
Larsson describes a method for rendering the hazy cornea of all but advanced cases of glaucoma temporarily transparent to enable ophthalmoscopy to be performed—a procedure particularly useful in unilateral cases of doubtful aetiology. The cornea having been anaesthetised, strong uniform pressure is applied to its central area for 30-60 seconds by means of a small glass rod with a spherical end, the diameter of which should be 3-4 mm. A clear round spot 3 mm. in diameter results.

ARNOLD SORSBY.


On clinical evidence Salus holds that contrary to the accepted view, the fovea is nourished by a complicated system of fine vessels. He holds, moreover, that the whole of the retina and not only its inner layers is supplied by the retinal vessels. Ophthalmoscopically he distinguishes between vascular changes at the fovea due to (1) senile arteriosclerosis (2) hypertension and (3) arteriosclerosis.

ARNOLD SORSBY.


Ford and Walsh provide in this paper an interesting account of the ways in which eye movements are brought about, so far as the supranuclear mechanisms are concerned. There are five possible sources for stimuli. (1) The posterior part of the second frontal convolution interference with which causes paralysis of some or all of the voluntary movements of the eyes. It is notable that movements and not individual muscles are affected. Thus in a case of loss of voluntary lateral movement to the right, the left internal rectus muscle may still act normally during convergence. (2) The occipital cortex adjacent to the area striata. This regulates "fixation reflexes" and causes the eyes to follow a slowly moving object even when volitional control is lost. If the fixation reflex is lost but volitional control remains, the eyes seem to oscillate about the object of fixation and cannot be fastened
on it for more than a moment. The symptoms are more severe if
the object or the head be in movement. (3) The semi-circular
canals, which are stimulated by movements of the head in space or
more properly by acceleration. The effect on the eyes is to produce
brief contractions of the extra-ocular muscles, designed to maintain
fixation during head movements. In cases of paralyses of voluntary
movements with retention of labyrinthine reflexes, rotation in a
Barany chair, causes deviation of the eyes in a direction opposite to
that of the rotation, while at the end of the rotation, the eyes deviate
in the direction of previous rotation without nystagmus. Loss of
the semi-circular canals reflex results in loss of accurate fixation
when the head is in movement. The test object seems to oscillate
whenever the head is not stationary, but becomes clear when the
head is immobilised, even if the test object is in movement. (4) The
otolith apparatus. This is stimulated by static changes in position
of the head and impulses are sent to the extra-ocular muscles which
result in tonic deviations and rotations of such a type as to compen-
sate for changes in position of the head and to preserve as far as
possible the normal orientation of the visual fields. Changes in
position of the head about a vertical axis will obviously produce no
otolith reflexes. (5) The neck muscles. Apparently impulses from
these produce compensatory eye movements in much the same way
as those from the otoliths. They are of little importance in man
and produce movements of only small degree. A further possible
source of supranuclear stimuli may be located in the orbicularis
muscles since, in cases where conjugate upward movements of the
eyes are lost as the result of a supranuclear lesion, strong efforts to
close the eyes against resistance may cause them to roll up
involuntarily. The paper concludes with the account of two cases
of ophthalmoplegia. In the first, where there was loss of volitional
lateral movements in disseminated sclerosis, the fixation and semi-
circular canal reflexes were still present. In the second, there was
loss of voluntary eye movements in the vertical plane, probably from
a developmental defect; but there was preservation of the otolith
reflexes, the eyes moving upwards or downwards when the head
was respectively flexed or extended. That this was not due to
preservation of the fixation reflex, was shown by the observation
that the movements occurred just as well with the eyes closed.

F. A. W-N.

(4) Brown, E. V. L. (Chicago).—Retroretinal tissue from the
choroid in Kuhnt-Junius degeneration of the macula. *Arch.
of Ophthal.,* June, 1940.

(4) Brown gives an interesting account of the anatomical
findings in a case of senile degeneration of the macula. The patient
was a woman, aged 74 years, who had had defective vision for two
years previously. There were some lens opacities and bilateral simple glaucoma. The right eye had a central scotoma, accounted for by a large mass of tissue behind the macular area of retina. Both eyes were trephined. the left successfully, but the right developed severe irido-cyclitis after three months and was removed. Histologically, the mass of tissue behind the macula was found to consist of a broad stratum of proliferating pigment epithelium with a richly vascularised connective tissue layer separating it from the choroid. In front it was joined by glial proliferation to the retina, in the outer layers of which was a gap. There were many breaks in the lamina vitrea of the choroid (54 were counted) which allowed blood, fibroblasts, capillaries and several larger vessels to enter the tissue between the choroid and retina. Choroidal vessels were badly sclerosed, especially behind the lesion, but the vessels of the optic nerve, retina and anterior part of the uvea were unaffected.

The steps in the development of this condition are probably as follows:—

1. Changed secretion or a marked increase in normal secretion from the choroid results in the formation of a vesicle of albuminous fluid beneath the pigment epithelium which excites its proliferation.
2. Collagenous tissue is then formed from the membrane of Bruch, and glial proliferation also occurs.
3. The mass is pushed forwards by transudate and blood coming through gaps in Bruch's membrane. The rôle of the sclerosis which was found in the choroidal vessels underlying the disc of connective tissue is not mentioned by the author, but the occurrence of such a change is possibly a pre-requisite to the development of stage 1.

F. A. W-N.


(5) Asbury and Vail comment on the rarity of metastatic carcinoma of the iris in the absence of such deposits elsewhere in the uveal tract. They claim that their case is the only complete one on record in which the diagnosis of the primary growth and the affected eye have been confirmed histologically.

From their review of the literature they found seven cases of metastatic carcinoma in the iris. The author's patient was a married white woman, aged 49 years. In August, 1937, her right breast and axillary lymph nodes were resected on account of carcinoma. In November, 1937, her right eye showed ciliary injection, oedema of the corneal epithelium and a spongy yellow-white mass projecting from the iris stroma on the temporal side, 6 or 7 mm. long and 4 mm. wide, and covered with
innumerable small tortuous capillaries. The pupil was displaced nasally and there was an almost complete posterior synechiae. The anterior surface of the lens and posterior surface of the iris were studded with gelatinous exudate and the floor of the anterior chamber was filled with yellowish-white gelatinous and globular débris. Secondary glaucoma was evident.

The tumour was adherent to the cornea in the vicinity of the filtration angle, but Descemet's membrane appeared to have impeded its progress and the substantia propria was not invaded. The carcinoma cells had large round and oval nuclei, a moderate amount of cytoplasm and numerous mitotic figures were present. In the filtration angle the cells were compressed into a spindle shape. Inflammatory wandering cells were evident around Schlemm's canal and in the ciliary body.

The primary neoplasm in the breast showed a scirrhus carcinoma. The patient died on February 2, 1938, from metastases in the brain and stomach.

In discussing this case the authors comment that the only case recorded in the literature in which metastatic carcinoma infiltrated the cornea was one by Kreibig in which the sclera was also perforated. The rarity of metastases in the anterior part of the uveal tract may be explained by the vascular supply which is confined to two long ciliary arteries, whereas the choroid has ten to twenty posterior ciliary arteries. The relatively high incidence of the primary growth being in the mammary gland (60-71 per cent.) is the higher frequency of carcinoma in this organ and its non-vital function.

The authors in discussing the differential diagnosis mention the inflammatory signs evident in some cases of metastatic carcinoma of the iris which lead to a mistaken diagnosis of a tuberculoma or a syphilide.

H. B. Stallard.


(6) Johnson and Eckardt in a previous communication have stated that with diet deficient in riboflavin the most consistent ocular change was the appearance of corneal vascularisation, and their present paper gives some of the reasons for this, also some of the results of treatment with this substance. It is present in milk, liver, egg white and many other foods; but is destroyed by visible light and by ultra-violet rays, and its function in association with other flavo-proteins seems to be to catalyse the oxidation of carbohydrates and of amino-acids. This action is probably carried out, especially
in avascular tissue, by "Warburg's yellow enzyme" of which riboflavin is an important constituent. When there is a failure in the supply of this substance to a tissue such as the cornea, nature tries to make good the deficiency by supplying an alternative method of oxidation with haemoglobin and so grows new blood vessels over the affected part. In addition to its vulnerability to light, riboflavin is also destroyed when the solution containing it is neutral or alkaline, hence the occurrence of riboflavin deficiency in acne rosacea in which there is a lack of gastric hydrochloric acid.

Thirty-six patients with rosacea keratitis were treated by the addition of riboflavin to their diets and thirty-two of them showed prompt healing of the corneal lesions with no recurrences during treatment. The best method of giving this substance was found to be intravenous administration of 1.5 mg. in 5 c.c. of fluid, once to three times daily.

F. A. W-N.

(7) Cogan, David G. (Boston).—Experimental production of so-called bullous keratitis. Arch. of Ophthal., May, 1940.

(7) Cogan considers that bullous or vesicular keratitis is not a disease entity by itself, but a condition found in association with several different pathological disturbances. Since it is non-inflammatory it could more suitably be called corneal vesiculation. It occurs most frequently in association with long standing glaucoma, dystrophies, and post-operative aphakia where the vitreous is in contact with the back of the cornea. It is also seen in connection with some forms of deep seated keratitis. Up-to-date the most commonly accepted explanation of corneal vesiculation is that the fluid is derived from the aqueous, percolating through the substantia propria of the cornea. For this to happen, however, it has been shown experimentally that the intra-ocular pressure must be in the region of 200 mm. Hg, a condition which does not occur during life. A more satisfactory pathogenesis is therefore needed and seems to have been provided by the author's experiments. In his first series he placed a number of freshly enucleated eyes into various hypertonic solutions for three to four hours, and then transferred them to water or normal saline. Corneal oedema and superficial haze developed within a few minutes, and a little later, sub-epithelial bullae formed which gradually increased in size so as eventually to raise practically the whole of the epithelium. The same results were achieved more rapidly by injecting hypertonic solutions into the anterior chambers of enucleated eyes, provided that the surface of the cornea was kept moistened with a solution relatively hypotonic to that injected into the anterior chamber. If the corneal surface were kept dry, vesicles failed to develop. The action is
reversible, because the bullae can be abolished by immersing the eye in a hypertonic solution. It would appear therefore that corneal vesiculation can be due to an increase in the osmotic attraction of the cornea bringing about absorption of fluid from the tears. In this respect, it is interesting to note that Fischer has reported an increased salt content of the cornea in glaucoma.

F. A. W-N.


(8) Under the heading "Acute exudative choroiditis" Duggan includes the usual lesions of acute choroiditis and that known as Jensen's disease or chorido-retinitis juxta-papillaris. The retina over-lying the affected patch of choroid is oedematous and in severe cases, plasma and cells pass into the vitreous and produce opacities in it. The disease occurs principally in young adults who are usually in good physical condition, the commonest time of the year being late winter or early spring. In many cases, in spite of exhaustive tests, no apparent cause can be found and with the usual methods of treatment, cure does not seem to be accelerated. The author states that death has followed fever therapy and the use of sulphanilamide, and that he considers such treatment unjustified in a disease which is self-limited and exposes the patient to no hazard of life and limb. After pointing out that the onset of acute exudative choroiditis is strikingly similar to that of acute retrobulbar neuritis and acute spastic closure of the central retinal artery, the suggestion is made that the origin is vascular and probably due to acute localised arteriolar spasm affecting an area of the choroid and accompanied by increased capillary permeability. The factor which determines the location of a lesion in the choroid, optic nerve, or retina, is an increased sensitivity of the smooth muscle in the arteriolar wall to some substance in the circulating blood; which in the author's opinion is histamine or a substance allied to it.

If this theory be true and the primary lesion be one of localised vaso-constriction, vaso-dilatation is the obvious treatment. The author gives details of five cases (three of them of the juxta-papillaris type) in which this was carried out. One case was unaffected, but the remainder reacted favourably and recovered normal vision in from two to six weeks. The vaso-dilator employed was sodium nitrite, the usual procedure being to give up to fourteen daily intravenous injections of the drug in doses of 0·1 grm. In some cases, the effect was prolonged by administering erythrityl tetranitrate 30 mg. each night by mouth for a further two or three weeks.

F. A. W-N.