Neovascular glaucoma (NVG) is a difficult-to-manage form of glaucoma, which consists of vessel proliferation involving the iris and anterior chamber angle. Diseases resulting in retinal ischemia, such as central retinal vein occlusion, diabetic retinopathy, central retinal artery occlusion, carotid artery obstruction, and orbital tumor, are known to cause NVG.1-4 According to the degree of progression, treatment methods consist of panretinal photocoagulation, anterior chamber angle photocoagulation, filtering surgery, glaucoma implants, cyclophotocoagulation, cryotherapy, β-blockers, carbonic anhydrase inhibitors, and enucleation. Important aspects of management of NVG involve the detection of vessel proliferation and performing preventive treatment to stop further progression. The mainstream of early NVG treatment is panretinal photocoagulation and adjuvant anterior chamber angle photocoagulation.5-7 The delivery of panretinal photocoagulation reduces retinal ischemia, which causes vessel proliferation and thus inhibits progression of the disease.

Use of an endoscopic laser allows visualization of anatomical structures that are not visible with conventional microscopes, such as the peripheral retina, pars plana, ciliary body, and the posterior part of the iris. Furthermore, this laser is not hindered by visual obstacles, such as corneal opacity, miosis, cataract, and intraocular gas, thus making it suitable for panretinal photocoagulation of the entire retina.8 In this study, we report the therapeutic results of vitrectomy.
and aggressive endoscopic laser photocoagulation extending to the extreme periphery in NVG.

Patients & Methods
The subjects consisted of 7 patients with NVG (8 eyes) who received vitrectomy and aggressive retinal photocoagulation. The diagnosis of NVG was made on the basis of presence of vessel growth on the iris and anterior chamber angle with an intraocular pressure (IOP) of more than 21 mm Hg. All patients were retrospectively studied for systemic diseases, preoperative panretinal photocoagulation, operative procedure, change of vision, and IOP. All patients underwent preoperative and postoperative slit-lamp, gonioscopic, and fundus examinations.

All patients were treated with vitrectomy and endoscopic laser photocoagulation. We could not observe the entire retina without the aid of an endoscope. The endoscope imaging unit we used was the FV-2000EF (TDH Company, Tokyo, Japan) with the illumination source and image input system located in the front and the monitor and S-VHS connectors located in the back of the unit. Additional systems included an argon laser unit (Novus 2000, Coherent, Palo Alto, Calif), monitor (Sony PVM-1943MD, Tokyo, Japan), and S-VHS video recording unit (Victor HR-X7, Tokyo, Japan). A standard vitrectomy was performed through 3 sclerotomy sites through the pars plana, while panretinal photocoagulation was performed with the endoscopic laser. Laser photocoagulation was delivered to all visible sites using the endoscope (Fig 1).

The average number of laser burns delivered was 1515, with a coagulation time of 0.1 to 0.2 seconds and energy of 150 to 500 mW. In addition, we performed extracapsular cataract extraction (ECCE) (in 2 eyes), ciliary body coagulation (1 eye), and glaucoma valve insertion (1 eye).

Results
The subjects were 4 men (4 eyes) and 3 women (4 eyes), who ranged in age between 40 and 60 years, with the average age being 54.3 years. The follow-up period ranged from 4 to 15 months and averaged 9.5 months. Past medical history revealed 6 patients with diabetes mellitus and 1 with combined hypertension. The duration of diabetes mellitus averaged 10 years (range, 1 to 20 years), and all 6 patients had proliferative diabetic retinopathy. One patient had central retinal vein occlusion and 2 patients with diabetes mellitus had undergone cataract surgery. The patients had received panretinal photocoagulation before development of NVG. The conditions thought to have provoked the onset of NVG included proliferative diabetic retinopathy in 7 eyes and central retinal vein occlusion in 1 eye. To control preoperative IOP, both acetazolamide (Diamox) at 1000 mg/d and 2% carteolol hydrochloride (Mikelan) eye drops twice a day were administered in 6 eyes, acetazolamide (Diamox) at 1000 mg/d was given in 1 eye, and carteolol was given in 1 eye. Despite antiglaucoma therapy, the IOPs were all above 21 mm Hg. Preoperative vision was hand motion in 4 eyes, finger counting in 3 eyes, and 15/200 in 1 eye. All eyes presented with iris neovascularization, and partial anterior chamber closure was observed in 2 eyes.

There were no intraoperative complications, but in 2 eyes postoperative corneal abrasion and recurrent corneal erosions occurred. New vessels of the iris and anterior chamber disappeared or stabilized after surgery in 7 eyes, while there was progression in 1 eye. The average IOP 1 week after surgery was 20 mm Hg, and the average IOP at final follow-up was 25 mm Hg. The IOP 1 week after surgery decreased in all the eyes but one, in which the IOP of 35 mm Hg was due to angle closure. During the final follow-up, 7 eyes maintained an IOP of 25 mm Hg or less. For control of IOP, the eyes received antiglaucoma eye drops. Preoperative vision improved in 4 eyes, showed no change in 1 eye, and decreased to no light perception in 3 eyes (Table 1).

Discussion
Neovascular glaucoma may be divided into 3 stages: neovascularization of the iris, early glaucoma, and end-stage glaucoma. Accordingly, the treatment is largely divided into the prevention of new vessel progression and treatment of increased IOP. The preventive treatment is mandatory in neovascularization of the iris, while treatment of increased IOP is directed at the glaucoma. Preventive treatment modalities involving the destruction of the ischemic retina are panretinal photocoagulation and panretinal cryotherapy. It has been reported that destruction of the ischemic retina eliminates the production of angiogenic factors and thus prevents new vessel growth of