anesthesia and did not require retrobulbar or other anesthetic. Table I lists the patients, the number of treatment sessions each had, the total joules delivered during all of those treatment sessions, the initial pressure when considered for treatment, the average pressure drop and duration with each treatment, the pressure at the most recent visit, and the time elapsed since the most recent laser treatment. Glaucoma medications were unchanged throughout the laser treatment period. Patient JED was diabetic and had bilateral neovascular glaucoma. Patients ALD and MSR had central retinal vein occlusions. Patient JBS had neovascularization associated with retinoschisis and retinal detachment in one eye. JSB was aphakic, the other three patients all had their lenses in place. There was no apparent difference in treating the ciliary body processes in the phakic and aphakic eyes.

The first patient had what would appear to be a dramatic drop, however, it should be noted that the other, untreated eye showed a similar drop and, therefore, this is just a fluctuation in this particular patient's case. Review of his record shows a parallel change in the pressure and apparently no long-range effect from his multiple treatments. The second patient had a slight increase in pressure when comparing his initial pressure and the current pressures. The third case had what appeared to be rather dramatic responses but had a detachment of the retina. On reattachment the pressure went up again to pretreatment values. It is possible the laser played a role in the detachment, although the retinal holes did not correspond specifically to areas of ciliary body treatment. The last patient had no significant change in ocular pressure.

Subjectively, all of the patients found this method of treatment comfortable. One patient had received cryo treatments to the ciliary body and felt the reaction to laser was nothing compared to the discomfort after the cryo treatments. However, in our hands the laser treatment was not successful in the long run, whereas, in some cases cryo treatment has been. Both would appear to be temporary. We did not see any change in either the lens or the amount of vascularization during these treatments.

After the treatment of these four patients we have decided to abandon this treatment modality, as in our hands it appears to be ineffective in the long-range lowering of intraocular pressure.

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Electron microscopic observation of a gap at the junction of the ciliary epithelium with the retina in a human eye. J. GÄRTNER.

The membrane of Bruch is considered a five-layered continuous structure between the choroid and the retina. This implies that between the choroid and the vitreous body no direct connection exists. However, in two cases of malformed human eyes, gaps in the membrane of Bruch and in the ciliary epithelium directly at the ora serrata could be histologically demonstrated, through which choroid vessels penetrate into the vitreous base. In a later electron microscopic observation, ciliary capillaries were found to pierce the two nonseparated layers of the ciliary epithelium in an area between a macro- and a microcyst; situated likewise directly anterior to the ora serrata.
The most peripheral retina at the ora serrata shows the well-known transition from the retinal neuroepithelium into the epithelial layers of the ciliary body. There are large cystic spaces within the retina close to the ora serrata. On the pars plana side, vacuoles and small cysts are also present within the corresponding cytoplasm of the nonpigmented epithelia. The nonpigmented epithelia have an extremely irregular, serrated outline. The pigment epithelium on both sides of the ora serrata appears without any remarkable differences. Between the two layers of the ciliary epithelium widened intercellular spaces may be noted. Bruch's membrane and choriocapillaris are structurally very similar on both sides of the ora serrata, as already described by other authors.\(^1\) \(^4\)

In one place, we found at the ora serrata a total interruption of the two layers of the eye cup, extending from the vitreous cortex onto Bruch's membrane (Figs. 1 through 4). At the side of the ora, the wall of the gap is formed by a thin sheet of cells, the staining characteristics of which suggest that they belong to the ciliary epithelium rather than to the retina proper. This suggestion is supported by the occurrence of multilayered stacks of cisternae of the rough-surfaced endoplasmic reticulum within the cytoplasm of these cells; a formation usually not observed within the cells of the retina proper, but very characteristic for the cells of the ciliary epithelium. Occasionally,
Fig. 2. Vitreal opening of the gap. Vitreous body (VB), nonpigmented ciliary epithelium (NPE), and cystic space (CY). (Uranyl acetate. ×5,000.)
Fig. 3. Middle portion of the gap. The asterisk indicates a small space between the neural retina (R) and the monolayered sheet of nonpigmented epithelia (NPE), limiting the ora serrata side of the gap. Microvilli from the nonpigmented epithelium extend into this space. The basement membrane-like material within the gap contains bundles of collagen fibrils (C), large empty cavities, cell remnants (CR), and other sparsely distributed osmiophilic debris (D). (Uranyl acetate. ×5,000.)
Fig. 4. Uveal opening of the gap. Inset: boxed area, higher magnification. In the left upper corner part of a cystic space (CY) of the retina (R). The uveal stroma (S) shows the five layers of Bruch’s membrane. The thickened basement membrane of the pigment epithelium (PE) at both sides of the opening is adjacent to nonpigmented epithelia (NPE), and connected to the basement membrane-like material within the gap. Elastic fiber (EL), capillary lumen (CL). (Uranyl acetate. ×5,000. Inset ×23,000.)
seems possible that as an abnormality in man it was also given 7 for the retinochoroidal occur, the vessels of the lateral portion of the front of this area (i.e., pars ciliaris and pars iridica retinae) are formed by forward growth of the margin of the cup. When this begins to occur, the vessels of the lateral portion of the tunica vasculosa lentis, indenting the edge of the cup in their passage to join the early choroidal vasculature, normally atrophy. Should these branches of the hyaloid vessel system persist, a vascular mesodermal connection from the vitreous to the choroid in the area of the later ora serrata must be the result. This explanation was given for the occurrence of vessels running through the layers of the eye cup in the cases cited above, and it was also given7 for the retinochoroidal venous anastomoses in the ora serrata as described by Daicker. As it is pointed out by Mann, the edge of the cup before closure of the fissure only reaches as far as the equator of the lens and the structures which appear subsequently in front of this area (i.e., pars ciliaris and pars iridica retinae) are formed by forward growth of the margin of the cup. When this begins to occur, the vessels of the lateral portion of the tunica vasculosa lentis, indenting the edge of the cup in their passage to join the early choroidal vasculature, normally atrophy. Should these branches of the hyaloid vessel system persist, a vascular mesodermal connection from the vitreous to the choroid in the area of the later ora serrata must be the result. This explanation was given for the occurrence of vessels running through the layers of the eye cup in the cases cited above, and it was also given7 for the retinochoroidal venous anastomoses in the ora serrata as described by Daicker. It must be stressed that gaps in the cup situated exactly at the point of junction of the retina proper within the ciliary epithelium, and containing anastomoses between retinal and choroidal vessels, normally are present in the West African python, Indian python, and black and white cobra. In her work, Mann11 pictures in Figs. 175 and 176, a possible way in which these gaps might develop. In the normal snake or in abnormal man), by persistence and engulfment by the ectoderm of vessels corresponding with the capsulopapillary vessels of mammals. “It seems possible that as an abnormality in man such foramina might be formed. . . . The end result if the provocative vessel atrophied, would be a hole either at the junction of the pars plana and the retina proper. . . . or farther forward at the root of the iris. . . .” Such a hole should be filled with various components of the connective tissue, especially cell remnants, other debris, and basement membrane material, as is found in the present observation.

Gaps in the layers of the eye cup localized at the ora serrata might be of clinical importance because they are a suitable path for the migration of inflammatory cells per continuitatem. It is known that choroidal infiltrates in sympathetic uveitis, situated in the anterior uvea, show a tendency to penetrate from the choroid into the vitreous body cortex exactly at the ora serrata.12

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