# Abnormal Ocular Pneumoplethysmographic Results in Unilateral Neovascular Glaucoma.

Eric P. Shakin MD

Thomas M. Bosley MD

William Gee MD, FACS
*Lehigh Valley Health Network*

James F. Reed III PhD
*Lehigh Valley Health Network*

Gary C. Brown MD

*See next page for additional authors*



## Published In/Presented At

Shakin, E. P., Bosley, T. M., Gee, W., Reed, J. 3., Brown, G. C., Sergott, R. C., & Savino, P. J. (1989). Abnormal Ocular Pneumoplethysmographic Results in Unilateral Neovascular Glaucoma. *Archives Of Neurology*, *46*(12), 1308-1310.

## Authors

Eric P. Shakin MD; Thomas M. Bosley MD; William Gee MD, FACS; James F. Reed III PhD; Gary C. Brown MD; Robert C. Sergott MD; and Peter J. Savino MD

# Abnormal Ocular Pneumoplethysmographic Results in Unilateral Neovascular Glaucoma

Eric P. Shakin, MD; Thomas M. Bosley, MD; William Gee, MD; James Reed III, PhD; Gary C. Brown, MD; Robert C. Sergott, MD; Peter J. Savino

**• Little is known about the predictive value of ocular pneumoplethysmography in patients with ophthalmic disease. We evaluated eight patients with unilateral increased intraocular pressure due to neovascular glaucoma who did not have evidence of severe extracranial carotid stenosis by duplex scanning and continuous-wave Doppler ultrasound. The ophthalmic systolic pressure measured by ocular pneumoplethysmography was decreased in the affected eye of all eight patients, indicating that neovascular glaucoma may be a cause of abnormal ocular pneumoplethysmographic results. Patients with neovascular glaucoma tended to have larger interocular ophthalmic systolic pressure differences than other patients with false-positive ocular pneumoplethysmographic results by noninvasive criteria.**

*(Arch Neurol.* **1989;46:1308-1310)**

Ocular pneumoplethysmography (OPG-Gee) is a sensitive and reliable carotid noninvasive test,[1] but little is known about the predictive value of OPG-Gee in patients with ocular disease. Increased intraocular pressure (IOP) has been reported to have no effect on OPG-Gee testing,[2,3] but we found abnormal OPG-Gee results in several patients with unilateral neovascular glaucoma who had no evidence of significant extracranial carotid artery stenosis by duplex scanning or continuous-wave Doppler ultrasound. This observation prompted the study of additional patients with neovascular glaucoma to determine if abnormal OPG-Gee results are consistent in this ophthalmologic problem.

Accepted for publication March 13, 1989.

From the Retina Service (Drs Shakin and Brown) and the Neuro-Ophthalmology Service (Drs Bosley and Sergott, and Mr Savino), Wills Eye Hospital, Philadelphia, Pa; and the Vascular Laboratory (Dr Gee) and Research Department (Dr Reed), Lehigh Valley Hospital Center, Allentown, Pa.

Reprint requests to Neuro-Ophthalmology Service, Wills Eye Hospital, Ninth and Walnut streets, Philadelphia, PA 19107 (Dr Bosley).

## PATIENTS AND METHODS

Nine patients with unilateral severe neovascular glaucoma were studied in the Wills Eye Hospital (Philadelphia, Pa) Vascular Studies Laboratory between January 1 and December 31, 1987, with OPG-Gee, duplex scanning, and continuous-wave Doppler ultrasound to evaluate the possibility of carotid atherosclerotic disease. Each patient had a complete ophthalmologic examination within 1 week of carotid noninvasive evaluation, and IOP was measured by Goldmann applanation tonometry immediately prior to testing.

One patient had ultrasound evidence of hemodynamically significant extracranial carotid artery disease and was excluded from further evaluation because significant carotid stenosis is known to cause abnormal OPG-Gee results. Eight patients had unilateral neovascular glaucoma without evidence by duplex scanning of hemodynamically significant extracranial carotid artery stenosis (Table 1). The cause of iris neovascularization was proliferative diabetic retinopathy in four patients and ischemic central retinal vein occlusion in four patients. Three patients had panretinal photocoagulation in the affected eye prior to developing neovascular glaucoma. No patient had iris neovascularization or elevated IOP in the unaffected contralateral eye. Mean IOP was 53.5 mm Hg in the eye with neovascular glaucoma and 16.6 mm Hg in the unaffected eye. Each patient had an IOP difference between the two eyes of at least 25 mm Hg.

Ocular pneumoplethysmography was performed with a pneumoplethysmograph (Model OPG-3LP, Electro-Diagnostic Instruments, Burbank, Calif) in the recommended fashion.[1] Ophthalmic systolic pressure (OSP), the IOP at which detectable blood flow reappeared in each globe, was recorded and compared with the brachial systolic pressure measured with a cuff and stethoscope using the standard nomogram. Ocular pulse amplitude, the maximum amplitude of the pulse wave-form, was also measured for each eye. Duplex scanning (Biosound 2000SA, Biosound, Inc, Indianapolis, Ind), and continuous-wave Doppler ultrasound (Echoflow II, Waltham Precision Inc, Waltham, Mass) were also performed.

Results of noninvasive testing in patients with neovascular glaucoma were compared with a control population consisting of all patients studied with OPG-Gee and duplex scanning in the Vascular Studies Laboratory during the same period who were older than 50 years, did not have the diagnosis of glaucoma, and had no evidence of hemodynamically significant extracranial carotid stenosis. Intraocular pressure was not known in these patients.

Student's $t$ test was used to assess the differences between the glaucomatous eye and unaffected eye and between the glaucoma group and control group. A Bonferroni adjusted significance level of .007 was used to compensate for type I errors due to multiple statistical tests.

Table 1.—Patients With Neovascular Glaucoma*

| Patient No./ Age, y/Sex | VA R | VA L | IOP, mm Hg R | IOP, mm Hg L | Stenosis, % R | Stenosis, % L | Ocular History |
|---|---|---|---|---|---|---|---|
| 1/73/F | NLP | 6/9 | 54 | 16 | 0 | 10 | PDR, HBP, DM |
| 2/69/F | NLP | 6/60 | 55 | 17 | 20 | 30 | CRVO, CVA |
| 3/55/M | NLP | 6/6 | 46 | 16 | 10 | 30 | CRVO, HBP, PRP |
| 4/79/F | 6/18 | NLP | 16 | 43 | 20 | 20 | PDR, PRP, DM |
| 5/73/M | 6/12 | NLP | 18 | 48 | 10 | 50 | CRVO, HBP |
| 6/75/M | NLP | CF | 60 | 21 | 10 | 10 | CRVO, HBP, PRP |
| 7/66/M | 6/120 | NLP | 17 | 52 | 0 | 10 | PDR, DM |
| 8/80/F | NLP | 6/120 | 70 | 12 | 20 | 0 | PDR, DM |

*VA indicates visual acuity; IOP, intraocular pressure; NLP, no light perception; PDR, proliferative diabetic retinopathy; HBP, hypertension; DM, diabetes mellitus; CRVO, central retinal vein occlusion; CVA, stroke; and PRP, panretinal photocoagulation.

Table 2.—Ocular Pneumoplethysmographic Results in Neovascular Glaucoma*

| Patient No. | BSP, mm Hg | HR, No. of Beats per Minute | OSP, mm Hg GLAU | OSP, mm Hg UNAF | OPA, mm GLAU | OPA, mm UNAF |
|---|---|---|---|---|---|---|
| 1 | 180 | 65 | 118 | 130 | 18 | 18 |
| 2 | 190 | 75 | 107 | 140 | 2 | 4 |
| 3 | 140 | 70 | 102 | 107 | 9 | 10 |
| 4 | 125 | 60 | 105 | 131 | 6 | 6 |
| 5 | 200 | 60 | 129 | 140 | 19 | 19 |
| 6 | 190 | 70 | 122 | 132 | 9 | 5 |
| 7 | 160 | 100 | 114 | 122 | 7 | 3 |
| 8 | 155 | 90 | 106 | 120 | 6 | 6 |

*BSP indicates brachial systolic pressure; HR, heart rate; OSP, ophthalmic systolic pressure; OPA, ocular pulse amplitude; GLAU, glaucomatous eye; and UNAF, unaffected eye.

Table 3.—Carotid Noninvasive Data*

| Variable | Glaucoma Group | Controls Total | Controls Normal | Controls Abnormal |
|---|---|---|---|---|
| No. of patients | 8 | 248 | 234 | 14 |
| Age, y | | | | |
| Mean | 71.1 | 68.0 | 67.9 | 68.4 |
| Range | 55-80 | 51-86 | 51-86 | 53-80 |
| Sex, M/F | 4/4 | 120/128 | 113/121 | 7/7 |
| BSP, mm Hg (SD) | 167 (25) | 151 (22) | 149 (21) | 169 (24) |
| Heart rate, No. of beats per minute (SD) | 74 (13) | 69 (12) | 69 (11) | 73 (15) |
| Carotid stenosis, % | | | | |
| Ipsilateral (SD) | 18 (14) | 14 (14) | 14 (14) | 17 (12) |
| Contralateral (SD) | 14 (11) | 13 (13) | 13 (13) | 15 (15) |
| Interocular OSP difference, mm Hg | | | | |
| Mean (SD) | 14.9 (9.0) | 0.8 (2.5) | 0.5 (2.1) | 4.8 (4.6) |
| Range | 5-33 | 0-17 | 0-4 | 6-17 |

*In the control group, normal indicates normal ocular pneumoplethysmographic results and abnormal, abnormal ocular pneumoplethysmographic results. BSP indicates brachial systolic pressure; OSP, ophthalmic systolic pressure.

## RESULTS

The results of OPG-Gee testing are shown in Table 2. All eight study patients had OSP at least 5 mm Hg lower in the eye with neovascular glaucoma than in the unaffected eye, although only one OSP value fell below the normal range predicted from brachial systolic pressure. Ocular pulse amplitude was quite variable but was not significantly different between glaucomatous and unaffected eyes ($t = 0.27$, $P > .50$).

The control group consisted of 248 patients (120 men and 128 women) with ages ranging from 51 to 86 years (Table 3). Mean interocular difference in OSP was 0.79 mm Hg, which differed significantly from the mean interocular OSP difference of 14.9 mm Hg in the study population ($t = 24.08$, $P < .001$).

Eight patients in the control group had OSP differences between the two eyes of greater than 5 mm Hg, and in six additional patients, one or both OSP values fell below the normal range predicted from brachial systolic pressure. These patients also met accepted criteria for abnormal OPG-Gee results and, like the study group, had no independent evidence of hemodynamically significant carotid stenosis. The study group was indistinguishable from this group in age, sex, or degree of carotid stenosis. Patients with neovascular glaucoma had larger interocular OSP differences.

In the neovascular glaucoma group, mean carotid stenosis was 20% ipsilateral to the eye with neovascular glaucoma and 14% on the contralateral side, but the difference of these means was not statistically significant. The mean extracranial carotid artery stenosis of 13.3% in the control group was not significantly different from that of patients with neovascular glaucoma.

## COMMENT

In recent years, noninvasive evaluation of the extracranial carotid arteries has taken on greater importance in the detection of atherosclerotic disease.[4] Ocular pneumoplethysmography is a sensitive and specific test for detecting hemodynamically significant carotid artery disease[1] and is a common component of carotid noninvasive testing.[5] Giant cell arteritis, a systemic vasculitis with predominantly visual manifestations, alters OPG-Gee results,[6] but little is known about the effect of other ophthalmologic disorders on OPG-Gee testing. We describe here abnormal OPG-Gee results in eight patients with neovascular glaucoma.

Criteria for abnormal OPG-Gee testing include an interocular OSP difference of greater than 5 mm Hg or OSP values falling below a normal range predicted from brachial systolic pressure.[1] These criteria were established by a statistical analysis of patients with conventional carotid arteriography showing no evidence of hemodynamically significant carotid stenosis. Recognized causes of abnormal test results when OPG-Gee results are compared with duplex scanning and Doppler ultrasound of the extracranial carotid system include: (1) instrument error in older devices; (2) tortuous carotid artery causing functional stenosis[2,3]; (3) hemodynamically significant stenosis of the common carotid artery or carotid bifurcation not detected by duplex scanning (false-

negative duplex scanning); (4) hemodynamically significant stenosis of the internal carotid or ophthalmic artery beyond the area visualized by duplex scanning; and (5) statistical variability (because some normal individuals will fall beyond two SDs from the normal mean).

Ophthalmic systolic pressure was decreased ipsilateral to the eye with neovascular glaucoma in all eight patients in this series who had no evidence by duplex scanning or Doppler ultrasound of hemodynamically significant stenosis of the extracranial carotid arteries. During the same time period, less than 6% of patients in the same age range without neovascular glaucoma had abnormal OPG-Gee results without evidence by noninvasive testing of hemodynamic carotid stenosis. This low rate of abnormal OPG-Gee results is in agreement with reports from other laboratories[5] and contrasts starkly with a 100% abnormal OPG-Gee rate in neovascular glaucoma. Therefore, unilateral neovascular glaucoma with striking IOP elevation should be considered another possible cause of abnormal OPG-Gee test results. These results do not address the issue of whether idiopathic glaucoma with more modest asymmetries in IOP might affect OPG-Gee results.

Several factors may contribute to decreased OSP in neovascular glaucoma. Iris neovascularization may be one manifestation of the ocular ischemic syndrome,[7] and it is possible that each patient with neovascular glaucoma had a hemodynamically significant stenosis of the intracranial internal carotid artery or ophthalmic artery. However, these patients did not meet the clinical criteria for the ocular ischemic syndrome.[7,8] Central retinal vein occlusion, proliferative diabetic retinopathy, or panretinal photocoagulation might disturb the retinal or choroidal circulation,[9] although they have not been reported to cause abnormal OPG-Gee results.

These patients had striking unilateral elevations in IOP with interocular IOP differences greater than 25 mm Hg in every case. Intraocular pressure asymmetry has not been recognized as a significant factor in OPG-Gee testing,[2,3] but previous reports did not evaluate patients with interocular differences in IOP as large as those in the present group. The OPG-Gee technique assumes that IOP is elevated to equal levels bilaterally when the same vacuum is applied to both eyes, but this assumption may be incorrect when one eye has a significantly elevated resting IOP compared with the other. Intraocular pressure in glaucomatous eyes may have been elevated during OGP-Gee testing to levels higher than anticipated so that apparent OSP was artifactually reduced when compared with the normal eye. Interocular IOP differences did not correlate significantly with interocular OSP differences, as might be expected if this hypothesis were correct. However, neovascular glaucoma may be associated with a profound derangement of pressure and mechanical factors in the globe, and the relationship between vacuum application and IOP change in the glaucomatous eye may be different from that in normal eyes.

Many patients with abnormal OPG-Gee results are subsequently evaluated with more extensive and invasive carotid studies because of the possibility that extracranial stenosis of the carotid artery might be treatable with carotid endarterectomy. Physicians involved in carotid noninvasive testing should realize that unilateral neovascular glaucoma with strikingly elevated IOP is one possible cause of abnormal OPG-Gee results, and these individuals should be spared further testing unless there is evidence by duplex scanning of significant atherosclerotic disease at the carotid bifurcation. These patients may have a disturbance of blood flow to the globe at a more distal point (intracranial internal carotid artery, ophthalmic artery, posterior ciliary arteries, or choriocapillaris), but a stenosis in one of these locations would not be treatable by extracranial carotid surgery. If an accurate ophthalmologic history is not available at the time of noninvasive testing, clues about the presence of neovascular glaucoma may be obtained from a history of total blindness in the involved eye and from the magnitude of OSP difference between the two eyes.

Dr Gee is coinventor of the ocular pneumoplethysmograph and receives royalty payments from, and is a paid consultant to, Electro-Diagnostic Instruments.

### References

1. Gee W. Ocular pneumoplethysmography. *Surv Ophthalmol.* 1985;29:276-292.
2. Eikelboom BC. *Evaluation of Carotid Artery Disease and Potential Collateral Circulation by Ocular Pneumoplethysmography.* Utrecht, the Netherlands: Uitgeversmaatschappij Juisartsenpers BV; 1981.
3. Wiebers DO, Folger WN, Younge BR. Ocular pneumoplethysmography and ophthalmodynamometry in the diagnosis of central retinal artery occlusion. *Stroke.* 1982;13:379-381.
4. Bosley TM. The role of carotid noninvasive testing in stroke prevention. *Semin Neurol.* 1986;6:194-203.
5. Castaldo JE, Nicholas GG, Gee W, Reed JF. Duplex ultrasound and OPG concordance in detecting severe carotid stenosis. *Arch Neurol.* 1989;46:518-522.
6. Bosley TM, Savino PJ, Sergott RC, Eagle RC, Sandy R, Gee W. Ocular pneumoplethysmography can help in the diagnosis of giant cell arteritis. *Arch Ophthalmol.* 1989;107:379-381.
7. Brown GC, Magargal LE, Simeone FA, Goldberg RE, Federman JL, Benson WE. Arterial obstruction and ocular neovascularization. *Ophthalmology.* 1982;89:139-146.
8. Brown GC, Magargal LE, Schachat A, Shah H. Neovascular glaucoma. *Ophthalmology.* 1984; 91:315-320.
9. Brown GC, Shah HG, Magargal LE, Savino PJ. Central retinal vein obstruction and carotid artery disease. *Ophthalmology.* 1984;91:1627-1633.