Effect of Infusion Air Pressure on Visual Field Defects After Macular Hole Surgery

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• PURPOSE: **A defect in the visual field is one of the serious complications of macular hole surgery. We investigated the relationship between the occurrence of visual field defect and the location of infusion cannula and air pressure during fluid–air exchange.**

• METHODS: **We studied 100 eyes from 90 patients with macular holes. All patients underwent preoperative and postoperative visual field testing. Vitreous surgery was performed in a standard three-port fashion, with surgically induced posterior vitreous detachment, fluid–air exchange, and sulfur hexafluoride gas injection. We analyzed differences in surgical methods in three groups. In group A, the infusion cannula was placed inferotemporally, and the air pressure was set at 50 mm Hg. In group B, the infusion cannula was placed inferonasally, and the air pressure was set at 50 mm Hg. In group C, the infusion cannula was placed inferotemporally, and the air pressure was set at 30 mm Hg.**

• RESULTS: **Eighteen eyes (18%) showed visual field defects after vitrectomy. The defect was always located contralateral to the infusion cannula. There was no statistically significant difference in the incidence of visual field defects in groups A and B. Decreased air pressure reduced the occurrence of visual field defects significantly (24% in group A versus 4% in group C, $P = .011$).**

• CONCLUSIONS: **The location of the visual field defect correlated with the location of the infusion cannula. The incidence of this visual field defect was influenced strongly by the infusion air pressure. The visual field defect may be caused by the mechanical damage of air infusion. (Am J Ophthalmol 2000;130:611–616.**

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VITRECTOMY HAS BEEN USED SUCCESSFULLY FOR treatment of idiopathic macular holes and such other diseases as diabetic retinopathy, retinal detachment, and epiretinal membrane of the macula. In the treatment of idiopathic macular holes, especially, although several studies have shown vitrectomy to be effective in producing significant improvement in visual acuity, a variety of complications has been reported during and after surgery, including retinal breaks,[1] rhegmatogenous retinal detachment,[2,3] enlarging of the hole, late reopening,[2,4] cataract formation,[5] and peripheral visual field defect.[6–13]

Visual field defects after vitrectomy, which were first reported in 1995,[6] were relatively unique for macular hole surgery. The incidence varied from 7%[13] to 71%[8] among institutions. Although recent studies have shown an association of this complication with fluid–air exchange,[14,15] its mechanism is still unclear. Furthermore, there is no report in humans regarding the effects of infusion air pressure on the occurrence of this complication.

We report herein the results of a prospective study of visual field defects in 100 eyes of 90 patients operated on for idiopathic macular holes. We addressed two issues: the relationship of the incidence of visual field defects with the location of the infusion cannula and the association of incidence of visual field defects on air pressure during fluid–air exchange.

METHODS

PROSPECTIVELY, 100 EYES OF 90 CONSECUTIVE PATIENTS who underwent surgery for idiopathic macular holes and preoperative and postoperative visual field testing between January 1996 and May 1999 were included in this study.

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Informed consent was obtained from all of the patients, and the study was carried out in accordance with the guidelines of the human studies committee of Kumamoto University. All patients underwent a preoperative examination that included visual acuity measurement with best correction, applanation tonometry, fundus examination with contact lenses, and Goldmann peripheral kinetic visual field examination. Each idiopathic macular hole was graded according to the classification proposed by Gass.[16]

Surgeries were performed with a standard three-port pars plana vitrectomy under local anesthesia. Briefly, after a core vitrectomy, for stages 2 and 3 macular holes, posterior vitreous detachment was surgically induced with a brush backflush needle using active aspiration around the disk and then extended outward to the equator. Vitrectomy was completed, with the vitreous gel removed as far out toward the periphery as possible. For stage 4 macular holes, when an epiretinal membrane was present around the hole, it was peeled off after vitrectomy. Fluid–air exchange was performed by passive aspiration using a brush backflush needle over the disk with humidified air, as described previously.[17] After the fluid–air exchange, side ports were immediately plugged with scleral plugs. The side ports and the infusion port were then sutured and closed with three bites of running shoelace suture. At the end of surgery, 1.0 ml of pure sulfur hexafluoride gas was slowly injected into the vitreous cavity with a 30-gauge needle to a final concentration of 20%. Cataract surgery was combined with phacoemulsification and aspiration and with intraocular lens implantation in 82 eyes.

To analyze the relationship between the location of the infusion cannula with the occurrence and location of visual field defects, the infusion cannula was placed inferotemporally for 42 eyes (group A) or inferonasally for 12 eyes (group B). Infusion air pressure during fluid–air exchange was set at 50 mm Hg in both groups. The value of 50 mm Hg for fluid–air exchange had been used during all vitrectomy procedures in our hospital.

To test the association of the infusion air-pressure and the incidence of visual field defect, the air-pressure was set at 30 mm Hg with the cannula placed inferotemporally for the remaining 46 eyes (group C).

Patients were instructed to maintain a face-down position as much as possible for 14 days. Slit-lamp examination and applanation tonometry were performed every day until the patient was discharged. Postoperative visual field testing was performed when there was no residual gas in the vitreous cavity. For each patient, preoperative and postoperative visual fields were reviewed by three of us (Drs Hirata, Yonemura, and Hasumura). Changes were considered significant only when the V4 and I4 isopters were indented by the defect.

The following data were collected to allow statistical comparison between eyes with visual field defects and those without and to find differences in incidence of visual field defects associated with different surgical procedures: age, sex, classification, operation time, preoperative and postoperative intraocular pressure, and incidence of visual field defects. Postoperative data collected and analyzed were only for initial surgeries, although four eyes required repeated vitrectomy or gas tamponade. Data were tested by analysis of variance and the Mann-Whitney *U* test for comparison of the patients' clinical data and by Fisher exact probability test for detecting the difference in the incidence of visual field defects. Results were considered significant when *P* was less than .05.

RESULTS

THE CLINICAL FEATURES OF ALL PATIENTS ARE LISTED IN Table 1. At the time of surgery, the macular hole staging of the 100 eyes was as follows: 51 eyes were stage 2, 36 were stage 3, and 13 were stage 4. After the surgery the macular hole ultimately was closed in 90 eyes (90%). Twenty-five eyes (25%) had an intraocular pressure of 20 mm Hg or more 1 day after surgery, but none had an intraocular pressure more than 20 mm Hg after the first day. Seventy-seven of the 90 eyes (85.5%) in which the macular hole was anatomically closed had an improvement in visual acuity of 2 or more lines during a mean follow-up of 16.5 months. In all 100 eyes, postoperative fundus examination was performed when residual gas in the vitreous cavity disappeared, showing no detectable retinal abnormality in the retina corresponding to the visual field defect.

Of the 100 eyes, 18 eyes (18%) had postoperative peripheral visual field defects that were not present preoperatively (Table 1). Patients with 12 of the 18 eyes were women and ranged in age from 59 to 73 years (mean, 65.2 years). The visual field defect, when present, was evident just after intravitreous gas disappeared, and no progress was detected during the period of follow-up. The clinical features of the patients with visual field defects were compared with the features of patients without visual field defects, and the mean age, sex, macular hole stage, operation time, preoperative and postoperative intraocular pressure, and rate of anatomic closure were similar in the two groups (Table 1).

Forty-two eyes underwent vitrectomy with the infusion cannula placed inferotemporally and infusion air pressure set at 50 mm Hg (group A). Ten eyes of the group (24%) showed visual field defects after surgery (Table 2). The defect, a focal and a triangle-shaped depression, was always observed at a lower temporal quadrant (Figure 1, A and B).

Twelve eyes underwent vitrectomy with the infusion cannula placed inferonasally with infusion air pressure set at 50 mm Hg (group B). Six eyes of the group (50%) showed visual field defects after surgery. In contrast to the defects found in group A, the defects in group B were located at the lower nasal quadrant (Table 2; Figure 1, C and D). The defects in group B resembled those in group A in shape, but some of them exhibited nasal steplike depressions and af-

TABLE 1. Clinical Characteristics of Patients With and Without Postoperative Visual Field Defect

	Visual Field Defects		
	+ (n = 18 eyes)	− (n = 82 eyes)	Total (n = 100 eyes)
Age (years)	65.2 ± 4.2	66.7 ± 5.8	66.4 ± 5.5
Sex			
Male	6 (33%)	3 (35%)	35 (35%)
Female	12 (67%)	9 (65%)	65 (65%)
Stage			
2	7 (39%)	44 (54%)	51 (51%)
3	10 (55%)	26 (32%)	36 (36%)
4	1 (6%)	12 (15%)	13 (13%)
Operation time (minutes)	67.3 ± 9.7	66.2 ± 13.5	66.4 ± 12.9
IOP (mm Hg)			
Preoperative	12.7 ± 2.7	13.1 ± 3.0	13.0 ± 2.9
Postoperative (at 24 hours)	18.9 ± 5.8	18.6 ± 4.6	18.7 ± 4.8
Anatomic closure	16 (89%)	74 (90%)	90 (90%)

IOP = intraocular pressure.
Values are means ± SD.

TABLE 2. Clinical Characteristics of Patient Groups

	Group A (n = 42 eyes)	Group B (n = 12 eyes)	Group C (n = 46 eyes)
Age (years)	66.6 ± 4.8	65.2 ± 4.5	66.6 ± 6.4
Sex			
Male	16 (38%)	3 (25%)	16 (35%)
Female	26 (62%)	9 (75%)	30 (65%)
Stage			
2	19 (44%)	8 (67%)	24 (52%)
3	18 (50%)	3 (25%)	15 (33%)
4	5 (6%)	1 (8%)	7 (15%)
Operation time (minutes)	65.2 ± 15.5	68.0 ± 11.1	67.0 ± 10.7
IOP (mm Hg)			
Preoperative	13.4 ± 3.1	12.9 ± 4.1	12.6 ± 2.3
Postoperative (at 24 hrs)	17.9 ± 4.8	21.1 ± 7.4	18.4 ± 3.9
Anatomic closure	36 (86%)	11 (92%)	43 (93%)
Visual field defect	10 (23.8%)	6 (50.0%)	2 (4.3%)*

IOP = intraocular pressure.
Values are means ± SD.
*Significant difference between groups A and C, $P < .05$, Fisher exact probability test.

fected up to the central 20 degrees of the field of vision (Figure 1, C). There was no statistical significance in the effect of the location of the infusion cannula on the incidence of visual field defects (24% in group A versus 50% in group B, $P = .16$).

Forty-six eyes underwent vitrectomy with the infusion cannula placed inferotemporally and infusion air pressure set at 30 mm Hg (group C). Only two eyes showed postoperative visual field defects (Table 2), and they were located at the lower temporal quadrant (Figure 1, E and F). The shape and extent of the defects were similar to those in group A. However, the incidence of visual field defects in group C was significantly lower than that in group A ($P = .011$, Fisher exact probability test), whereas there was no statistical significance in the differences in clinical data of the patients in these two groups.

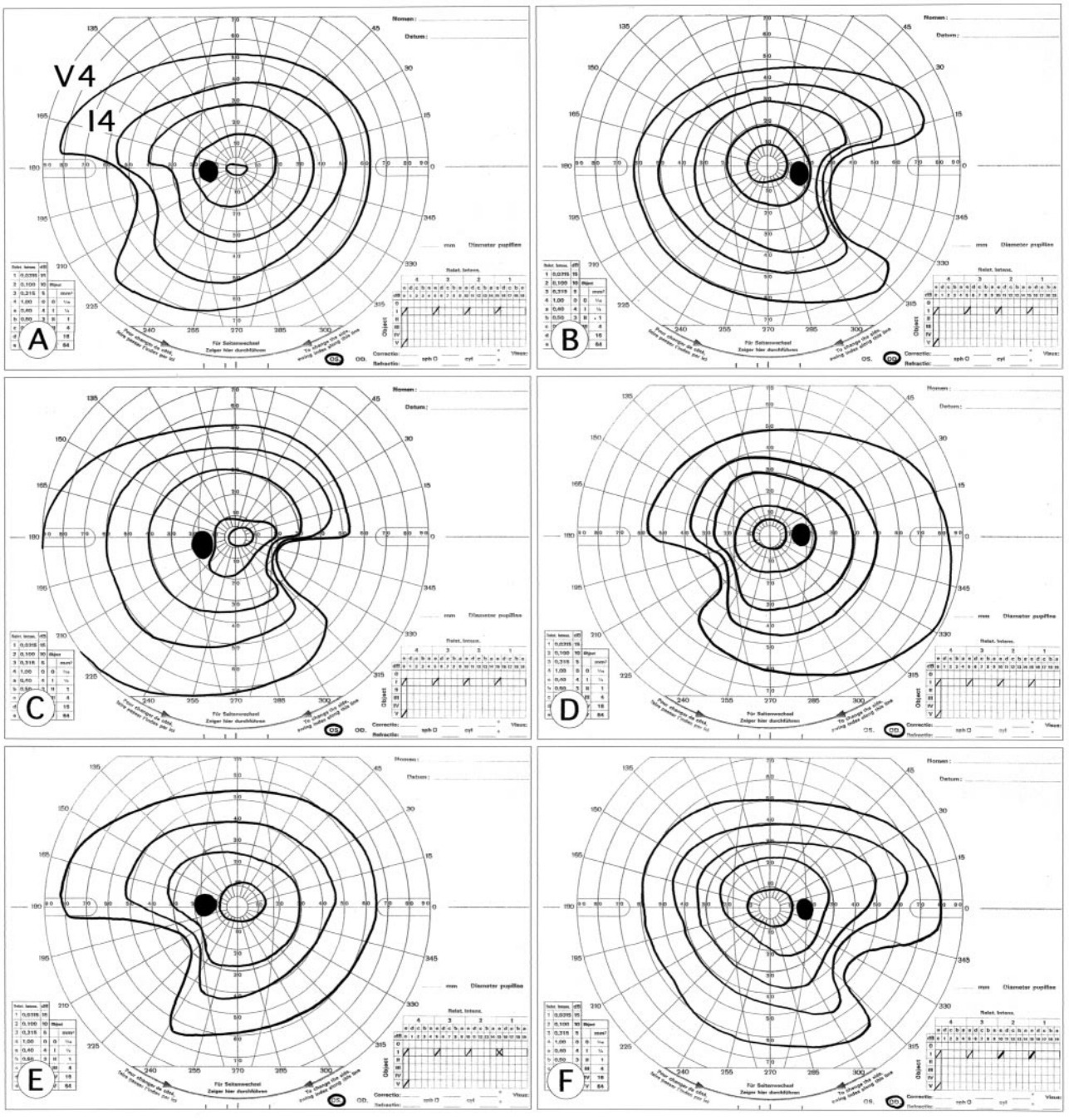

FIGURE 1. Goldmann perimetry of the postoperative visual field defects after macular hole surgery. (A and B) Visual field defects in group A. (C and D) Visual field defects in group B. (E and F) Visual field defects in group C.

DISCUSSION

IN THIS STUDY, 18 OF 100 EYES (18%) SHOWED A VISUAL field defect postoperatively. All of these defects were located at the opposite side from the infusion cannula, whether the infusion cannula was placed temporally or nasally. Also, to clarify the influence of infusion air pressure on the incidence of visual field defects, infusion air pressure during surgery was changed from 50 to 30 mm Hg. The result demonstrated that the decreasing air pressure during fluid–air exchange resulted in a significant decrease in the incidence of visual field defects from 24% to 4%.

Visual field defects after vitrectomy can be seen after any surgery involving fluid–air exchange, including macular hole, diabetic retinopathy, and subretinal surgery. Since the first description of a visual field defect after vitrectomy,[6] numerous papers have proposed hypotheses to

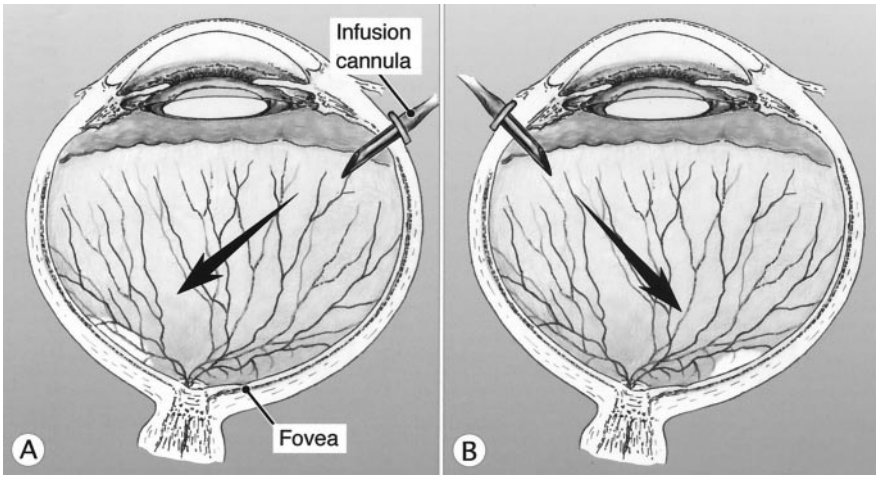

FIGURE 2. Schematic diagrams showing the relationship of the position of the infusion cannula and corresponding retinal damage. (A) Infusion air from the infusion cannula placed inferotemporally can cause nasal retinal damage resulting in a temporal visual field defect. (B) Concomitantly, infusion air from the inferonasally placed cannula can cause temporal retinal damage resulting in a nasal visual field defect.

explain the mechanism of this complication, such as mechanical trauma to the optic nerve head during surgery,[6] damage to the optic disk induced by artificial posterior vitreous detachment,[9,10] disturbance of retinal and/or choroidal circulation,[7,18] retinal damage induced by the gas bubble for postoperative tamponade,[7,8] and elevation of postoperative intraocular pressure.[11,12] However, our results reveal that the location of the visual field defect is strongly affected by the location of the infusion cannula, as described previously.[14,15] Welch[15] also postulated that the dehydration of the retina by infusion air was a main cause of this complication.

Ohji and associates[17] described the effects of humidified air during fluid–air exchange. We have been using humidified air for fluid–air exchange, but it was impossible to prevent the complication completely. We cannot precisely account for the difference between the findings in this study and those presented by Ohji and associates. Surgical procedures, including air pressure during fluid–air exchange or duration of exposure of the retina to the jet stream, might produce a difference in damage to the retina resulting in different incidence of visual field defects. Although the value of 50 mm Hg for fluid–air exchange may now seem relatively high, when this prospective study commenced in January 1996, no published information was available concerning the relationship of infusion air pressure to visual field defects.

Postoperative fundus examination performed just after residual gas disappeared revealed no detectable changes in the retina; however, it is important to examine carefully the area of the retina corresponding to the visual field defect, because in some eyes the retina showed detachment of internal limiting membrane, subretinal fibrosis, and atrophy of retinal pigment epithelium, which have corresponded with the location of the visual field defect (Yonemura N, Hirata A, and Negi A, unpublished data, 2000).

Although not statistically significant, there was a trend toward a higher incidence of postoperative visual field defect in eyes with the infusion cannula placed inferonasally. The defect seems to be located fairly centrally. This might be caused by the narrow space resulting from the relatively tight lid fissure at the nasal side. Consequently, the tip of the infusion cannula is located more posteriorly (Figure 2).

We have examined the effects of infusion air on the retina with experimental animals. The retinal area facing the infusion air showed irregularity and detachment of the internal limiting membrane and exposure of Müller cell footplates and nerve fiber layer.[19] Based on these results, the infusion air might cause not only dehydration injury but also direct mechanical damage to the contralateral region of the retina. Furthermore, the damaged retinal area could be eliminated significantly with decreased infusion air pressure (Hasumura T, Yonemura N, Hirata A, and Negi A, unpublished data, 2000). These results seem to support our clinical investigation.

In addition, serious consideration should be given to improving procedures and/or instruments with respect to how we can eliminate the impact of infusion air on the retinal surface for further prevention of this complication, because we did not achieve complete prevention of visual field defects with humidified air and an infusion pressure of 30 mm Hg. Other interventions, such as decreasing the duration of exposure of the retina to the jet stream of air or aiming the infusion cannula at multiple areas, might be helpful to decrease the incidence of visual field defects as well as to lower the infusion air pressure. We are currently

creating a new infusion cannula to diffuse the infusion air. Use of this new instrument appears to result in less retinal damage and therefore to contribute to the prevention of visual field defects (Hirata A, Hasumura T, Yonemura N, and Negi A, unpublished data, 2000).

In conclusion, this study clarified that the location of the visual field defect correlated with the location of the infusion cannula and that the incidence of this visual field defect was strongly influenced by the infusion air pressure. This study also clarified the benefits of using low infusion air pressure during surgery. We strongly recommend that the infusion air pressure used for fluid–air exchange be set at less than 30 mm Hg to reduce the incidence of visual field defects.

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