expressed in terms of negative cylinders. SIRC is based on the theory that the combination of two crossed spherocylinders produces a third spherocylinder.\(^{12}\) This provides a vector of induced cylinder for each eye at each time point. In order to analyse group changes, we also performed vector decomposition\(^{13}\) which gave us a mathematical expression of the change in astigmatism “with the rule” (WTR) or “against the rule” (ATR) (WTR astigmatism is defined as corneal steepening in the vertical meridian corresponding to a negative induced cylinder at 180 degrees and ATR being the reverse). We used simple \((m)\sin^2(x)\) and \((m)\cos^2(x)\) functions, multiplied by the scalar magnitude, in this case the astigmatic magnitude in dioptres (m), to decompose the astigmatism by algebraic vector theorem at meridian (x). This results in magnitude of change WTR and ATR for each eye. Wilcoxon signed rank analysis was performed on the collective WTR and ATR data at each time point to determine whether there was a statistically significant induction of astigmatism in either axis with respect to the other at each time point.

In addition to the above we performed a reproducibility analysis on 10 measurements of a single eye which had not undergone surgery. After each scan, the patient was asked to sit back away from the Eyesys machine to simulate a separate examination episode. The first scan acted as the baseline and all other scans were compared with it, resulting in nine “induced vectors” to analyse and decompose. These data thus provided an Eyesys “machine and operator measurement error” for WTR and ATR.

**Results**

In all, 22 eyes of 19 patients were entered into the study. Two eyes from two patients failed to complete all the data points, because of an administrative error with one and with the other the patient died. In order to reduce bias from including two eyes of a patient in the analysis, the results of 16 eyes from 16 patients are presented, the first eye to be operated on being selected when both eyes were originally enrolled in the study. The mean (SD) age of the patients was 68.2 (8.9) years at surgery and there were four women and 12 men. All patients were white Europeans. The mean (SD) preoperative intraocular pressure (IOP) on medications was 26.4 (7.0) mm Hg and the mean IOP at the 1 year review was 13.6 (3.8) mm Hg (p=0.0005 by Wilcoxon signed rank test). None was receiving antiglaucoma medications at 1 year following surgery and all had intraocular pressures of 21 mm Hg or less.

Mean vector powers for SIRCs for the 16 eyes at each time point are given in Table 1. Figures 1 and 2 depict the SIRC vectors for eyes with >0.25 D induced cylinder at 3 months postoperatively, Figure 1 by manual keratometry and Figure 2 by computerised corneal videokeratoscopy.

Tables 2 and 3 indicate the mean decomposed vectors at each time point, Table 2 displaying results from manual keratometry and Table 3 from the Eyesys system. Statistical p values relate to comparison of WTR and ATR induced cylinders at each time point by the Wilcoxon signed rank test.
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Table 2 Mean (SD) decomposed vectors measured by manual keratometry for 16 eyes following microtrabeculectomy

<table>
<thead>
<tr>
<th>Month</th>
<th>WTR (D)</th>
<th>ATR (D)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.52 (0.29)</td>
<td>0.16 (0.27)</td>
<td>0.026</td>
</tr>
<tr>
<td>3</td>
<td>0.22 (0.25)</td>
<td>0.16 (0.31)</td>
<td>0.28</td>
</tr>
<tr>
<td>6</td>
<td>0.23 (0.22)</td>
<td>0.29 (0.39)</td>
<td>0.50</td>
</tr>
<tr>
<td>12</td>
<td>0.24 (0.27)</td>
<td>0.31 (0.31)</td>
<td>0.33</td>
</tr>
</tbody>
</table>

WTR = with the rule astigmatism (vertical steepening) measured in dioptres; ATR = against the rule astigmatism (vertical flattening) measured in dioptres. P = Wilcoxon signed rank p value.

Table 3 Mean (SD) decomposed vectors measured by topographic keratometry (Eyesys) for 16 eyes following microtrabeculectomy

<table>
<thead>
<tr>
<th>Month</th>
<th>WTR (D)</th>
<th>ATR (D)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.64 (0.49)</td>
<td>0.12 (0.13)</td>
<td>0.003</td>
</tr>
<tr>
<td>3</td>
<td>0.57 (0.67)</td>
<td>0.09 (0.12)</td>
<td>0.006</td>
</tr>
<tr>
<td>6</td>
<td>0.42 (0.37)</td>
<td>0.17 (0.21)</td>
<td>0.063</td>
</tr>
<tr>
<td>12</td>
<td>0.34 (0.37)</td>
<td>0.30 (0.32)</td>
<td>0.80</td>
</tr>
</tbody>
</table>

WTR = with the rule astigmatism (vertical steepening) measured in dioptres; ATR = against the rule astigmatism (vertical flattening) measured in dioptres. P = Wilcoxon signed rank p value.

For the reproducibility study with the Eyesys machine, the mean WTR and ATR astigmatism were 0.093 D and 0.074 D respectively (p=0.26). The coefficient of reproducibility was calculated at 1.02%.

Discussion

Small flap trabeculectomy (microtrabeculectomy) has been shown to control IOP at least as well as previous reports of larger flap procedures in the medium to long term. Altered visual function induced by changes in corneal curvature following filtration surgery may be distressing to the patient, particularly when changes are marked and continue beyond the first few postoperative months.

The first study to report the changes in corneal curvature following trabeculectomy was by Hugkulstone. He used an automated keratometer which recorded a mean induced with the rule astigmatism of 1 D at 7 weeks following a traditional sized procedure (5 × 5 mm scleral trap door) in 10 eyes. Cunliffe et al in a study on 16 eyes with manual keratometry utilising a slightly smaller scleral trap door (5 × 3 mm), found a significant WTR astigmatism up to 2 months but not at 10 months. Unfortunately, there were no intermediate analysis time points in this study.

When measured by manual keratometry, the astigmatism induced in our study was significantly greater WTR than ATR only at the 1 month data point and the mean WTR had decayed to only 0.22 D at 3 months (Table 2). In order to provide some comparison with previous reports, we analysed the raw keratometric results provided in Rosen and others’ paper. By vector analysis, the mean vector power induced at 3 months in Rosen and others’ study was 1.24 D as opposed to 0.38 in our study (p=0.001 by Mann–Whitney U). Vector decomposition resulted in a WTR mean vector of 1.18 D in comparison with our 0.22 D (p<0.0001). It is interesting to note that Rosen used a 3 × 2 mm scleral trap door and, in contrast with our study, used laser suture lysis in six of the eight eyes reported.

More recently, Hong et al using vector analysis, calculated the induced astigmatism along the 180 degree meridian to be a mean of 1.24 D with the rule in a cohort of 14 eyes undergoing a 4 × 4 mm scleral trap door trabeculectomy. Their data are, unfortunately, not solely from keratometry as 50% of their eyes had data entered from refraction.

Changes in corneal curvature on topographic simulated keratometry following trabeculectomy have been found to be larger than those from manual keratometry. Rosen et al found that five of the eight eyes studied developed between 1.5 and 2.5 D induced astigmatism at 3 months postoperatively when measured with the topographic modelling system (TMS) (Computed Anatomy, New York, USA). Zarnowski et al found, using the Eyesys system, a mean SIRC of 0.4 D at 3 months in their unenhanced “conventional trabeculectomy” group, almost double our mean value of 0.22 D.

Claridge et al found, using the same TMS system as Rosen et al, that they could identify three subgroups of eyes at 1 and 3 months postoperatively. The largest group had an induced superior steepening of the cornea resulting in a mean WTR astigmatism of about 1D (measured by polar values) which persisted to 1 year following surgery. These results were on eyes which had had a 4 × 3 mm scleral trap door with two 10/0 nylon sutures. Dietze et al using the Eyesys system on a mixed group of 13 eyes, used a simple subtraction of the data readout values to estimate the net astigmatic change. We consider the use of vector analysis with decomposition as performed in our and Zarnowski’s study to be a superior method of determining change. Unfortunately details of the “standardised trabeculectomy” are not given in Dietze’s study and four of the 13 eyes had suture lysis within the first 2 weeks following surgery. They were, however, able to determine that the majority of the change following surgery is found in the superior semimeridian, as would be expected. Unfortunately many of our Eyesys scans were unretrievable following completion of the study making direct comparison of semi-meridian changes not possible (simulated “k” values were recorded on the data sheet at the time of scanning).

A number of suggestions have been put forward to explain the WTR astigmatism induced by the trabeculectomy procedure. Hugkulstone (and later Dietze et al) mentioned the possibility of tight sutures and suggested a “posteriorly placed wound gape” from the internal sclerostomy as the cause. Cunliffe et al suggested that the cautery was the main factor as the induced astigmatism appeared to be greater when excessive cautery was used in one patient. Hong’s group, as they found a...
significantly lower induced astigmatism in a group of eyes undergoing microtrabeculectomy with mitomycin C, considered the wound healing process to be active in inducing the with the rule astigmatism. Which of these possible explanations apply to microtrabeculectomy? It is noteworthy that we did not perform suture lysis or any other form of manipulation on any of our cases. As our sutures are nearer the limbus than in a full sized procedure, an equal tension would tend to induce greater WTR astigmatism. As the reverse was found in our study, we consider sutures as a mechanism of astigmatism induction to be unlikely to play a significant role in our hands (unless we routinely suture at less tension than other surgeons). Although the smaller internal osteum may play a role in the reduced astigmatism observed following microtrabeculectomy, we consider the reduced cautery necessary when operating in a smaller surgical field to be of more importance (we have occasionally observed corneal shrinkage changes with the operating microscope where more cautery has been required when operating on eyes that have had previous surgery). The small size of the microtrabeculectomy usually allows the surgeon to position the procedure in between aqueous veins visible before commencing surgery, thereby reducing the need for excessive cautery (it is not of note, however, that avoidance of vascular structures was not practised in this study owing to the restrictions of the protocol). The smaller surgical area as far as the sclera is concerned would also tend to reduce any astigmatism produced as a result of the healing process if Hong’s theory is correct.

Whichever mechanisms are involved in the production of postoperative corneal changes, it would appear that the combination of a small scleral trap door and a controlled internal sclerostomy has the capacity of inducing an acceptably low degree of central corneal astigmatism in most eyes. Keratometry and the use of simulated k values on videokeratoscopy do, however, simplify the picture of irregular astigmatism which is often the result of trabeculectomy surgery. The use of a small flap should, of course, help to minimise the torsion which can be produced in the superior cornea from horizontal flap misalignment. Although not formally reported in this paper, we have noted rapid visual recovery in patients undergoing microtrabeculectomy, with the continued use of previously prescribed spectacles being the norm. This would be consistent with the observed rapid return to insignificance in the WTR/ATR astigmatism induced by the procedure (Tables 2 and 3).

We believe that the results we have reported in this paper are very encouraging and should be reproducible by other glaucoma surgeons practising similar techniques. The low levels of corneal astigmatism induced by microtrabeculectomy add to the other potential advantages of the procedure previously reported,13–16 and justify a randomised trial of all aspects of postoperative changes following trabeculectomy by independent surgeons who are practised in both large and smaller flap techniques.

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