

EDITORIAL

Visual Field Defects After Vitrectomy With Fluid–Air Exchange

GREGG T. KOKAME, MD

FLUID–AIR EXCHANGE HAS BEEN A STANDARD AND widely used part of modern vitreous surgery for the past three decades.¹ Initially, this technique was primarily used to reattach the retina in vitrectomy for retinal detachment repair. Only recently because of marked advances in macular surgery, including macular hole surgery and subretinal surgery, has fluid–air exchange been increasingly performed in eyes with attached and otherwise normal peripheral

See also pp. 611–616.

retina. In such eyes after vitrectomy, predominantly inferotemporal, peripheral visual field defects were initially reported in 1995² and subsequently confirmed by multiple investigators.^{3–13} Various causes were postulated and included mechanical damage to the optic nerve by the extrusion needle during drainage,^{2–4} tractional damage to the peripapillary nerve fiber layer during posterior hyaloid removal,^{3–6} impaired retinal or choroidal circulation,^{6–7} toxic effect of gas contact to retina,^{3,7} optic nerve damage from retrobulbar injection,⁴ retinal vascular occlusion,⁸ increased intraocular pressure,^{4,9} and light toxicity.⁶ More recently, the potentially damaging effect of the pressurized air infusion itself was recognized by Welch in 1997,¹⁰ who postulated dehydration injury to the retina in the path of the pressurized air flow from the infusion cannula. The article by Hirata and associates¹¹ in this issue of *THE JOURNAL* provides compelling evidence for the potential retinal damage by pressurized air infusion, highlights the risks of high infusion air pressure, and suggests precautions during vitreous surgery using fluid–air exchange to decrease the risk of these complications.

Clinical studies of patients with visual field defects after macular hole surgery with fluid–air exchange showed the location of damage to be the inner layers of the retina.^{3,5} The findings of the nerve fiber layer analyzer (Laser Diagnostic Technology, Inc, San Diego, California) showed marked loss of the nerve fiber layer in the retinal

region corresponding to the visual field defect in six of eight patients.³ The focal electroretinogram, which is a measure of outer retinal function, was normal in scotomatous regions of the retina.^{3,5} Although these studies implicated the injury site to the inner layers of the retina, initial theories concentrated on tractional damage to the peripapillary nerve fiber layer during peeling of the cortical vitreous from the optic nerve^{3–6} or mechanical trauma to the optic nerve during drainage of fluid with the extrusion needle.^{2–4} Subsequently, attention has now been focused on the potential damaging effect of the pressurized air infusion itself on the inner layers of the retina.^{10–13}

Strong evidence of the damaging effect of pressurized air infusion was provided by clinical studies showing the relationship of the location of visual field defects to location of the infusion cannula. Predominantly inferotemporal visual field defects were noted in clinical studies using conventional three-port vitrectomy.^{3–9} In a conventional three-port vitrectomy, the infusion cannula is placed inferotemporally, and the infused air is directed toward the superonasal midperipheral retina, explaining inferotemporal field loss. When the infusion cannula was moved inferonasally, with the infused air directed at the superotemporal retina, visual field defects were predominantly inferonasal.^{11,12} When the infusion cannula was placed superiorly, with the infused air directed at the inferior retina, then field defects were predominantly superior.¹⁰

In an animal model of vitrectomy with fluid–air exchange in rabbits, retinal whitening and narrowing of the retinal vessels were observed in the path of the pressurized air infusion, supporting the potential damaging effects of the air infusion.¹⁴ Histologic studies of the areas of pale retinal injury in this study showed full-thickness damage to the retina.¹⁵ However, more recent animal studies have shown damage limited to the inner layers of the retina on histopathology.^{16–18} Sharply demarcated retinal lesions were noted on scanning electron microscopy, with damage to the inner limiting membrane and nerve fiber layer, implicating direct mechanical damage resulting from the pressurized air flow. Light microscopic study of these eyes showed damage to the inner limiting membrane, retinal ganglion cell layers, and inner plexiform layer.¹⁶ Electron microscopic studies of retina damaged by pressurized air flow showed defects of the internal limiting membrane, nerve fiber layer, and inner portions of

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From the Division of Ophthalmology, Department of Surgery, University of Hawaii School of Medicine, Honolulu, Hawaii; and The Retina Center, Kapi'olani Medical Center at Pali Momi, Aiea, Hawaii.

Reprint requests to Gregg T. Kokame, MD, Medical Director, The Retina Center at Pali Momi, 98-1079 Moanalua Rd, Suite 470, Aiea, HI 96701; fax (808) 487-3699; e-mail: retinahi@aol.com

Mueller cells, but not to outer retinal layers.^{17,18} In addition, damage was not seen in control eyes undergoing vitrectomy without fluid–air exchange.^{14–18}

The cause of the inner retinal damage from the pressurized air infusion is important to understand in an attempt to minimize the risk of this newly recognized complication. Initially, dehydration damage to the inner retinal layers was postulated by Welch.¹⁰ Attempts to decrease the risk of dehydration have included using humidified air through the infusion line,¹³ leaving a puddle of vitreous cavity fluid during the fluid–air exchange to humidify the circulating air,¹⁹ decreasing the duration of the pressurized air infusion,^{18,19} and decreasing the flow rate of the air through the vitreous cavity by placing scleral plugs.²⁰ More recent evidence has focused on the direct mechanical effect of pressurized air infusion on the retina. This is supported by the sharply demarcated retinal lesions noted in the animal studies,^{16–18} and by the recent work of Hirata and associates in this issue of *THE JOURNAL*.¹¹ While already using humidified air for pressurized infusion, as suggested by Ohji and associates to prevent retinal dehydration,¹³ the authors still noted a 24% incidence of visual field defects with an infusion air pressure of 50 mm Hg. A reduction of the infusion air pressure to 30 mm Hg resulted in a marked reduction of the incidence of visual field defects after fluid–air exchange. In animal models the use of humidified infused air also did not alter the incidence of histopathologic damage to the inner retina.^{16,17}

Mechanized air infusion systems have been used throughout the era of modern vitreous surgery. The recently recognized complication of postvitrectomy visual field defects highlights the potential damage induced by the pressurized air infusion on the retina. This damage is possible in any vitrectomy procedure that requires the use of fluid–air exchange, and precautions should be taken in vitrectomy for many different disease entities, including macular hole, subretinal surgery, retinal detachment, and proliferative diabetic retinopathy. New designs in infusion cannulas^{11,15} and mechanized air infusion systems could be considered to minimize the effects of the pressurized air flow on the retina. Alteration in technique, such as placement of the infusion cannula superiorly to minimize symptomatic field loss, has been proposed by Welch.¹⁹ Currently, the simplest and most important method to minimize the risk of visual field defects after vitrectomy with fluid–air exchange is highlighted by Hirata and associates in this issue of *THE JOURNAL*:

Keep the infused air pressure during fluid–air exchange at 30 mm Hg or less.

Although this does not entirely prevent visual field defects, the incidence is markedly decreased from 24% to 4%.¹¹

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