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UNIX Workshops

The Cambridge Glaucoma Foundation, Inc. has organized a series of Unix workshops in order to acquaint readers of the Cambridge Glaucoma Letter at minimal cost with the techniques and advantages of professional computing. Unix is the trademark of Bell Laboratories for what is widely acknowledged to be the most versatile and useful computer operating system available today. There is no prerequisite for participation in our workshops except willingness to use a typewriter keyboard. No computing experience is required.

The costs of computing continue to decrease. At the present time the prices of small Unix computers begin at somewhat under $10,000. It is estimated that within four years that cost will have been halved. A powerful and versatile system such as Unix is likely to enable even a small professional practice to save the annual salary of at least one and possibly two clerical assistants.

We estimate that a series of four sessions of approximately four hours each should probably suffice to convey a working knowledge of Unix essentials. Specific times, preferably weekday evenings or weekend mornings, will be arranged to suit the participants. Each participant will have the use of a video display terminal and his own Unix file system. We will provide participants in our workshops with various programs for maintaining financial records, for generating statements of account, and printing out insurance forms. We offer to assist them with modifying such programs to their own uses. We will share with them our experience with various computers on which we have used Unix.

Participants may attend as many or as few of the sessions as they wish and are not necessarily expected to stay for any entire session. Participation is free of charge. Those who have found the workshops useful may wish to make an appropriate donation to the Foundation in return. More information and registration forms may be obtained by writing to the Cambridge Glaucoma Foundation, Inc., 1679 Massachusetts Ave., Cambridge, Mass. 02138, or telephoning 617-868-4666.

Angle Closure Glaucoma - 2

Belaboring the Obvious

One of the most striking examples of the accessibility of the eye to visual inspection is the demonstration of the anterior chamber angle and the direct examination of the trabecular meshwork by gonioscopy. Since the occlusion of the meshwork appears to explain almost all kinds of glaucoma, gonioscopy literally and figuratively gives insight into the mechanisms responsible for this disease. Our understanding of glaucoma derives in large measure from our ability to look into the anterior chamber angle. Gonioscopy answers many questions about glaucoma. It also raises new ones, answers to which are not yet apparent.

Gonioscopy makes possible the inference that tension elevation in any given eye is attributable to closure of the angle. It also makes it reasonable to attempt to arrive at a statistical prediction of the risk of angle closure for any given patient. When the cornea is hazy, gonioscopy may be impossible. Then we manage the glaucomatous eye without knowledge of the width of the anterior chamber angle, and we base our decision to perform peripheral iridectomy primarily on the depth of the peripheral anterior chamber and the value of the intraocular pressure. When the cornea is hazy, we find ourselves constrained to perform some iridectomies which properly interpreted gonioscopic data, if it had been available, would have rendered unnecessary. Unfortunately this is sometimes true also in eyes where the cornea is clear.

The most important determination to be made by gonioscopy is whether or not "the angle is closed". The phrase sounds self-explanatory, and when we analyse specifically what it means, we risk belaboring the obvious. In the first place, the anterior chamber angle, as is well known, is a circular structure. Frequently the angle is well open in some sectors of that circle, while in others, it is narrow or closed. Therefore we must always specify to which portions of the angle our descriptions of angle width are applicable. Furthermore, for reasons not

at all clear, the hydrodynamic effect of angle closure is not a linear function of the amount of angle closed. Thus if less than fifty percent of the angle is closed, the outflow facility of the eye is frequently normal, while if more than fifty percent of the circumference is occluded, the outflow facility is often disproportionately impaired.

A second ambiguity, more difficult to dispose of, flows from the imprecision with which we use the words "open" and "closed." Specifically, when we say that the angle is open, we mean that we are able to see the angle structures at least to the depth of the scleral spur, and that therefore the inner wall of the trabecular meshwork, being exposed to our diagnostic gazes, is necessarily also exposed to the inflow of the aqueous humor. Elementary considerations, however, will make clear that the converse is by no means necessarily true. The fact that we cannot visualize the filtering portion of the meshwork through the gonioscope does indeed raise the possibility that the meshwork is blocked by iris, but we cannot be sure. To clarify this ambiguity, we distinguish between "optical" closure of the angle, which is all that we can properly claim to see on gonioscopy, and hydrodynamic closure of the angle, which we can infer only from the pathologically elevated intraocular pressure. Optical closure implies that the trabecular meshwork is blocked from our line of sight. Hydrodynamic closure means that aqueous is unable to reach the trabecular meshwork. The failure to distinguish between optical and hydrodynamic closure is a major stumbling block to the proper diagnosis and treatment of this type of glaucoma.

To understand the phenomenon of optical angle closure, we must recall some of the details of the optics of gonioscopy and of the surface anatomy of the anterior chamber angle. The view of the angle as of all other intra-ocular structures, takes place through the cornea, the transparency of which ends somewhat anterior to the corneoscleral limbus. The anterior chamber angle is peripheral to the limbus. Therefore one must look into the eye obliquely if one wishes to see into the angle. Indeed, the line of sight is so inclined from the perpendicular, that a ray of light aimed into the apex will be reflected from the cornea by internal refraction. In order to see into the angle, one must artificially alter the refractive properties of the corneal surface. This is accomplished by applying to it a diagnostic contact lens. The Koeppe lens, used in conjunction with a hand-held binocular telescope, serves in effect as a corneal prosthesis that provides a surface sufficiently steep to permit the refraction of the line of sight into the angle. The Goldmann lens similarly neutralizes the refractive surface of the cornea. It contains a mirror in which the angle structures are reflected.

The aim of gonioscopy is to identify the filtering portion of the trabecular meshwork, the area just anterior to the scleral spur, on the premise that if this structure is exposed to the line of sight, it must also be accessible to the inflow of aqueous humor. The task is easy if the cornea is clear, the iris plane flat, and if those well-known landmarks of the angle, the ciliary body band, the scleral spur, and Schwalbe's line, are prominent. Sometimes one can even identify the semi-translucent meshwork itself and follow the narrow slit-lamp beam into its depths. Such an angle is so evidently wide that one may confidently affirm that it cannot close, and forego the distinction between optical and hydrodynamic closure as gratuitous. But if the iris plane is convex and the landmarks are lightly pigmented or not at all, if the angle entrance is narrow and one of the layers of the cornea is optically imperfect, then the identification of optical angle closure may become very difficult indeed, while at the same time the distinction between optical and hydrodynamic closure becomes correspondingly important.

It is helpful to understand why in these circumstances gonioscopy is so difficult. In the first place, the line of sight into the angle passes tangentially to the layers of the cornea. When the line of sight is perpendicular to the cornea, irregularity of the endothelial cells, for example, interferes little if at all with the slit lamp examination of the iris, but when the line of sight is tangential to the cornea, the distortion induced by the endothelial dystrophy makes identification of angle structures all but impossible. Then too, the convexity of the iris may require that the meshwork be viewed from a very acute angle, with the line of sight almost parallel to its inner wall. One must, as it were, look over the top of the iris convexity to see the meshwork "on end." In this perspective, the landmarks fall very close to one another, and may be difficult to distinguish especially if they are but faintly pigmented and the media are not entirely clear. In this situation one tries to shift the position of the telescope if one is using the Koeppe lens, or the position of the mirror of the Goldmann lens, to get the widest possible view of the angle. Often one sees only a single dark ring in the depth of the angle recess, and one is then hard put to say whether it is the ciliary body band that one sees, in which case the angle is not closed, or merely the shadow of the iris on the cornea, in which case the issue of angle closure is unresolved. If, notwithstanding all efforts to look over the iris convexity, one remains unable to see the region of the trabecular meshwork just anterior to the scleral spur, one concludes that in the particular meridian under observation, the angle is

optically closed.

The angle that is optically closed, however, may be hydrodynamically open. We can easily imagine a space between iris and meshwork which inspite of our best efforts we cannot discern. Alternatively, optical closure may indeed correspond to mechanical apposition. The iris may touch the meshwork, but not so firmly that a thin sheet of aqueous might not insinuate itself between the two structures and drain into the Canal of Schlemm. The facility with which water, driven by a pressure differential, will permeate structures impenetrable to light is well known to anyone who has ever tried to stop a leak in the basement wall, and any waterproofing specialist will explain why the simple equation of iris-meshwork apposition with impairment of outflow, which has dominated the theory of angle closure all these years, is remarkably naive. We must ask, to put it more exactly, under what circumstances apposition of the iris to the meshwork will block the drainage of aqueous. The mere touch of iris to the trabecular meshwork does not necessarily suffice to establish a water-tight seal. Only miniscule separation of the two tissues of the order of microns is required to permit the virtually unobstructed egress of aqueous. One could hardly expect to be able to see such separation with the 16 or 25 or 40 fold magnification afforded by goniocopy.

When the iris occludes the surface of the trabecular meshwork, one of two circumstances must obtain: either the iris must have become adherent to the meshwork or forces other than those productive of mere apposition must be at work. When closure is adhesive, there occurs an actual bridging of the iris-meshwork interface with fibrous tissue which, it is assumed, blocks outflow by plugging the surface pores of the meshwork. When closure is appositional, the iris-meshwork interface remains potentially open, and the impairment of outflow at any given time depends on the balance of those forces tending to push the iris into the meshwork on the one hand, and those tending to push or pull the iris away from the meshwork, on the other. The effects on the angle width of illumination and of darkness, as well as of those drugs which decrease the flow of aqueous, and those which dilate or constrict the pupil, are well understood. What we really need to consider, and what we cannot possibly see, is the magnitude of the intrinsic forces pushing and pulling the iris into and away from the meshwork, and how these forces might be affected by incremental changes in the intraocular pressure.

Clinical observations permit us to make some interesting and important inferences. Consider first those situations where a patent peripheral iridectomy excludes the possibility of pupillary block. In "malignant" glaucoma, an increase in the volume of the vitreous cavity, be it from aqueous diversion or anterior rotation of the ciliary processes, pushes the peripheral iris against the meshwork to induce glaucoma. In "plateau iris syndrome" the contraction of the radial fibers of the iris, from physiologic or pharmacologic stimuli as the case may be, is also sufficient to cause hydrodynamic angle closure. We tend to forget that mechanisms other than pupillary block often contribute to close the angle, only to be forcefully reminded of them, when the patient whom we thought we had cured of glaucoma confronts us with a shallow chamber, a closed angle, a pressure of sixty, and a patent iridectomy.

How do we explain the sudden, severe, and uncontrolled escalation of intraocular pressure in angle closure glaucoma? When the iris begins to impinge on the meshwork and the outflow resistance increases, one would expect the rise in anterior chamber pressure to exceed the rise in the posterior chamber pressure, if only by a small amount. With an increase in intraocular pressure, there would be at least a small diminution in the rate of aqueous production corresponding to the so-called pseudo-facility of outflow. Given a constant resistance of flow through the pupil, that diminution of flow would result in a decrease of the pressure differential between the two chambers. Thus one might expect angle closure to cause the pressure to reach an equilibirum at a relatively small enhancement of the normal tension. Perhaps this explains the behavior of the eye with mild chronic angle closure glaucoma. The circumstance that the pressure does in many instances rise so precipitously, and that the iris-meshwork interface acts like a pressure sensitive resistance with positive feedback, suggests that an as yet undefined biological servo-mechanism must be at work.

There are two explanations for the acuteness with which angle closure glaucoma sometimes presents itself. First, we are reminded that in the healthy eye the major resistance to outflow appears to be located in the juxtacanalicular meshwork. We have explained, in a previous issue of the Glaucoma Letter, various mechanisms by which this resistance might diminish when the intraocular pressure increases. If such a decrease in juxtacanalicular resistance with increasing intraocular pressure did in fact occur in angle closure glaucoma also, as well it might, then the as the intraocular pressure rises, the downstream pressure in the meshwork itself will fall, and the iris will be sucked into the meshwork ever more tightly. The second possible explanation is based on the work of John Battaglioli, (The Role of Vessel Collapse on the Flow of Aqueous Humor, M.I.T. Thesis, 1981), who showed that high intraocular pressures can compress circumferentially coursing

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vessels within the sclera to the point where flow resistance increases. It is possible therefore that as the pressure increases, compression of the vortex veins causes congestion of the blood vessels in the choroid and ciliary body. Such congestion would lead to anterior displacement of the lens-iris diaphragm and consequent exacerbation of the angle closure. The two mechanisms cannot be simultaneous, but there is no reason why they might not function sequentially.

It is helpful to reflect on the uncertainties of determining gonioscopically whether or not aqueous has access to the trabecular meshwork, before venturing to predict the risk of future acute angle closure glaucoma on the basis of gonioscopy. Gonioscopy gives a fixed view of angle width because we cannot observe motion of the angle walls. But if one repeats gonioscopy at appropriate intervals, one may observe changes, small and sometimes not so small, in the width of the angle. Fluctuations in angle width do occur, brought about by factors such as variations in aqueous flow, ciliary body tone, pupillary size, lens size and shape, choroidal volume, and, not least, vitreous volume. When the chamber is deep, small fluctuations in the width of the angle are clearly immaterial. When the angle is narrow, however, a small variation in its width may suffice to make the difference between patency and apposition. If iris-meshwork touch does occur, there is no way of predicting whether, within a given interval of time, apposition will lead to an attack of angle closure, or to chronic gradual pressure elevation, or to whether it will turn out to have no functional consequences at all. It is these uncertainties which make the attempt to predict angle closure by means of gonioscopy alone so difficult. That is why, before subjecting a patient to prophylactic surgery, we must obtain affirmative evidence that to the optical closure which we can see, there corresponds hydrodynamic closure as well, and that the meshwork obstruction which we think we can discern is actually accompanied by a rise in the intraocular pressure.

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