The picture demonstrated in Fig. C. is less frequently seen. The tear in the iris is more complete, going beyond the root to form a very slight iridodialysis, consequently the iris pillars are more widely separated.

In Fig. D. it is evident that excessive tension on the iris pillars during operation caused an iridodialysis along the whole base of the coloboma. This is the exceptional finding.

REFERENCES

CARBONIC ANHYDRASE AND CATARACTA LENTIS*  
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Problems concerning the genesis of cataract have engaged the attention of innumerable investigators throughout centuries. Measures, taken against the sequels of cataract are as old as mankind and in our days have reached a high degree of perfection. Preventive measures, however, had only success under special, limited circumstances. Cases in which we succeed in protecting individuals who may be considered as to be predestined to acquire cataracts are not very numerous, but in spite of the limited possibilities, benefits are great. Some examples may elucidate this. By the use of adequate goggles we are able to protect people against the injurious effect of radiant energy. When diseases of the thyroid gland make operation necessary, we can prevent lens opacities by taking precautions not to injure the parathyroid glands. When, however, it is not possible to avoid injury to these glands, develop-

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ment of cataract can be prevented by administering irradiated ergosterol.

As prevention of course is always better than cure, we must always endeavour to get to know more and more about the causes of cataract and this means that it is necessary to have a profound knowledge of the normal functions of the lens. Now it is just this chapter of physiology which is rather inadequate, in spite of the fact that a lot of details are known about lens-metabolism (see for instance Bellows' excellent text-book which gives an exhaustive report of these questions).

As it is not yet possible to build up a logical, comprehensive entity of all the known facts, we must be content for the present to gather new material. We may suppose that all causes of cataract depend on interferences with the normal metabolic processes in the epithelium cells of the lens. The lens, suspended in a nutrient medium (aqueous humour), not supplied with blood vessels and nerves, can only maintain its transparency when all the equilibrium mechanisms go on normally. For these physiological activities the permeability of the lens capsule is of considerable importance. It is just the exceptional position of the lens which make it possible to imitate normal conditions in experiments with surviving lenses. The only difficulty is to gather nutrient fluids of adequate composition in sufficient quantities. It may be that this was the cause of the failure of many former investigators. In 1937 my attempts to find a solution for these difficulties were at last rewarded with favourable results. It could namely be established that a Ringer's solution, containing 1 per cent. glucose, injected into a rabbit's abdominal cavity, underwent so many changes in its composition within the short space of two hours, that it resembled aqueous humour very closely. With the aid of this fluid and a special culture technique, known as de Haan's perfusing culture method, I succeeded in keeping lenses alive for several weeks. R. Weekers continued my experiments and obtained many good results in studying metabolic processes of the surviving lens.

This culture method enabled me to point out some characteristics of the enzyme carbonic anhydrase. It is most probable that this enzyme plays an important part in lens metabolism. The lens contains a high concentration of it, sometimes, higher than the blood. As the aqueous humour does not contain a trace of this enzyme, it is almost certain that the lens produces its own carbonic anhydrase. The capsule is probably impermeable to it. In the explanted lenses at least, its concentration does not diminish. When sometimes the lenses in the culture vessel became opaque (a common cause was an infection of the nutrient fluid), the enzyme content decreased.
A normal rabbit's lens has an enzyme concentration of about 5 units; a lens with opacities for instance only 2 units and in cases of mature cataracts no trace of carbonic anhydrase was ever detectable. This was an invariable fact: the more the cataractous changes were pronounced, the lower the enzyme concentration was. I examined cataracts in albino rats, experimentally caused by galactose, naphthalin cataracts in rabbits, and spontaneous total cataracts in rats, dogs, rabbits, guinea-pigs, hens and a pigeon.

The human lens proved to be no exception. Two normal lenses of children had a high enzyme concentration (about the same as rabbits). It is not impossible that the true concentration was still higher, as unfortunately the measurements could not be carried out immediately after the extraction.

In two tetany patients with immature cataracts the carbonic anhydrase content was decreased to two units. In scores of mature cataracts, obtained by intra-capsular extraction, no trace of the enzyme was detectable.

**Conclusion**

A parallelism in the sense of decreased carbonic anhydrase activity coinciding with a lowered transparency of the lens does not absolutely prove that an inhibition of this enzyme is the only essential cause of cataract. On the contrary. All the complicated reactions which are normally and continually going on in the lens, make it probable that a disturbance of even one link of the chain of reactions will coincide with the development of lens opacities. That, however, the catalyzation by carbonic anhydrase is of great importance is undeniable. A striking example to illustrate this opinion is the fact that a drug which inhibits the function of the enzyme (*e.g.*, sulfanilamide), causes cataract.

**REFERENCES**


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