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ARGON LASER TREATMENT OF OPEN ANGLE GLAUCOMA

II

Although the efficacy of argon laser therapy for diabetic retinopathy has been recognized for many years, it was not until February 1979, that Dr. James B. Wise and Dr. Stanton L. Witter, writing in the Archives of Ophthalmology, (Vol. 97, page 319), reported successful treatment of open angle glaucoma with laser photocoagulation. Their technique has aroused much interest and seems likely to assume an important part in the treatment of open angle glaucoma. In the last issue of the Glaucoma Letter we described in some detail the mechanism by which laser photocoagulation is thought to work. Since one cannot observe or measure the anatomic changes induced by laser treatment, the theories which purport to explain its efficacy must remain tentative. It it important not to overestimate their validity and to be guided in the treatment of ones patients by observation rather than by theory.

Preliminary reports suggest that laser photocoagulation of the trabecular meshwork is effective in lowering the intraocular pressure not only in open angle glaucoma, but also in pigmentary glaucoma and in glaucoma associated with pseudoexfoliation of the lens capsule. Presumably in pigmentary and pseudoexfoliative glaucoma the normal outflow channels have not been entirely obliterated and traction on the meshwork serves to enhance the facility of outflow in the same way as in open angle glaucoma. Effectiveness of the technique in congenital glaucoma, juvenile glaucoma, and inflammatory glaucoma is unknown. In glaucoma where the angle is closed with peripheral anterior synechias, the technique is inapplicable. In aphakic eyes, laser photocoagulation is relatively less effective. If, however, the patient with cataract and open angle glaucoma undergoes laser treatment prior to cataract extraction, then the effect of the photocoagulation is likely to endure beyond surgery.

The indications for laser photocoagulation of the trabecular meshwork are not yet firmly established, and tentative criteria will no doubt be modified as we gain experience with the technique. The indications for laser trabeculoplasty, let us call it that for the time being, like indications for any other surgical procedure reflect ones estimate of the probability that the procedure will make the patient better against the probability that it will make him worse. It is worthwhile to review our considerations governing this decision in some detail, not only for its own sake, but because it provides an opportunity to investigate this most important of all decision-making processes which so often devolves by rime rather than by reason.

If a discussion of indications for surgery is to be meaningful, the operation at issue must first of all be clearly defined. Such definition includes not only details of the surgical procedure itself, but the details of pre-operative and post-operative management which rival in importance the mechanics of the operation itself. A discussion of indications for surgery implies, most important of all, a definition of the disease to be treated, definition not as a verbal designation, but as a functional description of the pathologic process that requires the surgeon's intervention. If this most elementary of issues is still the subject of debate, this is largely the case because open angle glaucoma is so protracted a process that a precise and detailed history of even a single case still eludes our grasp, and the definitions on which we rely are a mosaic of facets from individual case histories which are assembled into a composite picture of the disease. It is therefore not surprising that different authors reach conclusions which differ sometimes only in emphasis, but on occasion also in the most important of dimensions. It is helpful, as we have suggested in previous issues, to interpret glaucoma in terms of models which define the essential characteristics of the disease.

The purpose of all surgical intervention in glaucoma, of course, is to prevent blindness from the disease or at least to postpone its onset and to retard its progression. Our model of glaucomatous destruction of the optic nerve specifies that the development of blindness in glaucoma is gradual and consists in progressive loss of field. It further holds that such loss of field is a consequence of excavation of the optic disc, and that any procedure which will arrest the development of excavation of the disc will also prevent the development of ensuing field loss. The model further stipulates that the rate of disc excavation is approximately equal to an exponential function of the tangential stresses induced by the intraocular pressure in the optic disc, and that these stresses are proportional to the pressure and the radius of the globe and inversely proportional to the thickness of the disc. A mathematical model with these characteristics was demonstrated in Issue No. 4 of the Glaucoma Letter. Clinical experience confirms the following corollaries: 1. Given a healthy disc, small elevations of pressure are tolerated for long periods of time. 2. The greater the excavation of the disc, the more sensitive it is to damage at any given level of pressure, and the lower the pressure level at which further disc damage is effectively arrested. 3. As the intraocular pressure rises, there is an approximately exponential increase of its destructive effect.

This view of the pathophysiology of glaucoma determines clinical judgment in instituting and advancing all glaucoma treatment. Laser trabeculoplasty must be considered as yet one more therapeutic modality available for the control of intra-ocular pressure. The reasoning that determines at what juncture in the course of treatment laser photocoagulation should be utilized is in essence no different from the reasoning that determines at what juncture one should resort to pilocarpine, or to filtering surgery, or for that matter, at what juncture one should begin treatment in the first place. With each therapeutic step, one must consider the statistical risks of the contemplated treatment and weigh them against the anticipated benefits. When one uses medications, it is easy to determine the effect of the treatment in lowering the pressure, and from that effect, one may reasonably infer the potential benefit in preserving vision. Thus it is the rule to employ one medication or another for a few weeks, assess its effect on the pressure, and determine whether or not it should be continued both in light of its effect on the pressure and in view of any side effects that it might have. With surgical operations, on the other hand, there is no going back, and the operation, once it has been done, for better or for worse, cannot be reversed.

In general, one resorts to laser trabeculoplasty at the same stage in the escalation of therapy at which, before its availability, one would have considered filtering surgery. But it is not correct to conclude that the indications for the two types of surgery are the same. Filtering surgery is inherently, and quite apart from the technique and skill with which it is performed, a proceeding fraught with numerous well-known complications, including peristent flat anterior chamber, cataract formation, scarring of the filtration bleb, and intraocular sepsis. For this reason the decision to perform filtering surgery is always reached with some reluctance, and it is often postponed while there remains a reasonable chance that the patient will retain useful vision without it. The complications of laser trabeculoplasty, on the other hand, are far less grave, and it seems likely that by appropriate modification of the technique, they can be almost entirely eliminated. That is why there are many clinical situations where the risks of filtering surgery are unacceptable yet laser trabeculoplasty seems well advised.

Whenever we consider laser therapy to the trabecular meshwork, we remind ourselves that while the procedure, aside from the transient, and it seems avoidable tension elevation, gives every appearance of being free of deleterious consequences, the operation is new, and it is possible that over the course of a protracted follow-up unanticipated complications may become evident. The theoretical possibility appears remote primarily because of the common observation that scar tissue is stable and undergoes little change even over a span of many years. We weigh these uncertainties against the apparent danger that the patient with elevated, uncontrolled tensions and a damaged disc will, given time, sustain loss of vision. To the best of our knowledge, the risks of the procedure, as well as its effectiveness, compare very favorably with the recognized morbidity and the limited effectiveness of filtering surgery. There is also no reason to assume that unsuccessful laser photocoagulation would in any way prejudice the performance of subsequent filtering surgery. For practical purposes, therefore, when the patient with open angle glaucoma has received pilocarpine, epinephrine, timolol and carbonic anhydrase inhibitors to the extent of his tolerance, and the pressure remains at levels where progressive nerve damage must be expected, a trial with the argon laser would seem not unreasonable.

The technique of argon laser photocoagulation differs from the slit lamp examination of the angle with the Goldmann lens only in so far as one must, in addition to visualizing the angle, adjust the aiming beam of the laser to fall onto that portion of the meshwork which one wishes to photocoagulate. The aiming beam is focused on the so-called filtering portion of the meshwork, about one third of the distance from the scleral spur to Schwalbe's line. It is wise to begin photocoagulation always in the same meridian, perhaps 12 o'clock in the mirror, which is actually 6 o'clock in the angle. One wishes potentially to place about 100 spots around the 360 degrees of the meshwork, one spot for each 3.6 degrees or about 8 spots for every 5 minute sector of the clock; 33 spots in the 20 minute sector which constitutes one third of the angle which is considered the optimal extent at least of the initial photocoagulation. The spot size, selected arbitrarily, is 50 microns; the duration 1/10 second; and the intensity 700 to 1200 milliwatt.

Since the proportion of energy absorbed varies with the amount of pigmentation, it has been customary to increase the power if the meshwork is light in color and to decrease the power if the meshwork is dark. The measure of the efficacy of photocoagulation is then taken to be the appearance of visible changes on the meshwork, a dispersion of pigment, a blanching of the tissue, and perhaps an occasional miniscule intra-trabecular explosion observable through the slit lamp. This effect cannot be achieved in some unpigmented meshwork with power of less than 1000 or occasionally 1200 milliwatts. Nonetheless patients treated with a machine capable of a scant 800 milliwatts seem to do well; it is an instance where the understandable prejudice that more is better deserves to be critically examined. The hypothetical mechanism of action of this therapy suggests that it may be effective even though no change in the treated area can be observed. It is possible, if not indeed likely, that photocoagulation would be effective even where there is no visible destruction of tissue. Scanning electron microscopic studies on an eye whose meshwork was experimentally irradiated with a range of spots from 700 to 1600 milliwatts shows little variation in the morphology of the treated tissue.

The commonly used spot size, 50 microns, is also arbitrary, as is the density with which the spots are placed, the value of 100 per eye reflecting not any discernable physiologic imperative but a numerical property of the decimal system. Wise and Witter recommend photocoagulation of the entire 360 degrees of the trabecular meshwork, and attribute the effectiveness of their protocol in part to the circumferential distribution of the laser burns. Yet, unless one assumes that photocoagulation significantly alters the elastic properties of the untreated meshwork as well, strictly mechanical considerations would lead one to suppose that in some instances at least treatment of only one or two sectors of 120 degrees each would be sufficient. In any event, there is no obvious reason why treatment should not be fractionated, one third or one half of the meshwork being treated at each session, a procedure which recommends itself because it avoids the complication of transient post-photocoagulation tension elevation. In some patients such limited treatment will reduce the pressure to an acceptable level, and for them it is clearly advantageous to restrict treatment to an extent sufficient for tension control at the time. Areas of untreated meshwork will then remain available for photocoagulation in the future, should it become necessary.

Since the visible stigmata of photocoagulation quickly fade, it seems best to refrain from filling in with additional laser bursts a segment already treated, while it is, of course, eminently reasonable to apply additional photocoagulation to a hitherto untreated sector. Because of the considerable risk in photocoagulating a previously treated area of the trabecular meshwork, it is important to keep accurate records of the extent of the treatment, and perhaps also to give a copy of this record to the patient in the event that he should at some future time be advised by a different physician.

Following the treatment, one inspects the anterior chamber for evidence of in-

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flammatory reaction and hemorrhage. Hemorrhage occurs seldom, and is rarely if ever severe. If one recognizes bleeding before the anterior chamber has become too turbid to permit further photocoagulation, one cauterizes the offending vessel with a spot 150 to 250 microns in diameter at 200 to 450 milliwatt for .1 to .2 seconds. If the bleeding cannot be stopped by cautery, one awaits its spontaneous cessation and treats the patient as for traumatic hyphema from any other mechanism. If there is significant anterior chamber reaction, one prescribes a topical corticosteroid. It is better to reserve topical steroid medication for manifest inflammation and not to prescribe it prophylactically, inasmuch as many eyes with open angle glaucoma respond to topical steroids with tension elevation which might obscure the beneficial effect of the laser treatment.

The Wise technique of photocoagulation of the meshwork, as we have noted, is inapplicable where the filtering portion of the meshwork is occluded with peripheral anterior synechias. It may be indicated, though much more difficult, when open angle glaucoma occurs in the presence of a convex iris and a narrow angle. Then the laser beam can reach the meshwork only very obliquely, the perpendicular path of the ray being obstructed by the iris. If one treats a meshwork in this situation, one will in the first place have much more difficulty in accurately placing the spot on the meshwork, inasmuch as geometry dictates that a small amount of rotation of the laser beam will subtend a relatively large meridional arc on the meshwork. Then, too, if one attempts photocoagulation of such an angle one runs the risk of precipitating inflammation of the iris at its extreme periphery and thereby provoking extensive permanent synechial closure of the angle. In this situation, one is left with four options: 1. One may attempt to widen the angle pharmacologically using carbonic anhydrase inhibitors, and possibly osmotic agents, with or without miotics. 2. One may attempt to widen the angle by laser photocoagulation of the iris, a safe distance central to the slit-like angle. 3. One may, if pupillary block appears to play a role, perform a peripheral iridectomy by conventional excision or by photocoagulation. 4. One may reassess the need for photocoagulation and perhaps decide not to perform it after all.

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