Blood Pressure, Arterial Stiffness, and Open-angle Glaucoma

The Rotterdam Study

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Objective: To investigate cross-sectional associations among blood pressures (BPs), arterial stiffness, and open-angle glaucoma (OAG).

Methods: Study participants came from the population-based Rotterdam Study. The baseline examination phase took place after an extensive home interview from March 20, 1990, to June 17, 1993, and the third phase between March 19, 1997, and December 16, 1999. Cases were classified into high-tension OAG (htOAG) and normal-tension OAG (ntOAG), according to an intraocular pressure greater than 21 mm Hg or 21 mm Hg or less. Pulse pressure was the difference between systolic and diastolic BP. Diastolic perfusion pressure was the difference between diastolic BP and the intraocular pressure; indicators of arterial stiffness were carotid-femoral pulse wave velocity and carotid distensibility. Associations were evaluated with logistic regression analysis, adjusted for age, sex, body mass index, smoking, diabetes mellitus, serum cholesterol level, and BP-lowering treatment.

Results: A total of 5317 participants were included in this study. In participants with a higher pulse pressure, the prevalence of htOAG was elevated (odds ratio [OR] per standard deviation, 1.32; 95% confidence interval [CI], 1.03-1.69). In persons treated for systemic hypertension, low diastolic perfusion pressure (<50 mm Hg) was inversely associated with ntOAG (OR, 0.25; 95% CI, 0.10-0.63) and positively associated with htOAG (OR, 4.68; 95% CI, 1.29-17.01). The lowest tertile of carotid distensibility compared with the highest had an OR for htOAG of 2.84 (95% CI, 0.99-8.10; P = .05).

Conclusions: We found that htOAG was associated with high pulse pressure, possibly with increased carotid arterial stiffness, and, only in persons treated for systemic hypertension, with low diastolic perfusion pressure. In these persons, ntOAG was associated with high diastolic BP, whereas the association between ntOAG and low diastolic perfusion pressure was inverted.

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VASCULAR ROLE IN THE PATHOPHYSIOLOGIC MECHANISM OF OPTIC NERVE DAMAGE IN OPEN-ANGLE GLAUCOMA (OAG) HAS BEEN STUDIED EXTENSIVELY. STILL, THE RELATIONS BETWEEN RISK FACTORS SUCH AS SYSTEMIC HYPERTENSION, SYSTOLIC OR DIASTOLIC BLOOD Pressures (BPs), OR PERFUSION Pressures AND OAG REMAIN CONTroversIAL. ASSOCIATIONS BETWEEN HIGH BP1–3 OR LOW PERFUSION Pressure1,2 AND OAG WERE FOUND, WHEREAS OTHER STUDIES FOUND A RELATION WITH THE ratio BETWEEN BP AND INTRAOCULAR pressure (IOP)4 OR NO relation.5−8 The different definitions of OAG used in studies, the complexity of the involved mechanisms, and different study designs may be reasons for these contradictory results.

In elderly individuals, systolic hypertension is the most observed type of hypertension, which is associated with arterial stiffness. Arterial stiffness develops with increasing age, when the elastic properties of the arteries decrease. Recently, it has been suggested that arterial stiffness is not an innocent process associated with aging but may be related to ischemic heart disease and stroke.7−9 The objective of the present study was to examine in a cross-sectional setting associations among systolic BP, diastolic BP, pulse pressure, arterial stiffness, and OAG.

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METHODS

STUDY POPULATION

The Rotterdam Study is a prospective cohort study by 4 teams with different foci among 7983 persons 55 years and older.¹⁰ The ophthalmic part of this study began after the pilot study of the other 3 teams had started and included 6872 participants (78% of eligible individuals). The baseline examination phase took place after an extensive home interview from March 20, 1990, to June 17, 1993, and the third phase between March 19, 1997, and December 16, 1999. The study was approved by the Medical Ethics Committee of Erasmus University, and written informed consent was obtained from all participants.

Of the 6872 participants, 6756 (98.3%) had complete data to diagnose OAG. Open-angle glaucoma was classified into definite, probable, and possible OAG based on the 97.5th and 99.5th percentiles of the optic disc parameters in our population, in combination with results of perimetry.¹¹ Definite OAG was defined as glaucomatous visual field loss (GVFL) with a possible glaucomatous optic neuropathy (GON) (vertical cup-disc ratio ≥0.7, asymmetry in vertical cup-disc ratio ≥0.2, or neuroretinal rim width <0.1) or probable GON (vertical cup-disc ratio ≥0.8, asymmetry in vertical cup-disc ratio ≥0.3, or neuroretinal rim width <0.05) in the same eye. Probable OAG was defined as a probable GON with a normal visual field or GVFL in the absence of any GON. Possible OAG was classified as a probable GON with a normal visual field or GVFL in the absence of any GON. Possible OAG was the presence of a field defect or unreliable test result. Participants with a field defect or unreliable second screening test result underwent kinetic Goldmann perimetry performed by a skilled perimetr ¡st, with at minimum the V-4, I-4, and I-2 isopters on both eyes.¹¹

Because pseudoexfoliative glaucoma was not excluded at baseline, we refer to the disease status as OAG instead of primary OAG, although pseudoxfolliation was not observed in any case at follow-up. For IOP the median of 3 measurements with the Goldmann applanator was obtained.¹⁴ Elevated IOP was defined as a measurement of more than 21 mm Hg in any eye or past (eg, surgical) or present IOP-lowering treatment. According to the presence or absence of an elevated IOP, persons with OAG were stratified into those with high-tension OAG (htOAG) and normal-tension OAG (ntOAG) so as to not miss potential differences in associations between these 2 groups in case they have different etiologies. All participants with GVFL or unexplained visual field loss on Goldmann perimetry were referred to the university eye clinic, where once again similar tonometry and gonioscopy were performed.

Blood pressure was assessed using the mean of 2 measurements in a sitting position with a random-zero sphygmomanometer during 1 session.¹³ We defined pulse pressure as the difference between systolic and diastolic BP and mean arterial BP as diastolic BP + 1/3 (systolic BP–diastolic BP). Differences between systolic BP, diastolic BP, or pulse pressure and IOP were called ocular systolic, diastolic, and pulse perfusion pressures. Systemic hypertension was defined as a systolic BP of 160 mm Hg or higher or a diastolic BP of 100 mm Hg or higher or both or the use of systemic antihypertensive medication.

Diabetes mellitus was defined as a nonfasting or postload serum glucose level of 200 mg/dL (11.1 mmol/L) or higher or the use of antidiabetic medication. Nonfasting total and high-density lipoprotein cholesterol levels were determined using an automatic enzymatic procedure.

Arterial stiffness was assessed only during the third phase of the Rotterdam Study.¹⁶ Pulse wave velocity was calculated as the ratio of the distance traveled by the pulse wave in meters and the time delay in seconds between the pulse wave in the carotid artery and femoral artery on the same side.¹⁶ The distance between the carotid and femoral arteries was measured over the body surface using a tape measure. Common carotid artery distensibility¹⁸ was assessed by measuring the vessel wall motion with a Duplex scanner. The distensibility coefficient (DC) was calculated as

\[ DC = \frac{(2\Delta D/D)\Delta P}{(10^{-3})/kPa}, \]

where \( \Delta D \) is the stroke change in diameter during systole, D is the end-diastolic diameter, and \( \Delta P \) is the pulse pressure. Participants who were taking and participants who were not taking drugs for systemic hypertension were included.

MEASUREMENTS

Information on smoking and eye medication was obtained during the baseline interview. A standardized eye examination was performed during the baseline phase of the study.¹¹ Optic disc characteristics were assessed with a digital image analyzer on simultaneous stereo optic disc color transparencies (Imagenet; Topcon Optical Co, Tokyo, Japan) and in case of missing or poor images with ophthalmoscopy.¹³ Visual fields of both eyes of each participant were screened with a modified 52-point suprathreshold test of the central field with 24° radius (Humphrey Field Analyzer; Carl Zeiss Meditec Inc, Dublin, Calif), and tests were repeated in case of a field defect or unreliable test result. Participants with a defect or unreliable second screening test result underwent kinetic Goldmann perimetry performed by a skilled perimetr ¡st, with at minimum the V-4, I-4, and I-2 isopters on both eyes.¹¹

Odds ratios (ORs) were calculated with logistic regression analysis, pooling cases with definite and probable OAG that were stratified into htOAG and ntOAG. Blood pressure analyses were performed again including only the cases with definite OAG and those with probable OAG based on GVFL, omitting those with probable OAG based on a probable GON.

Systolic BP, diastolic BP, and pulse pressure were examined both per standard deviation and in 4 categories, using predefined cutoff points estimated to lead to an equal number of cases in each category. Our a priori hypothesis was that there would be an inverse relationship between diastolic perfusion pressure and OAG,¹²¹¹ and thus we chose the highest category (>65 mm Hg) as the reference group. Indicators of arterial stiffness were categorized into tertiles. We included the
following as possible confounders: age, sex, diabetes mellitus, total and high-density lipoprotein cholesterol levels, body mass index, smoking (categorized as never, former, or current smoking), and BP-lowering treatment. Associations with arterial stiffness were also adjusted for mean arterial BP.

**RESULTS**

Baseline characteristics for persons with definite (n = 49) and probable (n = 166) OAG and for participants without OAG (n = 5102) are given in **Table 1**. Persons with definite OAG were older, more often male, and had a higher IOP than participants without OAG.

The ORs per standard deviation of systolic BP, diastolic BP, and pulse pressure for OAG are given in **Table 2**. A nonsignificant association was found between systolic BP and httOAG (OR, 1.21). A higher pulse pressure was associated with an increased prevalence of httOAG (OR, 1.32), and this OR increased to 1.60 when the probable OAG cases based on a probable GON were excluded. For diastolic BP, an association with ntOAG was found (OR, 1.18), which decreased and lost significance when the probable OAG cases based on a probable GON were excluded.

The presence of systemic hypertension was not significantly associated with OAG, whether with all cases together (OR, 1.29; 95% confidence interval [CI], 0.92-1.81), ntOAG cases (OR, 1.10; 95% CI, 0.73-1.66), httOAG cases (OR, 1.72; 95% CI, 1.95-3.12), or pooled definite OAG cases and probable OAG cases based on GVFL (OR, 1.64; 95% CI, 0.75-3.59).

**Table 3** gives the ORs of httOAG or ntOAG for 4 categories of BPs and pulse pressures. Participants with a diastolic BP of 85 mm Hg or higher had an OR of 1.84 for ntOAG. However, those with the highest pulse pressure of 80 mm Hg or higher had no significant association with ntOAG but an OR of 2.74 for httOAG compared with those with the lowest pulse pressure. When we excluded from these analyses the 130 cases with probable OAG based on a probable GON, the association with the diastolic BP became nonsignificant but the one with the pulse pressure was more marked, although with large 95% CIs (Table 3).

**ASSOCIATIONS IN PARTICIPANTS RECEIVING ANTIHYPERTENSIVE THERAPY**

Results of analyses between systolic perfusion or pulse perfusion pressures and OAG are not given because they did not significantly differ from those regarding systolic BP or pulse pressures and OAG (data not shown). We separately examined the associations among diastolic perfusion pressures, ntOAG, and httOAG because we expected a perfusion problem particularly in persons with low diastolic BPs, more often found in participants receiving systemic antihypertensive therapy. Because most tissue perfusion occurs during diastole, a higher risk of OAG might be found in cases with low diastolic BP given the hypothesis that low perfusion leads to OAG.

The associations among diastolic BPs, diastolic perfusion pressures, ntOAG, and httOAG in persons taking BP-lowering treatment are given in **Table 4**. Participants with the highest diastolic BP (>85 mm Hg) had an OR of 2.83 for ntOAG and a nonsignificant lower risk of httOAG than those with a diastolic BP less than 65 mm Hg. Those with a low diastolic perfusion pressure (<50 mm Hg) had a significantly lower risk of ntOAG, whereas the OR for httOAG was 4.68 compared with participants with a diastolic perfusion pressure higher than 65 mm Hg (Table 4), although these findings were based on small numbers.

**ASSOCIATIONS WITH INCREASED ARTERIAL STIFFNESS**

Participants with an increased pulse wave velocity and especially those with a low carotid distensibility coefficient, both indicative of high arterial stiffness, had a higher prevalence of httOAG, but results were not statistically significant (**Table 5**). Those with the highest pulse wave velocity had an OR of 1.91 for httOAG. Participants in the lowest tertile of distensibility had a nonsignificant OR...
of 2.84 (P = .05) compared with those with the highest distensibility. No associations were found between parameters of arterial stiffness and ntOAG. There was insufficient power to repeat these analyses after pooling cases with definite OAG and those with probable OAG based on GVFL.

This study revealed no clear associations between systolic BP and OAG, whether ntOAG or htOAG. A high diastolic BP led only to an increased risk of ntOAG. High pulse pressures were a consistent risk factor for OAG and

### Table 2. Odds Ratios (95% Confidence Intervals) for OAG per Standard Deviation of Systolic or Diastolic BP or Pulse Pressure

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>All OAG Cases (n = 215)†</th>
<th>Normal-Tension OAG Cases (n = 85)‡</th>
<th>All OAG Cases (n = 150)†</th>
<th>Normal-Tension OAG Cases (n = 49)‡</th>
<th>All OAG Cases (n = 65)†</th>
<th>Normal-Tension OAG Cases (n = 36)‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic BP</td>
<td>1.12 (0.98-1.29)</td>
<td>1.20 (0.97-1.48)</td>
<td>1.07 (0.91-1.26)</td>
<td>1.11 (0.84-1.47)</td>
<td>1.21 (0.95-1.54)</td>
<td>1.31 (0.96-1.79)</td>
</tr>
<tr>
<td>Diastolic BP</td>
<td>1.09 (0.96-1.25)</td>
<td>0.99 (0.80-1.22)</td>
<td>1.18 (1.01-1.37)</td>
<td>1.13 (0.86-1.47)</td>
<td>0.96 (0.75-1.23)</td>
<td>0.82 (0.59-1.15)</td>
</tr>
<tr>
<td>Pulse pressure</td>
<td>1.09 (0.95-1.25)</td>
<td>1.27 (1.02-1.58)</td>
<td>0.97 (0.82-1.15)</td>
<td>1.05 (0.78-1.41)</td>
<td>1.32 (1.03-1.69)</td>
<td>1.60 (1.17-2.18)</td>
</tr>
</tbody>
</table>

Abbreviations: BP, blood pressure; OAG, open-angle glaucoma.

*Odds ratios were adjusted for age, sex, diabetes mellitus, cholesterol level, body mass index (calculated as weight in kilograms divided by the square of height in meters), smoking, and the use of BP-lowering treatment. Both persons with normal-tension and high-tension OAG were included in the case group.

†Included were definite OAG cases and all probable OAG cases based on probable glaucomatous optic neuropathy or a glaucomatous visual field test result.

‡Excluded were probable OAG cases based on probable glaucomatous optic neuropathy.

### Table 3. ORs (95% CIs) for Definite and Probable OAG in Relation to Categories of BP and Pulse Pressure

<table>
<thead>
<tr>
<th>Analyses for All OAG Cases</th>
<th>No. of OAG Cases/Controls</th>
<th>OR (95% CI) for All OAG Cases</th>
<th>OR (95% CI) for Normal-Tension OAG Cases</th>
<th>OR (95% CI) for High-Tension OAG Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Analyses Excluding 130 Cases With Probable OAG Based on Probable GON</td>
<td>No. of OAG Cases/Controls</td>
<td>OR (95% CI) for All OAG Cases</td>
<td>OR (95% CI) for Normal-Tension OAG Cases</td>
<td>OR (95% CI) for High-Tension OAG Cases</td>
</tr>
</tbody>
</table>

Abbreviations: BP, blood pressure; CI, confidence interval; GON, glaucomatous optic neuropathy; OAG, open-angle glaucoma; OR, odds ratio.

*The ORs were adjusted for age, sex, diabetes mellitus, cholesterol, body mass index (calculated as weight in kilograms divided by the square of height in meters), smoking, and the use of BP-lowering treatment.
especially hTOAG. Systolic perfusion pressures showed no associations, but there was an inverted association between low diastolic perfusion pressures and ntOAG and a positive association with hTOAG. No significant associations could be found between pulse wave velocity or carotid distensibility and any type of OAG.

All participants underwent a standardized ophthalmologic examination during which signs of OAG were assessed independently in a masked fashion to ensure an unbiased diagnosis. The IOP level was not used for the OAG diagnosis, which enabled us to investigate associations separately in both persons with ntOAG and those with hTOAG.

Current OAG is considered to be a multifactorial disease in which it is likely that combinations of genetic and environmental determinants are responsible for the different phenotypes of the disease. In ntOAG, other factors may be important than in hTOAG. Risk factors that are involved in a subset of the heterogeneous group of OAG may become apparent only when analyzing these subsets independently. For this reason we separated ntOAG from hTOAG in our analyses, realizing that the distinction between an IOP greater than 21 mm Hg and an IOP of 21 mm Hg or less is artificial for a continuous measure such as IOP. Besides this artificial division, it remains controversial how many times around the clock and for how many days at a stretch the IOP should be measured and consistently found to be lower than 21 mm Hg before a diagnosis of ntOAG may be made. The value of 21 mm Hg was chosen because it represented the 97.5th percentile in our population and because it is a commonly used cutoff point.

Misclassification of ntOAG cases as hTOAG owing to the use of antiglaucoma medication in participants with IOPs less than 21 mm Hg cannot be excluded. It is not likely that such misclassification played an important role, because at baseline (March 20, 1990, through June 17, 1993) ntOAG was present in half our OAG cases and ntOAG was not treated as frequently and strictly as is currently recommended. Moreover, at baseline half of all OAG cases were newly detected.

<table>
<thead>
<tr>
<th>No. of OAG cases/controls</th>
<th>OR (95% CI)</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diastolic BP, mm Hg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;65</td>
<td>8/341</td>
<td>1 [Reference]</td>
</tr>
<tr>
<td>65-74</td>
<td>19/548</td>
<td>1.54 (0.66-3.62)</td>
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<tr>
<td>75-85</td>
<td>13/494</td>
<td>1.36 (0.55-3.36)</td>
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<tr>
<td>&gt;85</td>
<td>17/328</td>
<td>2.83 (1.17-6.82)</td>
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<tr>
<td>Diastolic perfusion pressure, mm Hg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;50</td>
<td>6/404</td>
<td>0.25 (0.10-0.63)</td>
</tr>
<tr>
<td>50-57.4</td>
<td>18/370</td>
<td>0.76 (0.39-1.49)</td>
</tr>
<tr>
<td>57.5-65</td>
<td>12/430</td>
<td>0.54 (0.27-1.11)</td>
</tr>
<tr>
<td>&gt;65</td>
<td>23/485</td>
<td>1 [Reference]</td>
</tr>
</tbody>
</table>

Abbreviations: CI, confidence interval; OAG, open-angle glaucoma; OR, odds ratio.
*The ORs were adjusted for age, sex, diabetes mellitus, cholesterol, body mass index (calculated as weight in kilograms divided by the square of height in meters), and smoking.
†Both definite and probable OAG cases were included in the case group.
The results of our subanalyses should be interpreted with care because our strict algorithm without subjective final classification leading to a definition of definite OAG, as well as the separation of ntOAG and htOAG cases, led to low numbers of cases in each category. We used strict criteria for definite and probable OAG, and we excluded a large group of possible OAG cases from the analyses. This approach also reduced the number of cases but potentially led to a better distinction between cases and controls. The same caveats hold for the analyses using pooled cases with definite OAG and those with probable OAG based on GVFL. The number of cases in the latter analyses was smaller but, in our view, reflected the cases with damage due to OAG better than those in which probable OAG, based on probable GON only, were included. Finally, we could have missed OAG cases because we used a suprathreshold visual field screening program, which is not as sensitive as detecting OAG as full- or modified-threshold programs that are currently available. On the other hand, this study is one of the few population-based studies in which each eye of every participant was screened and the participants who were diagnosed as having definite OAG had at least 3 perimetric tests.

Several population-based studies came to different conclusions about the relation between BP and OAG, partly because of varying definitions of OAG among studies. Our finding of an association between low diastolic perfusion pressure and htOAG confirms those of the Egna-Neumarkt Study. In the Baltimore Eye Survey no distinction was made between ntOAG and htOAG, and the investigators found a nonsignificant association between systolic BP and the total group of OAG cases along with a strongly increased risk of OAG for low diastolic perfusion pressure. The Barbados Eye Study showed in black persons an increased risk of OAG for low diastolic BP, a relation we found only in participants taking antihypertensive medication. Unlike our results, that study also found an increased risk of low systolic BP and low perfusion pressures and an inverse association between systemic hypertension and OAG. Comparison with our study is difficult because in the prevalence phase of the Barbados Eye Study, the classification of OAG was different from our study and the population was ethnically different. Besides, apart from adjusting for age and sex, as was done in the Barbados Eye Study, we also adjusted for main cardiovascular confounders, among them total and high-density lipoprotein cholesterol levels, body mass index, and smoking. Adjustment for cardiovascular risk factors changed the results remarkably: crude risks adjusted for age and sex only (data not shown) were stronger and more often significant than the risks we report in this article.

Increased pulse pressure and possibly low carotid distensibility may lead to htOAG by the following mechanism. In elderly persons, systolic hypertension with normal or even low diastolic BP levels is often observed, resulting in a high pulse pressure and accompanying arterial stiffness. We hypothesize that high pulse pressure and arterial stiffness may lead to disruption of ocular autoregulation. It is suggested that a perfusion pressure above or below a critical range may further disturb the autoregulation, which is already compromised in ocular vessels in patients with OAG. When the IOP increases in persons with a defective ocular autoregulation, vessels may not be able to respond to a low diastolic BP to maintain perfusion, resulting in ischemia and optic nerve damage. Ischemia has been proposed by many investigators as a cause of OAG because in these cases the blood flow in the optic nerve head and retina is reduced. Although low diastolic BP and high pulse pressure are often present together, each phenomenon may have a separate effect on OAG. The presence of low diastolic BP accompanying increased pulse pressure may further increase the risk of OAG by decreasing diastolic perfusion.

In participants who were being treated for systemic hypertension, an inverse association between low diastolic perfusion pressure and ntOAG was observed, whereas in contrast a positive association between low perfusion pressure and htOAG was found. One explanation for the former observation may be that the normal IOPs in these persons level out differences between diastolic perfusion pressure and diastolic BP. A high diastolic BP could cause OAG by microvascular damage with ischemia as a consequence. The association between low diastolic perfusion pressure and htOAG in our study was present only in participants receiving treatment for systemic hypertension, probably because the perfusion in the eye will be more hampered in case of an elevated IOP. Visual field progression in patients with OAG was observed with nocturnal dips in systemic BP. In particular when receiving systemic antihypertensive therapy, or in combination with a high nocturnal IOP. Our findings support the theory that treatment of systemic hypertension that leads to nocturnal hypotension may be a risk factor for htOAG.

Another explanation may be that recently discovered mechanisms that affect vascular tone and diameters are involved. In both arterioles and venules, smooth muscle cells play an important role in maintaining vascular patency. When venules become exposed to high pressures, smooth muscle cell hyperplasia occurs, resulting in venous narrowing. Eutrophic inward remodeling, a structural reduction in the vessel lumen without changes in its wall thickness, occurs in subendocardial arterioles when either the blood flow through these vessels is no longer pulsatile or the BP is lowered from 80 to 40 mm Hg. This process could be reversed into even outward remodeling by the calcium blocker amiodipine. Mean pressure or pulse pressure reduction thus leads to microvascular constriction and subsequent inward remodeling. Evidence exists that tissue-type transglutaminase acts as a kind of biological fixative for narrowed arterioles after 1 week and that this process is counteracted by factor XIII from monocytes or macrophages. Thus, one could also speculate that low diastolic perfusion pressure would lead to less venular inward remodeling and therefore less ntOAG. Another explanation for the inverse association between low diastolic perfusion pressure and ntOAG could be that this was a coincidental finding due to low numbers.

To our knowledge, no reports exist on associations between pulse pressure or arterial stiffness and either htOAG or ntOAG. We found no significant association between pulse wave velocity and OAG. The fact that only
In conclusion, we found that htOAG was associated with high pulse pressure, possibly with increased carotid arterial stiffness, and, only in persons treated for systemic hypertension, with low diastolic perfusion pressure. In these persons, ntOAG was associated with high diastolic BP, whereas the association between ntOAG and low diastolic perfusion pressure was inverted.

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Ophthalmological Numismatics

Ignacio Barraquer (1884-1965) of Barcelona, Spain, followed his father in the practice of ophthalmology. In 1941, he founded the Barraquer Institute, which is dedicated to the research and the interchange of scientific ideas. Ignacio Barraquer was especially famous for the invention of the cryosphen, which was used in intracapsular cataract extraction.

In 1968, a commemorative medal of Barraquer was struck by Calico of Barcelona, Spain. The obverse depicts the bust of Barraquer facing right. The reverse depicts the winged eye of Horus, the ancient Egyptian symbol of a healthy and safe eye.

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