GLAUCOMA AND SEPSIS*

BY

RICHARD KERRY, M.D.

Montreal

Metabolism is carried on mainly by water, which passes out from the arterial and is absorbed back into the venous circulation. As the veins are both much larger and more numerous than the arteries the energy of absorption must be quite considerable to enable venous pressure and the circulation of tissue fluid to be maintained. The aqueous humour, which contains practically no colloid, is readily absorbable and is in contact with abundant ciliary veins, so that inflammatory interference with absorption from the anterior chamber and ciliary body must be a potent cause of hypertension. The structure and relations of the pectinate ligament favour the passage of fluid to the ciliary veins, one long side of the ligament being in contact with the ciliary body, the other with the aqueous humour. The contrast between the open-meshed tissue of the roomy spaces of Fontana and the closely appressed fibres over the canal of Schlemm is quite striking, and one can see that the aqueous humour is continuous with the fluid which permeates the ciliary body. In addition, there is experimental evidence to show that absorption from the anterior chamber by the ciliary veins is quite active. As the iris comes forward the spaces of Fontana will be obliterated before the canal of Schlemm is affected, so that there is good reason to conclude that hypertension is due to insufficiency of the canal rather than to obstruction of it. It is evident, too, that the amount of aqueous humour that can normally pass through the canal alone is too small to prevent hypertension. Trabeculae in the canal oppose quite an obstacle to the movement of fluid, serving, with the pressure in the external veins, to buffer the outflow of the aqueous, and as resistance not only rises with hypertension but is as the square of speed, increased velocity of circulation of the aqueous humour or of the blood can afford practically no relief to the tense eye-ball.

Congestion of the Ciliary Body

It is not usually recognized that inflammatory congestion of the ciliary body is the cause of both hypertension and the shallow anterior chamber in glaucoma. Increased intraocular pressure is due to the tendency for more fluid to pass into the eye than can escape from it. Paracentesis shows that there is no material obstruction to the passage of fluid between the chambers, and, as the intake of the anterior chamber is so very much greater than the outflow, the shallow chamber cannot be due to loss of fluid and can only be caused by the iris being carried forward by the swollen ciliary body. That the shallow chamber is not due to hypertension can be shown by passing a hypodermic needle into the vitreous chamber; if pressure be raised and relaxed the iris will dilate and contract, but it will not advance. This observation has recently been confirmed for me by Dr. Stuart Ramsey of Montreal. Increase of bulk in the eye is due to infiltration by leucocytes and to retention of fluid by inflammatory colloids, largely in oedematous cells, as well as to interference with the outflow, but the movement of leucocytes is so slow that hypertension can be stated in terms of the circulation of fluid. The excess of colloid which is found in the aqueous humour in glaucoma shows that the ciliary epithelium has been damaged and inflammatory fluid forced through it into the chambers of the eye—a further cause of hypertension. A similar condition occurs in the formation and bursting of Schnabel's

bullae, but when they are produced the hypertension is relative and due to maintenance of free leakage from the anterior chamber. If the leakage be established by passing a hypodermic needle into the chamber, the needle will clog in a comparatively short time, showing that the amount of colloid in the pathological aqueous is considerable. That hypertension is due to uveitis is evident from cases of posterior glaucoma, which run their course without cyclitis and practically without increased intraocular pressure.

Specific Infection

The question naturally arises why it is that, with so many types of cyclitis, glaucoma occurs in only one of them. This must be due to specific infection, and the answer is, I think, suggested if one considers the size and consistence of a carbuncle, particularly the length of time required for its resolution, and the effect of brawny swelling of the ciliary body, which may be extensive enough to abolish the anterior chamber upon pressure in the eye. This, of course, suggests staphylococcus infection. About thirteen years ago I reported the results of the bacteriological examination of eight cases of glaucoma, here in Oxford, at one of the many interesting meetings of the Ophthalmological Congress. Seven cases showed a growth of staphylococci, one was negative, and there was no trace of any other germ. Five cases gave a very slow growth, confined to tissue which was removed from within the eye at operation and immediately plunged beneath the surface of an agar slope. We were disposed to consider that this growth was due to an attenuated type which was probably anaerobic. In two cases we could not say whether this strain was present or not as there was a profuse growth of aerobic staphylococci which smothered the tissue and involved the medium. In the last case the tissue was accidentally brought in contact with strong antiseptic, and there was no growth. That contamination should in every case result in a pure growth of the same germ seems to be quite improbable.

Evidence of infection is particularly strong in posterior glaucoma. The reported cases are not characterized by uveitis or hypertension, but by inflammation of the optic nerve with selective absorption of its fibres. There is no resemblance between the flat regular cupping found in uveal glaucoma and the excavation showing great loss of substance but no sign of pressure which distinguishes the chronic cases. The distinguishing feature of posterior glaucoma is diffuse inflammation of the optic nerve, which has been shown in different cases to extend a variable distance up its stem. The conditions present are such as can only be produced by a local cause, and the chronic course indicates that the infection can be comparatively non-virulent. That the infection is not always of low virulence is shown by a case in which the vision dropped from normal to nothing in six weeks, and the symptoms were so slight that even though the patient had been warned to watch her sight, the first thing she noticed was that she was colliding with people and things. This unfortunate woman had had nose and throat operations, but residual infection was neither recognized nor treated and the possibility of reinfection was ignored. These two types of glaucoma represent extremes, but cases showing some characteristics of both must be common.

Glaucoma runs its course like an infectious disease; if left to itself there comes a time when there is neither pain nor inflammation, no hypertension, no recurrences, and no sight. The various types of the disease may be accounted for by the differing virulence of a very variable germ, acting on tissues which oppose greater or lesser resistance to it.

Staphylococci can practically always be found in the nasopharynx, and not only had all the cases reported

* Read in the Section of Ophthalmology at the Annual Meeting of the British Medical Association, Oxford, 1936.

above chronic sinusitis but I was led to investigate the question because for some years I had noted that my glaucoma patients suffered from old nasal disease.

Naturally it will be pointed out that, as catarrhal troubles are so common and glaucoma relatively rare, the former cannot be the cause of the latter. The same argument may be used regarding deafness, but no one considers that catarrhal deafness is primarily an ear disease; infections of the eye and of the ear are practically always secondary. Why these organs become involved in one person but not in another depends principally on individuality, which we cannot analyse, and probably also on the strain of the germ causing the infection. That such involvements do occur is a matter which is much too frequently demonstrated in ordinary experience.

The opposition argument may also be urged. A friend whose opinion must be respected writes that his glaucoma cases show neither symptoms nor *x*-ray signs of sinusitis. I fear, however, that he has not excluded chronic sinusitis, which is almost always ignored and left unchecked, to cause, usually only after many years, its train of permanent disability.

Nasal treatment and sinus operations, the effects of the various rays and high-frequency currents, of anti-staphylococcus serums and toxoids, and of air-conditioning are beyond the scope of this paper, but compulsory use of

the last as a preventive measure, especially in crowded schoolrooms, should be seriously considered. As the dictum of the surgeon of fifty years ago against operating in chronic glaucoma is still sound, some early and effective treatment of the underlying cause must be found if we are to prevent these most distressing cases.

Conclusion

The cause of glaucoma can only be determined by further systematic observation, but with so many indications all pointing to the same conclusion, with no other probable cause of the disease apparent, and with chronic septic infection established practically in contact with the orbits, surely it is not wise simply to ignore the whole matter. It is about eighty years since the nature of glaucoma was first recognized, yet only last year a paper was read which stated: "We do not know the cause of glaucoma." This may be so, but it is not to the credit of ophthalmology, and an adequately organized attack on this problem under control of a central committee is long overdue.

Residual infection and reinfection must be dealt with by the rhinologist, but the prevalence of disease of the upper air passages and its deleterious effect on the chest, eye, and ear make one recognize that air-conditioning as a preventive measure is as important as the treatment of water.

THE SEDIMENTOMETER

A PHOTOGRAPHIC APPARATUS FOR AUTOMATIC RECORDING OF BLOOD SEDIMENTATION RATE

BY

TERENCE LEE, M.B.MELB., M.R.C.P.

MEDICAL OFFICER, PRESTON HALL SANATORIUM, KENT

Recently the estimation of the blood sedimentation rate has become a popular biological test in the evaluation of the general response of the patient to disease. The technique of the test is easy enough, but the chief objection from a practical standpoint is the time needed in order to obtain the readings. General physicians and practitioners are too busy to do this simple yet reliable test themselves, while in institutions such as sanatoria the medical staff has often to rely on non-qualified observers to take the readings. It is on account of this that the test is usually made as simple as possible, and a single reading at the end of a definite time interval, usually one hour, is the only observation taken. On the other hand, to obtain the maximum information the behaviour of the blood sedimentation should be studied throughout the whole time it is sedimenting. Cutler,¹ who has studied this extensively, reads the sedimentation fall every five minutes throughout an hour, and plots the results on graph paper, plotting the time in minutes along the abscissa and the height of the clear plasma above the sedimenting column of red cells along the ordinate. He has found that the more abnormal the blood rate the more rapid is the fall during the early stages.

The apparatus known as the sedimentometer dispenses with the need for an observer, and at the same time will take an accurate and permanent record throughout as long a period as may be considered necessary. The Cutler tube,² as used at Preston Hall, holds one c.cm. of blood

in a column fifty millimetres high with a relatively wide cross-section. This tube is a very convenient one to use in the sedimentometer.

Photographic Recording

The principle on which the sedimentometer works is simple. If a beam of light passes through a column of sedimenting citrated blood it will be interrupted by the opaque mass of red cells below, but will be readily transmitted through the clear blood plasma above. If a sheet of bromide paper placed behind the tube is exposed to this beam of light and then developed in the usual way, the upper part will come out black, while the part with the opaque red cell layer will be white, since no light can get through. The border between the white and black parts marks the height of the sedimenting column at the time. In order to obtain a continuous record throughout the time the blood is sedimenting, the bromide paper must move past the tube at a slow, even speed. A lens placed between the sedimentation tube and the photographic paper corrects the diffusion of light, and a sharp photograph is obtained; at the same time the millimetre markings on the tube are recorded as white lines on the print. It may be noted that such an instrument

FIG. 1.—Photograph of the sedimentometer.

(Fig. 1) will also produce an enlarged image of the blood column if such is desired. At the present moment a new method is being worked out by which a few drops of blood from a needle-prick can be used to estimate the blood sedimentation rate in a very short and relatively wide-bore tube.

In using the sedimentometer ordinary plate-holders are loaded with bromide paper in the same way as one loads plates in the old-fashioned plate camera. The plate-holder is placed in the cross arm at the back of the sedimentometer, and the slide withdrawn from the plate-holder. The clock which drives the plate across the