CORNEAL THICKNESS IN INTERSTITIAL KERATITIS*

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THE importance of the role played by changes in the thickness of the cornea in the induction of corneal vascularization has recently become increasingly recognized (Cogan, 1948; Ashton, Cook, and Langham, 1951; Langham, 1952, 1953; Ashton and Cook, 1953). However the relationship between the swelling and increase in opacity of the cornea to the rate and progress of vascularization of the cornea in humans has not hitherto been investigated.

In the present study, an attempt has been made to bridge this gap in our knowledge by correlating alterations in corneal thickness with the clinical progress of a series of ten cases of specific and non-specific keratitis receiving cortisone therapy. Changes in the general macrosopical and slit-lamp appearances and in the degree of corneal opacity were noted in addition to the measurements of corneal thickness.

Method

The thickness of the cornea was measured by the apparatus described by Maurice and Giardini (1951) mounted on a Haag-Streit slit lamp. Owing to the opaqueness of some corneae examined, it was found necessary to increase the normal intensity of the incident beam of light. The accuracy of the method decreases with the abnormality of the cornea. Readings of the central area of normal eye had a reproducibility of approximately 2 to 3 per cent. whilst in those taken on opaque corneae an accuracy of approximately 8-10 per cent. was obtained. Similarly the accuracy of readings taken towards the periphery of the cornea was probably not greater than 10 per cent., as the corneal surfaces are no longer parallel.

Results

Values reported for the thickness of the normal cornea are recorded in the Table, which also includes the present authors' observations on a series of normal eyes.

TABLE
 CORNEAL THICKNESS REPORTED BY VARIOUS AUTHORS

<i>Author</i>	<i>Date</i>	<i>Corneal Thickness (mm.)</i>	<i>Number of Experiments</i>
Blix	1880	0.482-0.576	10
Gullstrand	1909	0.46-0.51	2
Koby	1928	0.466-0.703	20
Fincham	1930	0.48-0.59	12
Sobanski	1934	0.40-0.57	20
von Bahr	1948	0.565±0.0077	224
Maurice and Giardini	1951	0.507±0.028	44
Cook and Langham ...	1953	0.536±0.04	10

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FIG. 1.—Specific interstitial keratitis before treatment. This shows a marked localized vascularization of the thickened cornea preceded by a wide zone of corneal infiltration.

FIG. 2.—Same case as Fig. 1 5 weeks later after eight subconjunctival injections of cortisone. Note marked reduction in both extent and degree of corneal swelling.

FIG. 3.—Non-specific deep keratitis before treatment.

The insert in all Figures shows the thickness of the cornea in millimetres.

FIG. 4.—Same case as Fig. 3, 3 weeks later, after five subconjunctival injections of cortisone.

In all the cases of keratitis examined in this investigation, the corneal thickness was abnormally increased and rose to its peak during the more florid manifestation of the condition, slowly diminishing thereafter as the inflammatory process regressed. There was also a close correlation between the degrees of corneal swelling and opacity during the progressive and regressive phases of the disease.

It is of interest to note that in every case in which either superficial or deep corneal vascularization was observed, a significant increase in the thickness of the surrounding cornea was found in association (Fig. 1), a finding strongly supporting the contention of Cogan (1948) that corneal vascularization is always preceded and accompanied by corneal swelling. There was a marked tendency for the thickness of the cornea to be maximal towards the centre, where in some cases the stroma was swollen to at least twice its normal thickness (Figs 3 and 4).

FIG. 5.—Same case as Figs 3 and 4 after a further 3 weeks' treatment with eight subconjunctival injections of cortisone.

In all cases the administration of subconjunctival cortisone was followed by a marked decrease in corneal thickness and in the associated infiltration and vascularization (Figs 2, 5, 6, and 7).

The arrest of the ingrowing vessels was associated with a progressive decrease in calibre of the vessel lumen, with a consequent reduction and eventual cessation of blood flow.

These vascular changes would appear to be directly associated with the reduction in thickness of the peripheral cornea, for in several

FIG. 6.—Non-specific keratitis before treatment.

FIG. 7.—Same case as Fig. 6, 3 weeks' later, after six subconjunctival injections of cortisone.

cases the central cornea remained markedly thickened long after the vascular invasion had become obliterated and the eye clinically quiescent.

These observations would appear to indicate that the influence exerted by cortisone on corneal swelling is more probably mediated through its effect upon the limbal vessels than by a direct influence upon the endothelial and epithelial cells of the cornea.

Discussion

The observations made in this study have much in common with those gained from experimental studies on animals. As in other species there is present in the human cornea a mechanism, dependent on the functional integrity of its limiting epithelial and endothelial cell layers, by which a constant degree of hydration and thickness is maintained. Impairment of this mechanism leads to corneal swelling and opacity and, in certain circum-

stances, to an ingrowth of vessels. A notable feature of the events leading to an ingrowth of vessels into the cornea of animals has been the finding that corneal swelling at the limbus always precedes the ingrowth; there is as yet no experimental evidence to show that vessels can grow into a cornea of normal thickness. In this respect the present findings harmonize well, since a marked swelling of the cornea was found to precede the path of the ingrowing vessels in all cases.

Summary

(1) The thickness of the normal and diseased cornea was measured in a series of ten patients.

(2) Changes were observed in corneal swelling, opacity, and vascularization after the topical application and subconjunctival administration of cortisone. It was found that a marked swelling of the cornea precedes vascularization, and that cortisone inhibits the extension of the corneal swelling and vascularization.

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