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Ischemic Hypertension of Pigeon Eye

Masao Nakabayashi

Department of Ophthalmology, Osaka University Medical School, Osaka, Japan

Purpose: To determine the etiology of open-angle glaucoma in human eyes, we tested the hypothesis that ischemia of the endothelial cells lining Schlemm's canal alters the intraocular pressure (IOP) by affecting the transport of fluids out of the eye.

Methods: Experiments were conducted on pigeons. After blocking the two major arteries to the aqueous sinus artery of one eye by laser photocoagulation, the episcleral arteries of both eyes were cauterized. The IOP was measured with a Mentor pneumatonometer before, immediately, and several days after blocking the major arteries.

Results: After the episcleral cauterization, the IOP of the laser-treated eye rose significantly and was maintained for at least 3 hours. By day 2, the IOP had recovered to the pre-cauterization level. The IOP of the control eye remained unchanged throughout the experiment.

Conclusion: These results strongly support the hypothesis that ischemia of the endothelial cells lining Schlemm's canal plays a role in the control of the IOP. **Jpn J Ophthalmol 2001;45:128–136** © 2001 Japanese Ophthalmological Society

Key Words: Ischemic theory, ocular hypertension, open-angle glaucoma etiology, pigeon eye.

Introduction

From earlier clinical and experimental observations, we have hypothesized that ischemia of the endothelial cells lining Schlemm's canal alters the transport of fluids out of the eye leading to a rise in intraocular pressure (IOP).[1] To test this hypothesis, we attempted to induce ischemia of the endothelial cells lining Schlemm's canal in the pigeon eye by blocking the two major arterial routes to the aqueous sinus artery, the homologue of Schlemm's canal artery.[2] However, we found that the IOP of the pigeon eye did not increase but remained unaltered. We discovered that arterial blood had been entering the aqueous sinus perhaps through arterio-venous (A-V) or A-sinus anastomosis and, consequently, ischemia had not been induced.

In the present study, we interrupted the two major arterial routes as we had done in our previous experiment, and also blocked an alternate route through the episcleral anastomosis. We then compared the IOP before and after these procedures. We shall show that there was a transient elevation in the IOP following the blockage of arterial flow.

Materials and Methods

The experiments were conducted on white pigeons, *Streptoperia risoria*, of both sexes and weighing 130–170 g. The rationale for selecting the pigeon as a model has been described.[2] The pigeons, obtained from a local vendor (Sakura, Aichi), were maintained in the laboratory and given free access to avian chow (Banbi Co., Aichi) and drinking water. This study was conducted in accordance with the ARVO resolution on the use of animals in research. Twenty pigeons were used including those used for the preliminary calibration and anatomical mapping studies.

Calibration of the Tonometer for the Pigeon Eye

The Pneumatonometer, Model 30 Classic (Mentor O&O, Norwell, MA, USA), referred to in this paper as the Mentor-PTG, was used because it provided a more objective value of the IOP. The instrument has

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Correspondence and reprint requests to: Masao NAKABAYASHI, MD, Department of Ophthalmology, Osaka University Medical School, 2-2 Yamadaoka, Suita, Osaka 565-0871, Japan

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a standard calibrator with a water column and a proxy membrane imitating the human cornea. This was modified by constructing a water column from an acrylate tube with a 6.0 mm inside diameter and a 9.0 mm setting hole for the test tissue (Figure 1).

A pigeon was euthanized by an intramuscular injection of 100 mg of ketamine hydrochloride (Ketalar™, Sankyo, Tokyo) and the anterior segment of the eye was cut off without enucleation. The lens and the vitreous were carefully removed without touching the cornea. The remaining anterior segment was fixed in the setting hole of the calibrator by clamping the sclera outside the osciculi. The calibrator was then filled with physiological saline solution, and the pressure in the column was elevated by injection of saline through a syringe and closed by a tap. The pressure was elevated to be equivalent to 5, 10, 15, etc. up to 75 mm Hg. The sensor-tip was placed on the cornea and the pressure was measured in the digital mode of the Mentor-PTG. Measurements were made at each level with the pressure first increased and then decreased. This procedure was repeated twice. Thus for each level, four values of pressure were obtained.

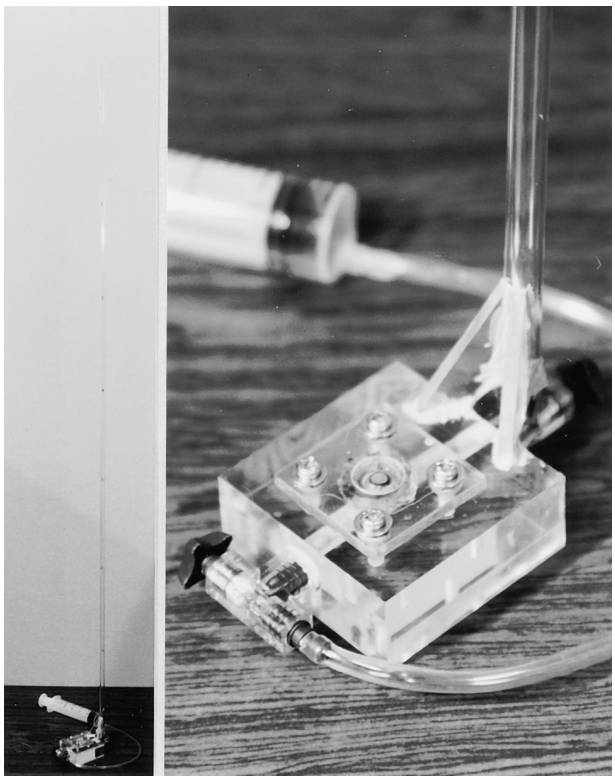

Figure 1. Calibrator (complete view and trunk with extracted pigeon cornea).

Mapping of Episcleral Vessels

A pigeon was euthanized by an overdose of ketamine, and 15 mL of a casting resin monomer (Mercox™, Dainippon Ink, Tokyo) mixed with 0.1 mL of polymerizing agent was immediately injected into the left ventricle of the heart by a shortened indwelling needle (Figure 2). After polymerization (10 to 40 minutes), the upper and lower eyelids of the eye were cut with care to avoid the veins on the inside of the lid. The flaps of the eyelid were turned up, and the veins that branched from the three collecting portions of the episcleral vessels were cauterized at the fornix with a platinum-wire cauterizer (Paquelin, Handaya, Tokyo), in order to seal the episcleral venous channels. The flaps of the lid and nictitating membrane were then excised and the head was immersed in 10% formalin solution.

The episcleral vessels were observed and photographed in the formalin solution. The same procedure was performed on the other side eye of another pigeon.

Procedures to Test the Ischemia Hypothesis

To test the ischemia hypothesis, the following six procedures were performed consecutively on 6 pigeons with the right eye tested in 3 pigeons and the left eye, in the 3 other pigeons. Procedures 2 and 3, below, were performed only on the experimental eyes and the other procedures were performed on both eyes.

1. Preliminary measurement of the IOP. Initially, the IOP of both eyes was measured to obtain the control IOPs. To record the IOP without using a lid-retractor, the bill of the pigeon, with the wings and the legs restrained, was held by one hand, and a drop of 0.4% oxybuprocain

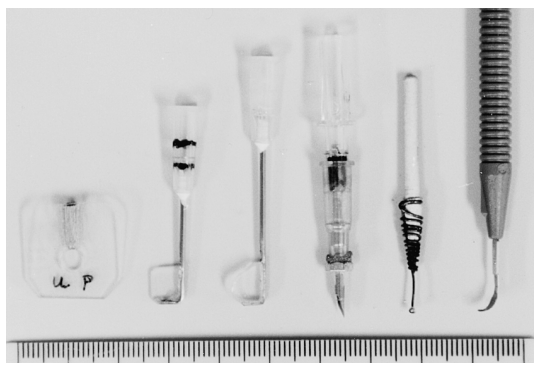

Figure 2. Bite-plate, applanators, needle for heart, altered tip of soldering iron, and transformed spatula.

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(Benoxyl™, Santen, Osaka) was instilled. The lower lid was pushed aside, if it covered the cornea, by the tip of the sensor, and the IOP was recorded by placing the tip of the sensor on the cornea.

2. Blocking the anastomotic artery by YAG-laser. Following the measurement of the control IOP, the bill was fixed by a clamp to a bite-plate (Figure 2) to maintain a clear breathing passage. The body was placed in the supine position, the clamp was fixed and another drop of oxybuprocain was instilled on the eye. To examine the lower chamber angle, an applanator, made of a piece of a microscope slide and an 18-gauge injecting needle (Figure 2), was placed on the upper section of the cornea. The ciliary cleft was identified through the applanator with a slit-lamp microscope equipped with a Q-switch 3000 LE YAG-laser (Alcon, Fort Worth, TX, USA). The anastomotic artery and accompanying white bridge was found crossing over the ciliary cleft, and was exposed to 4.0 to 5.0 mJ of the YAG-laser. The appearance of bleeding was a sign that sufficient treatment had been given. The contralateral eye was not treated.
3. Coagulation of the penetrating artery. After returning the body to the normal position, another applanator, constructed of a 3-mm-thick acrylate plate and a needle (Figure 2), was placed on the lower limbus of the same eye. To distinguish arteries from veins, the applanator was pressed gently on the limbus to collapse the veins while the artery remained patent. One exposure of a continuous wave (CW) Krypton-laser, Novus Omni (Coherent, Palo Alto, CA, USA) of 521 nm wavelength, 0.2 mm diameter, at 900 mW for 0.1 second, whitened the penetrating artery. However, at least 10 additional burns were placed along the artery in order to ensure the occlusion and prolong the duration of blockage of the penetrating artery. The contralateral eye was not treated. After this treatment the bill was released.
4. Intermediate measurements of IOP. Twenty and 40 minutes after the laser treatment, the IOP of the laser-treated and contralateral eyes was measured. The pigeons with elevated IOPs would have been eliminated from further study, but there were no such cases.
5. Cauterizing the episcleral arteries with the veins. After the intermediate IOP measurements, the bill was fixed again. Under an operating microscope, the posterior corner of the eyelid was pulled away from the sclera by a spatula made from a disposable micro-scalpel (Figure 2), and the posterior collecting point of the episcleral vessels was found. This was aided by examining the episcleral maps obtained from studies of the resin-filled episcleral vessels (Figure 4). Protecting the lid with the spatula, the collecting point was cauterized with the Paquelin at a tip-temperature of 690–580°C, or with a thin modified soldering iron (15 watts, 360°C; Taiyo-Denki, Hiroshima) (Figure 2). The tip of the cauterizer was placed on the conjunctiva only briefly to create a 1-mm burn and to avoid the cauterization of deeper structures.

   In a like manner, the inferior and anterior collecting points of the episcleral vessels were cauterized. For the anterior vessel, the nictitating membrane was also pulled away and protected. This procedure of cauterizing the three collecting episcleral vessels was performed on the contralateral eye as a control. After the episcleral treatment for both eyes, the restraints on the bird, other than the wings, were removed.
6. Follow-up measurements of IOP. After the episcleral cauterization, the IOP of the laser-treated and control eyes was measured every 20 minutes for 3 hours. At each time, four measurements were made and the average was used in the analysis. After 3 hours of observation, the wing restraint was removed and the pigeon was free in the cage. Thereafter, the IOP was measured on days 2, 5, and 9 after treatment.

Although the visual capacity of the pigeons was not tested systematically, the animals appeared well and functioned normally. All the animals survived to day 9.

Results

Calibration of Tonometer for the Pigeon Eye

The four values and the average recorded by the pneumotonometer at each height on the water column are shown in Table 1. The mean Mentor-PTG values (M) are also plotted against the height of the water column in Figure 3. As expected, there was a linear relationship between the height of the water column and the recorded values, although there were discrepancies at the higher pressures. The equation for this relationship is:

$$P = 1.73M - 5.4 \text{ mm Hg.}$$

Table 1. Calibration Data of Mentor-PTG with Pigeon Cornea

Water column in mm Hg (P)	Mentor value in mm Hg (M)				
	I	II	III	IV	Average
5	5	5.5	5.5	5.5	5.375
10	9	9	9.5	9	9.125
15	12.5	11	11.5	11	11.5
20	14.5	14	13.5	15.5	14.375
25	17	17	16.5	18	17.125
30	19.5	21	21	20	20.375
35	24	23	23.5	24	23.625
40	27	27	27	27.5	27.125
45	30	30.5	30	29.5	30
50	32	33	33	31	32.25
55	36	35.5	35.5	35	35.5
60	38.5	38.5	38	37	38
65	41	40.5	39.5	39	40
70	44.5	43	42.5	42.5	43.125
75	47.5	45.5	43.5	45	45.375

Because of the good correlation between the M-values and the water height, the IOP was expressed in M-values without conversion for further analysis.

The Episcleral Vessels

The resin-filled episcleral vessels were observed and photographed in the formalin solution (Figure 4). The arteries were distinguished from the veins by the green resin. The three collecting episcleral vessels were found near the anterior, posterior and inferior fornix. The veins gave off branches from there, and the arteries ran with less branching. The artery was often accompanied on both sides by two parallel veins. The inferior artery ran into the penetrating artery for the aqueous sinus artery and sometimes into one other branch. Although the arterial course was not consistent, there were no arteries flowing into the episclera from parts other than these three collecting segments.

Ischemia and the IOP

No case was eliminated in procedure 4 because of IOP elevation after laser treatment.

In the 6 pigeons, the IOP values of the laser-treated and the control eyes, measured every 20 minutes for 3 hours, and then on days 2, 5, and 9, are shown in Table 2 and plotted in Figure 5. These findings show that the IOP of the target eyes increased significantly while that of the control eyes remained unchanged following the episcleral cauterization. At each time point after the cauterization, the IOPs of the 6 target eyes were significantly higher than the

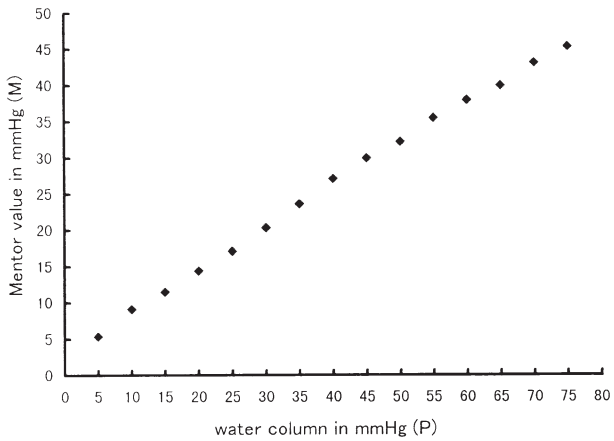

Figure 3. Calibration of Mentor-PTG with pigeon cornea.

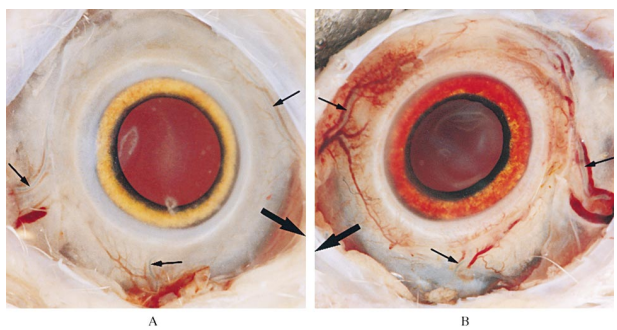

Figure 4. Episcleral maps of pigeon eye after intracardial injection of resin. (**A**) Small arrow: resin-filled artery, large arrow: toward bill. (**B**) veins were emphasized by elevating tail of pigeon.

Table 2. Intraocular Pressure Courses of 6 Pigeons

Pigeon	Eye*		Before†		20	40 (min)‡		20	40	60	80	100	120	140	160	180 (min)	2	5	9 (Days)
A	R*	Mean	26.5	lt	24.88	23.88	ec	45.63	49.88	50.75	47.75	54.63	50	44.75	39.5	36.38	28.5	25.13	23.38
		SD	0.91		2.29	2.75		5.48	2.59	2.36	5.95	1.65	2.74	1.19	2.45	3.09	1.91	1.7	2.29
	L	Mean	25.25		23.13	23.88	ec	23.13	22	24.88	20.75	22.38	22.88	22.5	22.88	22.63	24.38	23.38	22.38
		SD	2.22		1.25	2.06		2.14	1.08	1.44	2.9	2.5	2.75	1.68	2.36	0.95	2.36	1.11	1.31
B	R*	Mean	23.13	lt	22.88	22.75	ec	39	41.88	40.63	40.38	31.13	36.38	32.38	31.88	31	16.25	17.25	24.63
		SD	2.53		2.78	1.44		3.34	4.5	3.5	1.31	2.06	2.72	1.65	1.31	2.68	3.3	2.4	2.87
	L	Mean	23.25		26.25	24.38	ec	22	24.5	24.63	23.38	22.38	24.25	24.13	23.5	23.63	23.88	23.25	23.75
		SD	4.37		2.06	4.82		2.52	3.49	3	3.35	2.5	2.1	2.17	1.08	1.49	1.65	1.04	1.66
C	R*	Mean	24.88	lt	24.5	24.38	ec	36.5	49	51.13	49.24	47.88	46.13	44.13	41.5	41.63	23.13	24.5	23.5
		SD	1.49		2.68	1.49		4.53	2.16	2.75	3.66	2.5	2.93	1.38	3.16	4.39	2.4	2.38	1.83
	L	Mean	22.5		24.25	23	ec	22	22.63	21.5	21.5	23	24.13	24	21.63	25.13	25.38	22.75	26
		SD	1.08		3.07	1.35		2.12	2.29	0.41	1.58	2.65	1.03	2.68	3.33	2.17	1.25	2.53	23.5
D	R	Mean	24.38		24.88	23.88	ec	22.25	23.63	22.38	22.75	23.63	25.38	22.88	22	22.75	23.5	24.5	23.25
		SD	2.25		2.02	3.17		1.44	3.07	2.5	2.33	2.84	2.25	3.61	1.22	3.8	0.82	1.58	1.66
	L*	Mean	25.75	lt	24.88	25.38	ec	40	48	51.75	46.75	43	40.25	34.25	35.38	33.63	22.13	22.63	23
		SD	1.71		2.9	2.33		3.72	4.92	3.88	4.03	2.48	3.18	1.85	2.06	3.33	1.03	2.5	1.87
E	R	Mean	24.38		23	23.38	ec	21.13	18.75	21.38	19.75	21.75	21.63	21	20.38	20.25	21.75	25.13	23.38
		SD	1.03		1.22	1.49		1.84	2.06	1.55	1.55	3.1	0.95	1.08	0.85	1.44	1.71	1.7	2.29
	L*	Mean	23.88	lt	22.25	22.5	ec	33.38	43.5	46	51.63	51.38	52.88	51.13	52.63	53.5	22.13	23.38	22.38
		SD	2.1		2.22	3.34		2.17	2.86	3.19	2.95	3.15	4.25	4.75	4.5	3.39	0.48	1.11	1.31
F	R	Mean	24.88		23.5	21.63	ec	26.25	23.38	26	26.25	24.38	24.63	22	24.5	23.25	26.38	25	23.88
		SD	3.68		1.08	2.32		2.06	3.35	2.48	2.99	4.82	2.98	2.52	3.49	4.37	3.94	3.24	1.75
	L*	Mean	25.38	lt	23.75	23.75	ec	43.25	55.5	45.75	54	46.38	46.13	42	39	38.75	15.38	21.63	23
		SD	1.11		2.02	2.53		5.42	8.34	6.65	6.32	7.95	6.02	6.4	2.27	2.87	4.01	5.45	0.71
	6 Target eyes	Mean	24.92		23.85	23.77		39.63	47.96	47.67	48.33	45.73	45.29	41.44	39.98	39.15	21.25	22.42	23.31
		SD	1.92		2.44	2.35		5.61	6.17	5.36	5.84	8.4	6.62	7.23	7.05	8	5.03	3.7	1.85
	6 Control eyes	Mean	24.1		24.17	23.35		22.78	22.48	23.46	22.4	22.9	23.63	22.75	22.48	22.94	24.21	24	23.35
		SD	2.6		2.05	2.64		2.48	3.03	2.59	3.11	2.94	2.45	2.42	2.46	2.81	2.45	2.02	1.81

*Target eye.
†lt: YAG- and continuous wave-laser treatments.
‡ec: Episcleral cauterization.

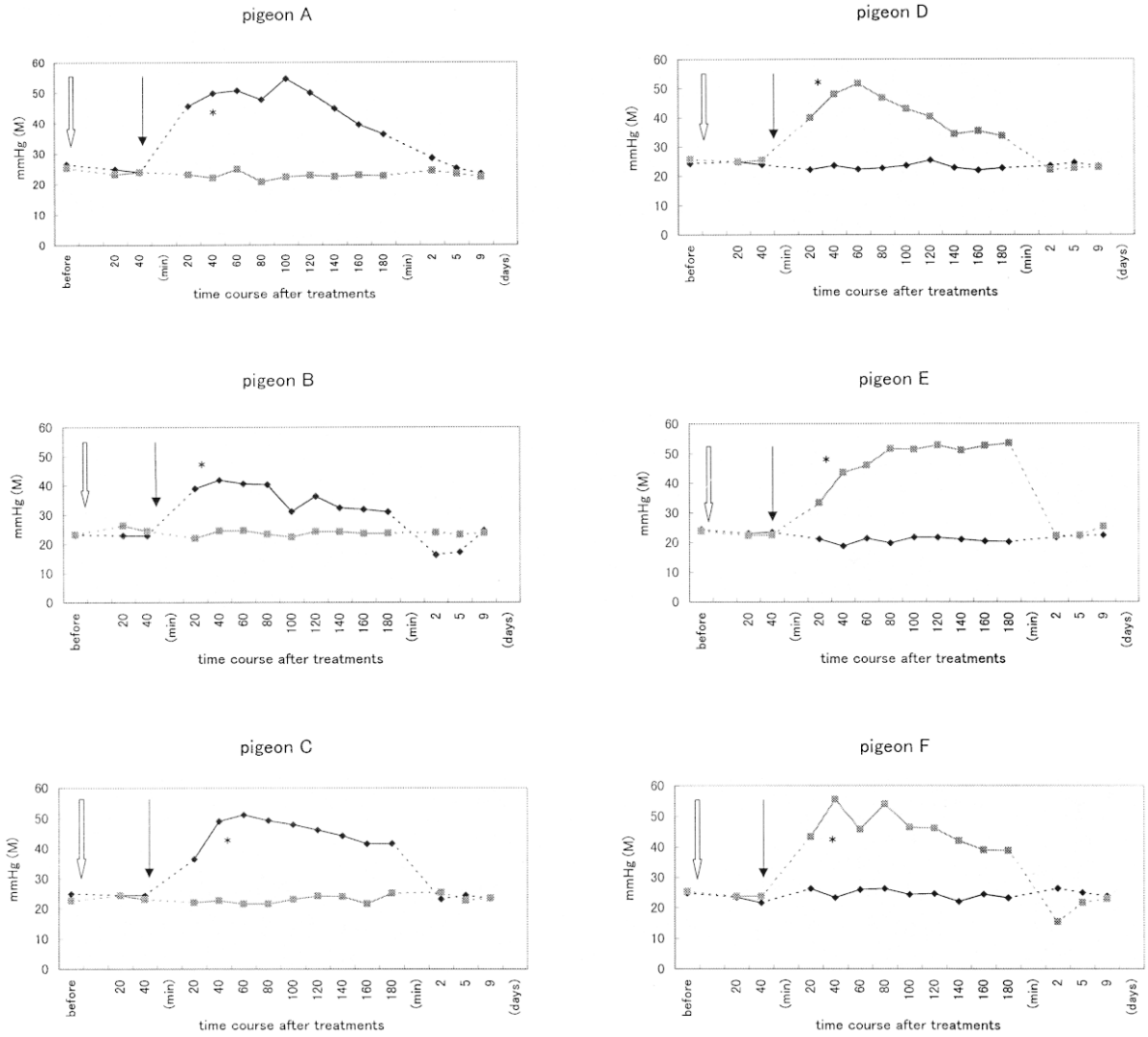

Figure 5. Time course of intraocular pressure after treatment. *: target eye, ◆: right eye, ■: left eye, white arrow: YAG- and continuous wave-laser treatment to target eye, black arrow: episcleral cauterization to both eyes.

IOPs before the cauterization. The IOPs were also significantly higher than the IOPs of the 6 control eyes at the same time points (paired *t*-tests, $P < .0001$).

Two days after the treatment, the IOP of the target eyes returned to normal, and remained at this level on day 9.

Regeneration of Arteries

The blood in the anterior chamber caused by the YAG-laser was resolved in 2 to 4 weeks, and the photcoagulated anastomotic artery was often regenerated as indicated by the absence of clot at the later time periods (Figure 6). The penetrating artery had also been regenerated earlier. The regeneration of these arteries probably accounts for the normalizing of the IOP. Whether there was regeneration of the cauterized episcleral vessels was more difficult to determine.

Repeat Photocoagulations

Six to 7 weeks after these experimental procedures, the control and the target eyes of different pigeons were photocoagulated as in the foregoing experiments with the YAG- and CW-laser. There was minimal bleeding into the anterior chamber at this time. Episcleral cauterization, however, was not done. As previously, there was a significant eleva-

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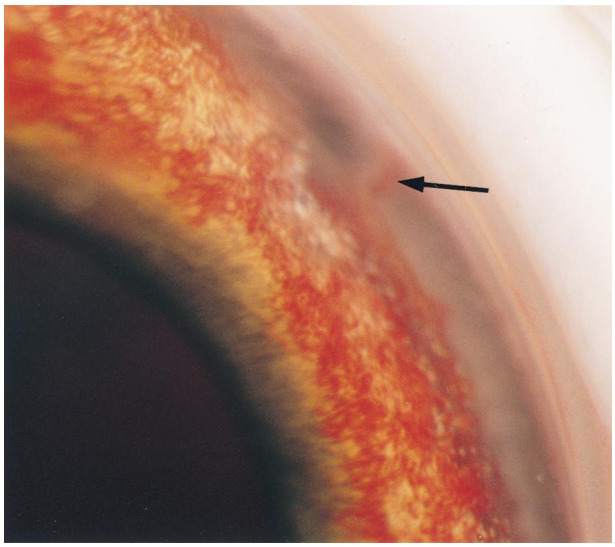

Figure 6. Regenerated anastomotic artery (arrow) of target eye of pigeon E 28 days after laser treatment.

tion of the IOP (Figure 7). These results demonstrated that the blockage of the episcleral route by cauterization was still in effect in both the target eyes and the control eyes.

Discussion

IOP Measurements of the Pigeon Eye

In our earlier study,[2] the IOP of pigeon eyes was measured with the pneumatonograph (Alcon), which was the predecessor of the Mentor-PTG. From the analog output of the Alcon-PTG, a plateau is selected subjectively by the examiner as the IOP. In the present study, the IOP was measured by the Mentor-PTG, which is basically the same instrument but the output is digital and the plateau is selected automatically by an internal computer program.

An analog output can be obtained from the Mentor-PTG (A) just as with the Alcon-PTG (B). Figure 8 shows two such successive measurements from a living pigeon eye. The IOP determined from each of the recordings was 25.5 (A) and 24.5 (B) mm Hg and is indicated on the chart. Record A was somewhat noisy but was not discarded. In record B, the plateau is more reliable and indicates a value of 24.5 mm Hg. In the digital mode of the Mentor-PTG, the values of record A and B are of equal importance, but in the case of the Alcon-PTG, only the plateau of the record B would have been accepted by the examiner as a more reliable value. The short plateau of the last part of record A also supports the reliability of the recording. This explains the greater accuracy of the Alcon-PTG and greater scatter of Mentor-PTG in the case of the pigeon eye. Although the digital mode of the Mentor-PTG was used for objectivity, the large increase in the IOP was still readily detectable.

As shown in the last part of the plateau of record B, prolongation and repetition of measurements lowers the IOP of the pigeon eye as in the human eye. Therefore, measurements of the living pigeon eye were limited to four.

Selection of the YAG-Laser

In the earlier study,[2] as well as in the present study, the anastomotic artery running from the iris artery to the aqueous sinus artery was blocked by photocoagulation with a Q-switch YAG-laser while the penetrating artery was blocked with a CW laser. Although the CW laser was also used to try and block the anastomotic artery in the preliminary experiments of the earlier study, we found that while one exposure whitened the anastomotic artery, the artery quickly recovered and became reddish within 1 hour. Additional exposures could not be made because the whitened artery did not absorb the light

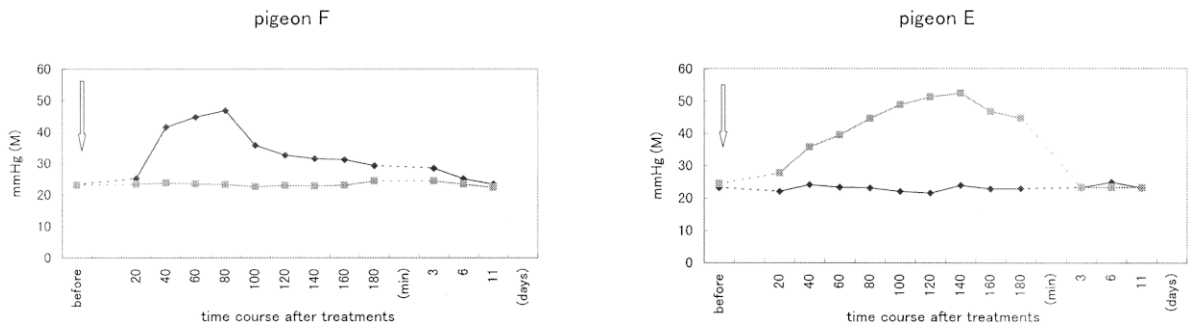

Figure 7. Intraocular pressure course after only laser treatment. Control eye of pigeon F 49 days later and target eye of pigeon E 42 days later. White arrow: YAG- and continous wave-laser treatment for 1 eye, ◆: right eye, ■: left eye.

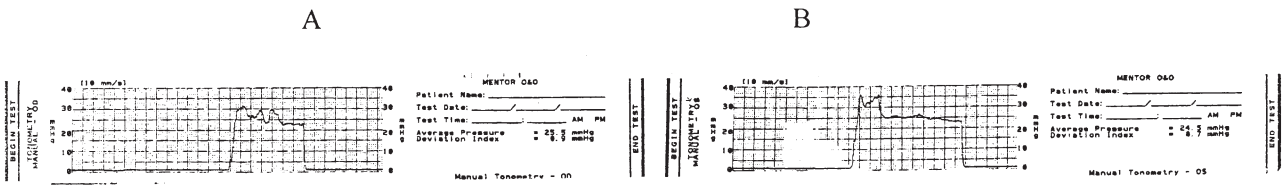

Figure 8. Successive chart-records of pigeon intraocular pressure by Mentor-PTG.

energy. In addition, the anastomotic artery was too short to add additional exposures along its course as in the case of the penetrating artery. Moreover, a misplaced exposure of the CW-laser coagulated the inner side of the sclera, which often lowered the IOP.

The Q-switch YAG-laser, on the other hand, made a pinpoint explosion, and misplaced shots had no effect. Therefore, we adopted the YAG-laser to block the anastomotic artery.

Bleeding Into the Anterior Chamber and the IOP

Bleeding into the anterior chamber following photocoagulation by the YAG-laser resulted in a clot in the aqueous that took up less than one-half of the anterior chamber. This did not elevate the IOP 20 minutes later, perhaps because of rapid hemostasis.

In an eye that was exposed to 6.0 mJ of the new YAG-laser, there was a complete filling of the anterior chamber by leaking blood that led to an increase in the IOP 20 minutes later. A broken iris artery was found during the follow-up study.

These results showed that the existence of a partial blood clot in the anterior chamber did not cause ocular hypertension 20 minutes later in an eye having a sound drainage system. An anterior chamber filled with blood by persistent arterial leakage, on the other hand, might cause ocular hypertension by exposure to arterial pressure or by blocking all of the trabecular surface.

For this reason, less than 5.0 mJ exposure was used in the present study, and the intermediate IOP measurement (procedure 4) was performed to eliminate the cases with elevated IOP caused by heavy bleeding into the anterior chamber. However, none of the cases was eliminated. Consequently the procedure demonstrated that anterior chamber bleeding brought no elevation of IOP. In the normotensive cases, normal IOP was maintained by arterial blood inflow to the aqueous sinus via A-V or A-sinus anastomosis as was found in our earlier study.[2]

After confirming the absence of an increase in the IOP (procedure 4), the A-V or A-sinus anastomotic route was then blocked by episcleral cauterization. The significant increase in the IOP indicated that aqueous drainage required an arterial blood supply to the aqueous sinus artery or to the aqueous sinus.

In this case, the only factors causing an increase in IOP were the aqueous drainage disorder induced by ischemia and the heavy bleeding into the anterior chamber. The theoretical upper limit of the IOP is the systolic blood pressure of the arteries inside the eye because arterial blood flow to the aqueous-forming structures would be blocked at a higher IOP. If there is bleeding into the anterior chamber, the upper limit of the IOP should be the same. The maximal IOP recorded in the target eyes was about 50 mm Hg Mentor-value, which is equivalent to about 80 mm Hg when converted by the equation. This should correspond with the systolic pressure of the intraocular arteries. Although the aortic systolic pressure of *Streptoperia risoria* has not been determined, the systolic pressure of the brachial artery of the rock pigeon, *Columbo livia*, is about 140 mm Hg.[3] Because the ocular-fundus arterial pressure is almost half of the brachial pressure in humans, and the clinically observed maximal IOP in the markedly glaucomatous human eye is approximately 60 mm Hg, a value of 80 mm Hg seems the maximal IOP for the pigeon eye, and may also be the systolic intraocular arterial pressure in pigeons having a brachial pressure of nearly 140 mm Hg.

The blockage of the episcleral veins by cauterization never induced venous congestion and an elevation in IOP. This was probably the case because of the network of veins.

Arterial Blood Supply to the Ocular Anterior Segment

In the human eye, the communication between the anterior ciliary artery outside the eye and the long ciliary artery inside the eye is performed by the major arterial circle in the ciliary body. In the pigeon

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eye, on the other hand, the communication between the penetrating artery from the anterior ciliary artery and the anastomotic artery from the long ciliary artery via the iris artery is performed by the circular aqueous sinus artery.[2]

In order to produce ischemia of the aqueous sinus of the pigeon eye, the external and internal arterial blood supply had to be blocked. In our earlier study,[2] the internal anastomotic artery and the external penetrating artery were blocked by laser coagulation. Ischemia, however, did not result because of the A-V or A-sinus anastomosis from an unknown artery. In the present study, the external episcleral artery was also blocked upstream at the divergence point of the A-V or A-sinus anastomosis. This anastomosis supposedly diverged from some external artery, and not from an internal artery as deduced from the A-V anastomoses of dog[4] and monkey[5] eyes.

In the duckling eye,[6] three anterior ciliary arteries on the episclera, which descend from the ethmoidal artery, the supraorbital artery, and the infraorbital artery, have been found. They were thought to be identical to the episcleral arteries of the pigeon eye that come from anterior, posterior, and inferior collecting portions, which were blocked in the present study. As shown in the episcleral map (Figure 4), the episcleral artery of the pigeon eye is often accompanied by two veins. This relationship might be the reason why the episcleral veins appeared in the fornix at the three collecting portions of the vessels that accompanied the three arteries, and acted as a landmark for cauterizing the arteries.

The internal arterial inflow to the aqueous drainage system was blocked, ie, the anastomotic artery, and the penetrating artery and the episcleral arteries from the outside were all blocked. The consequence was that the IOP of the pigeon eye increased markedly. Blocking only the episcleral arteries with the veins did not cause IOP elevation as shown in the control eyes.

In conclusion, the data indicate that the induction of ischemia of the aqueous sinus of the pigeon eye leads to an immediate elevation of the IOP. We suggest that this ischemia altered the aqueous drainage from the eye resulting in the elevation of IOP. These results suggest that open-angle glaucoma of human eyes could possibly be correlated with an ischemia of the endothelial cells lining Schlemm's canal.

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