

Fig. 2. (Top) The cellular localization of carbonic anhydrase in the central nervous system; (bottom) a proposed mechanism for the transport of chloride and sodium (see text).

cells in the nucleus of Deiters is the same as that of the corresponding nerve cells (about 0.20 $\mu\mu g/\mu^3$), it is appropriate to make a direct comparison of equivalent volumes of glial and nerve cells.

Studies in which the inhibition of carbonic anhydrase altered the formation and the electrolyte composition of the cerebrospinal fluid implicate this enzyme in its production (9, 10). The principal change is represented by a decreased Cl⁻ gradient in the cerebrospinal fluid (10); the active transport of Cl⁻ is indicated by its concentration in the cerebrospinal fluid against the electrochemical gradient (11). The demonstration of selective high localization of the enzyme in the glial elements of the central nervous system indicates the site where this process may be presumed to act. Figure 2 shows schematically the localization of carbonic anhydrase in the nervous tissue and a possible two-step mechanism for the transport of chloride (and eventually sodium), which can be summarized as follows.

1) Carbon dioxide, which has very recently been recognized (12) as the immediate product of the decarboxylation reactions in the brain, can rapidly diffuse inside the neuron and, from it, into the adjacent glial cells, where it

10 NOVEMBER 1961

is rapidly hydrated to carbonic acid (HCO₃⁻ at body pH) in the presence of carbonic anhydrase.

2) A selective exchange of chloride from the adjacent capillary into the glial cell and from there to the interstitial space and cerebrospinal fluid can then take place.

In this way the high intracellular HCO₃⁻ rapidly made available from CO₂ and H₂O in the presence of carbonic anhydrase may be linked with the active transport of chloride into the interstitial space and cerebrospinal fluid.

This view represents a further extension of the concept of a secretory system (10) localized not only in the choroid plexus but also in the glial tissue. The very small and probably artifactitious amount of carbonic anhydrase found in the neuron may be regarded as evidence that the role of this enzyme in the central nervous system is secretory, as it is in most other sites.

It can finally be pointed out that in the structure studied (the nucleus of Deiters of the rat), the anatomical interrelationship of the glial cells to the neuron and to the capillaries (5) gives further support to the view that the glia has a strategic position (see Fig. 2, top) in the postulated secretion mechanism (13).

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Ocular Lesions Produced by an Optical Maser (Laser)

Abstract. Ocular lesions have been experimentally produced in rabbit by a pulsed optical maser (laser). The high-energy density delivered in a single 0.5 msec pulse was sufficient to cause instantaneous thermal injury to the pigmented retina and iris of the brown rabbit. Ophthalmoscopically, the retinal lesions resembled flash burns from an atomic fireball.

It is well known that the visible and near-visible regions of the electromagnetic spectrum are capable of producing thermal injury to the eye (1). The retina is particularly vulnerable, since the energy focused upon its surface by the refracting media is readily absorbed by the pigmented layers of the retina and neighboring choroid (2). Chorioretinal burns from viewing a solar eclipse or the atomic fireball are typical of the lesions that may result (3, 4).

Recently, extension of molecular amplifier theory to shorter wavelengths has led to the development of optical masers, capable of generating coherent, essentially monochromatic radiation of high intensity (5). These devices, destined for use in communications, the medical sciences, and military installations, constitute another potential source of ocular injury due to accidental exposure.

From a consideration of the emission characteristics of a pulsed ruby maser and the transmission properties of the eye, estimates of the energy density at the retina indicate that the burn threshold may be greatly exceeded by exposing the eye to a single 0.5-msec burst (6). This report describes preliminary studies of retinal and iris lesions in rabbit produced by an optical maser.

A pulsed ruby maser (Vireo I laser developed by Technical Research Group) was employed. The ruby and its helical excitation source were enclosed in a cylindrical housing and mounted on an optical bench. Laser output was 0.1 joule/0.5-msec pulse, emitted in a coherent, monochromatic $(\lambda = 694.3 \text{ m}_{\mu})$ beam, 1 cm in diameter. An adult, pigmented rabbit was held in a restraining box and placed on an adjustable mount with the eye approximately 30 cm from the emission face of the ruby. Pupils were maximally dilated with 2 percent Cyclogyl and 10 percent Neo-synephrine.

In rabbit, regions proximal to the optic nerve head contain medullated nerve fibers that form an elliptical area in which energy absorption is least efficient. When the beam impinged on this area the visible lesion was minimal, but vitreous bubbling was evident. The laser beam was, therefore, directed toward the inferior pigmented portions of the retina. Since ophthalmoscopic sighting was not incorporated into the apparatus for these preliminary trials, the accuracy of alignment could be ascertained only after exposure and subsequent examination of fundus alterations. With practice, however, the desired target site could be readily attained by visual approximation.

Figure 1A shows the ophthalmoscopic appearance of normal rabbit retina in a pigmented region inferior to the optic disk. The pigment is irregularly dispersed. Figure 1B shows the same area in the fellow eye after exposure to a single pulse of the laser beam. There is a relatively discrete, circularly shaped lesion about 3° in diameter. The lesion consisted of blanched, coagulated retina elevated in crater fashion and contained a small, centrally placed hemorrhage. Figure 1C is a photograph of the same lesion 5 days later. The appearance was that of a flat white scar with pigment clumping in and around the area. Figure 1D shows the immediate effects of exposure to three closely grouped successive pulses in another rabbit eye. The fundus changes, al-

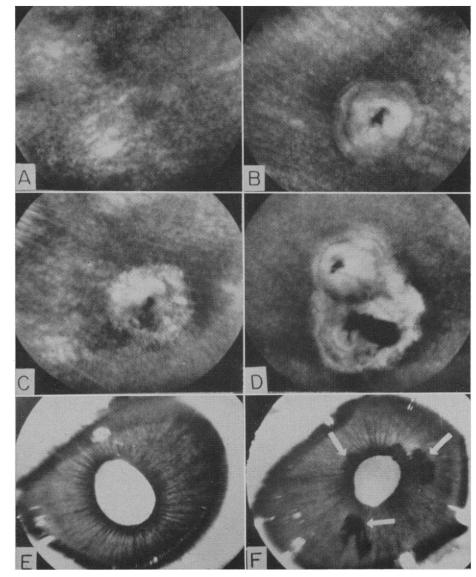


Fig. 1. Ocular burns produced by a laser beam. A, Fundus, showing a portion of normal, pigmented rabbit retina; B, lesion in the same area of the fellow eye following a single exposure to the laser; C, the lesion, 5 days later; D, lesions in another rabbit retina after three exposures to the laser beam; E, normal iris of a pigmented rabbit; F, iris burns produced by multiple exposures of a focused laser beam. Each arrow points to the lesion produced by a single exposure.

though more extensive, appear to be similar to those of Fig. 1B. Again, there were well-defined, elevated lesions. In addition, there was more pronounced pigment disruption, a larger area of blanched tissue, and a large hemorrhage extending into the vitreous. In general, the retinal lesions produced by the laser were profound and reminiscent of those occurring after exposure to atomic bomb explosions (4).

A second experiment was carried out with the pigmented iris of a brown rabbit as the biological target. Figure 1Eshows the normal rabbit iris. The iris of the other eye was exposed to several pulses from the laser. The beam was converged by a short-focus lens and directed to various positions on the iris surface. The results are shown in Fig. 1F. Each arrow points to the lesion produced by a single laser pulse. In every instance the lesion was observed immediately after exposure and was characteristically a dark brown, irregularly shaped burn. When the eye was examined several days later, the pupil constricted in a grossly eccentric manner suggestive of internal damage to the iris.

The ocular abiotic effects described in this report were produced by a coherent source of intense field strength. As amplified light systems are developed and adapted to fulfill military, industrial, and medical objectives, it is essential that attending personnel be fully cognizant of this potential hazard. Investigations are currently in progress to determine threshold levels for the production of ocular lesions.

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SCIENCE, VOL. 134