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Angle Closure Glaucoma

III

Treatment

Unlike our aim in treating open angle glaucoma, which is solely to achieve permanent control of intraocular pressure at such levels as will prevent destruction of nerve fibers at the optic disc, our concerns in the treatment of angle closure are more complex. Not only do we seek to protect the integrity of the optic nerve, but we wish also to prevent the meshwork and the angle from being damaged by chronic closure, which if allowed to persist, may cause permanent structural damage with consequent intractable tension elevation. Perhaps most important, we wish to protect these and all other intraocular structures from the ravages of an acute attack, which can severely damage not only the meshwork and the angle, but the cornea, the lens, the iris, the macula, and the optic nerve as well. It is seldom possible to predict with certainty what permanent effects angle closure will have on the integrity of the angle and of the meshwork. Other factors being equal, the higher the pressure, the greater the inflammation, and the longer the duration of closure, the more likely that the angle will be permanently damaged. Thus, even when the tension poses no immediate risk to the disc, angle closure glaucoma requires treatment because of the likelihood that without early therapy, irreversible damage to the angle will supervene. In later stages of the disease, when everything possible has been done to open the angle, treatment is reoriented toward preserving the disc and the visual field and then becomes very similar to the treatment for open angle glaucoma.

The threshold question with regard to therapy of angle closure glaucoma is whether, at any given time, treatment should be begun. This issue must be construed broadly enough to include the possibility that therapy should be postponed, in some instances indefinitely, which amounts to saying that in some instances there should be no treatment at all. It is erroneous to assume that every disease should be treated as soon as it is discovered. Sometimes the best treatment is to wait and to do nothing. The patient may not understand. "Do I have glaucoma or don't I?" he asks, thereby implicitly translating the unpredictable issue of pathophysiology into a question of nomenclature. If I have glaucoma, he reasons, it should be treated, and if what I have doesn't need treatment, it can't be as serious as glaucoma. In response to this argument, the ophthalmologist is tempted to coin a new term to designate the disease before it requires treatment and to use a different name for the later stage when it does. Such conceptual manipulation may satisfy the patient's desire for certainty, but it adds nothing to the physician's understanding of the disease. Indeed, the postulate that there are two distinct diseases is misleading when one cannot identify them in practice. The determination whether the patient has "only" narrow angles or whether he has actual angle closure glaucoma, is identical with the decision whether or not the angle closure glaucoma, construed more broadly, now requires treatment. What is important to understand is that angle closure glaucoma, like many another disease, passes imperceptibly from a stage in which treatment would be premature to a stage where treatment is imperative. There are many threatening situations where the best move is to do nothing. In those circumstances, the physician's duty is to stand by, to observe, himself to accept the uncertainty and to make it acceptable to the patient. Because of the the progression of the disease is unpredictable, the physician will, in retrospect, sometimes be blamed for having initiated treatment either too early or too late.

In the case of chronic angle closure glaucoma, there is a period of time during which the disease is developing, and the uncertainty whether it will ever cause the patient any problem is so great that the diseconomies of treatment clearly outweigh its potential benefits. There is a second stage during which the evidence is equivocal and the decision to treat or not to treat is discretionary with the physician. In a third stage where angle closure is the obvious cause of increased intraocular pressure, prompt treatment is imperative and delay would threaten sight itself. It is true that with diligence the uncertainty of when to begin treatment of early chronic angle closure glaucoma can be reduced, but it can surely not be eliminated

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entirely. The need to do something about acute angle closure glaucoma, on the other hand, is never in doubt, provided the eye retains the capacity for useful vision. Once a protracted period of time from the onset of the attack has elapsed, one has much reason to fear that treatment will be too late.

The first task in the treatment of angle closure glaucoma is to try to reopen those portions of angle that have been closed and to forestall closure of any additional sectors. The most obvious means of achieving this goal is suggested by the circumstance that dilation of the pupil may in susceptible eyes precipitate a disease otherwise indistinguishable from angle closure glaucoma. Therefore miotics are sometimes employed to counteract the effects of mydriatic and cycloplegic drops. Pilocarpine is used to constrict the pupil and to try to draw the iris away from the angle. For years this was the favored treatment for angle closure glaucoma and it is on occasion remarkably effective indeed. However, especially in higher concentrations, miotics also tend to make the anterior chamber more shallow and cause the angle to close. Three mechanisms that explain this effect immediately come to mind. In the first place, stimulation of the ciliary muscle tends to displace the lens-iris diaphragm forward. Secondly miosis tends to enlarge the area of iris lens apposition and thereby increase the resistance to flow through the pupil. Such an enhancement of relative pupillary block will push the iris anterior, shallow the chamber and narrow the angle. Finally, any reduction in intraocular pressure will, if all other factors remain equal, in itself cause the lens-iris diaphragm to move forward.

The hydrodynamic link between intraocular pressure and anterior chamber depth has been generally overlooked, probably because it is often masked by other determinants. Intuitive representation of the forces affecting the position of the retina and choroid tends to ignore the circumstance that the intraocular pressure is exerted not only radially but also tangentially to these tissues. The retina and choroid are tethered to the sclera at the optic disc; the choroid is bound to sclera by the vortex veins. When the intravascular pressures within either retina or choroid increase, these tissues expand and creep forward. Conversely, since neither retina nor choroid are tethered rigidly to the anterior sclera, the intraocular pressure, when it rises, tends to push them backwards. It follows that if intravascular pressures remain constant, the lens-iris diaphragm tends to move forward when intraocular pressure falls, and tends to move backward when intraocular pressure rises. This hydrodynamic model is strikingly corroborated when, on occasion, central retinal vein occlusion causes unilateral angle closure. Then, when venous outflow is blocked, capillary and venous pressures rise to the arteriolar level and the expanding vascular bed pushes the ciliary body forward. Corroboration from a different perspective must be inferred from observation of the eye at death. Then, although its pressure vanishes, the anterior chamber nonetheless remains formed, because the pressures in the choroidal and retinal circulations also drop to zero.

The effect of miotics on angle closure glaucoma is unpredictable. The two opposing influences, the widening of the angle from sphincter contraction and its narrowing from the other effects of miotics, make their appearance at variable intervals, so that the same pilocarpine concentration which initially widens the angle may in the course of time cause it to close. It behooves one therefore, if one undertakes to treat angle closure with miotics, to gonioscope the eye before and just after initiating treatment, and to repeat gonioscopy at sufficiently frequent intervals to make certain that the medication does not make the glaucoma worse. The foregoing considerations apply also to the use of miotics to prevent the development of angle closure in an anatomically narrow angle. It is certainly the case that many such angles are dramatically widened by use of pilocarpine, but it is also true that other angles, which, in the absence of medication might not have closed at all, are precipitated into closure by instillation of miotics.

The width of the angle is also affected by carbonic anhydrase inhibitors, presumably because these medications decrease aqueous secretion. Administration of such drugs might be expected to tend to relieve or prevent angle closure, inasmuch as any decrease in aqueous flow would tend to decrease the partial pupillary block that pushes the iris forward into the trabecular meshwork. Thus oral administration of carbonic anhydrase inhibitors by itself is likely to make a significant difference in the width of the angle. Carbonic anhydrase inhibitors also deepen the anterior chamber in the presence of a patent iridectomy, but the mechanism of this effect is not apparent. Their occasionally dramatic effect in relieving angle closure should not lead one to rely on them for permanent control. Many patients become intolerant to these drugs, and even if the medications can be continued, they occasionally lose their effectiveness. When angle closure recurs under such circumstances, one of the most valuable of temporary therapies has been exhausted.

The beta adrenergic blocking agent timolol has a role in the treatment of angle closure glaucoma similar to that of carbonic anhydrase inhibitors. Timolol resembles them in that it also tends to widen the angle, presumably by inhibiting aqueous secretion. Topical epinephrine, on the other hand, which is thought to lower intraocular pressure by a similar

mechanism, is considered inadvisable in the treatment of angle closure glaucoma because the risk of the mydriasis which it causes is assumed to outweigh any benefits of aqueous suppression that it might bring about. The circumstance that both of these drugs very likely affect the tonus and therefore the pressure within the choroidal vascular beds is usually overlooked. Atropine and atropine-like agents, as is well-known, dilate the pupil, push the iris against the meshwork, and often precipitate angle closure. However, these drugs also retract the lens-iris diaphragm. They tend to deepen the anterior chamber, and sometimes, paradoxically, in eyes with malignant glaucoma, they are the only pharmacological tools at the physician's disposal for opening the angle.

Three drugs which transiently increase the osmolarity of the blood, mannitol intravenously, glycerol and sorbitol by mouth, have limited but very specific value in the treatment of angle closure glaucoma. For an hour or two after they are administered, they lower the intraocular pressure by creating an osmotic gradient between the intraocular and the intravascular fluids, transfering water from the former into the latter compartments. The transient lowering of intraocular pressure, by reestablishing the sensitivity of the iris sphincter to parasympathomimetic stimuli or perhaps by other as yet unrecognized mechanisms, sometimes breaks the cycle of positive feedback that has precipitated an attack of glaucoma. The administration of osmotic agents may be repeated, but many patients tolerate them poorly, and there is some danger in patients with cardiac disease, of producing acute heart failure, and in patients with impaired renal function, severe electrolyte imbalance. Osmotic agents at best are temporary expedients in helping to abort an attack of angle closure glaucoma; the patient should be in the operating room before their effect has worn off.

Notwithstanding the availability of medication that may open the angle and reduce the tension in angle closure, the basic treatment of this disease is surgical, the mechanical establishment of a pupillary bypass. This is the case because relative pupillary block is usually the decisive factor in precipitating angle closure, and pupillary block can be relieved definitively only by making a passage peripheral to the pupil through which aqueous may flow into the anterior chamber. Under these circumstances it appears unwise to accept the uncertainties of pharmacological treatment.

When the mechanism of pupillary block was first discovered, the pupillary bypass operation was commonly attempted by puncturing the iris with a needle-knife introduced into the anterior chamber. Because of the proximity of the iris to the anterior lens capsule, lens injury was a not infrequent complication. It was also observed that the pin-point opening made with the needle-knife often closed, and that pupillary block then recurred. For these reasons, iridotomy was abandoned in favor peripheral iridectomy, and for some decades this remained the standard treatment for angle closure glaucoma. The clear disadvantage of peripheral iridectomy is that it is an intraocular operation which requires opening of the anterior chamber, thus entailing the unavoidable risks of infection, hemorrhage, and wound leakage. The argon laser became available to a generation of ophthalmologists who had no experience with iridotomy and were unimpressed by the limitations of this procedure. It is not surprising, therefore, that to their minds the value of being able to perforate the iris without opening the eye far outweighs the disadvantages of a pinpoint iris lesion which not infrequently closes.

Thus surgical peripheral iridectomy has largely been replaced, at least for the time being, by laser iridotomy. The uncritical enthusiasm with which the argon laser "operation" was accepted by the ophthalmologist is a reflection largely of his frustration and anxiety with the established intra-ocular procedure. The laser, this miracle weapon for inner and outer space, lavishly promoted by its manufacturer, attractively styled to enhance the decor of the physician's office, promises to circumvent the difficulties posed by conventional diagnosis and treatment. The purported ease and safety of its use seem to decrease the requirements of conclusive diagnosis. Where it appears so simple to burn a pinpoint perforation through the iris, one is inclined, I think unwisely, to dispense with the darkroom test and proceed with the laser surgery.

The eagerness with which laser iridotomy has been accepted becomes plausible when one reconstructs the perplexity that confronted the ophthalmologist who was required to intervene with surgery for an asymptomatic disease which manifested itself only with an occasional fleeting elevation of tension, and to do so by means of an operation, iridectomy, which, no matter how meticulously performed, entailed the risk, albeit small, of complications that could lead to loss of the eye. It is true that as the experience of the profession with peripheral iridectomy increased, its complications became less frequent and less devastating, but it remained nonetheless a formidable task for an ophthalmologist to advise a patient who had had no symptoms, whose vision was 20/20, and whose only evidence of glaucoma was a transient tension elevation, that the risk of intraocular surgery to both eyes was less that the risk of medical treatment or of no treatment at all.

The cost-benefit ratio of peripheral iridectomy was a function at one and the

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same time of the accuracy of diagnosis and of the complication rate of surgery. If one reserved peripheral iridectomy for those patients who had unequivocally positive darkroom tests or tension elevation in the presence of unmistakable angle closure, and if one then performed the operation without introducing instruments into the anterior chamber and without losing a sufficient amount of aqueous to require its reformation, then the cost benefit ratio was favorable indeed and a large proportion of patients subjected to it were literally cured of their glaucoma.

The risks of iridectomy is least in the patient who has a reactive pupil and whose glaucoma is incipient or has been temporarily relieved with medication. It is useful to premedicate the eye to be operated on with one drop of pilocarpine 2% about one hour prior to surgery, then under retrobulbar anesthesia, to make a 5 mm peritomy at the limbus. One opens the anterior chamber with an ab externo incision near the limbus, just anterior to the projected root of the iris. Before one enters the anterior chamber, a single suture of 8-Ø vicryl is preplaced. The incision, necessarily somewhat longer on the scleral surface, has the shape of a triangle whose apex is at Descemets membrane. As soon as iris, or a drop of aqueous appears in the wound, the incision is complete. Gentle indentation of the posterior lip of the wound will now cause the iris to prolapse sufficiently that it may be grasped with .12 mm toothed forceps. A small segment of tissue is excised. Since pupillary block has now been relieved, further stroking of the edges of the wound will tend to free the iris, and the miotic pupil will pull it into the anterior chamber. In many instances the amount of aqueous lost is so small that no shallowing of the chamber is apparent. The ends of the preplaced suture are threaded through the conjunctiva before the knot is laid down. Two days later the visual acuity will usually have returned to its pre-operative value.

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