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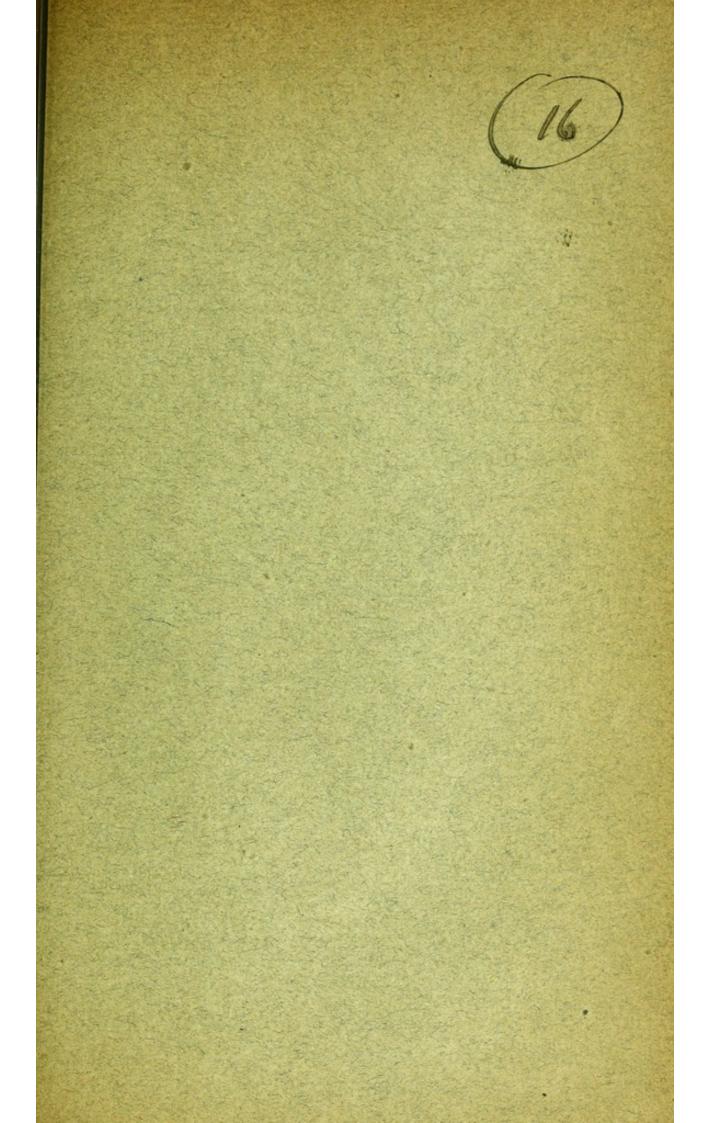
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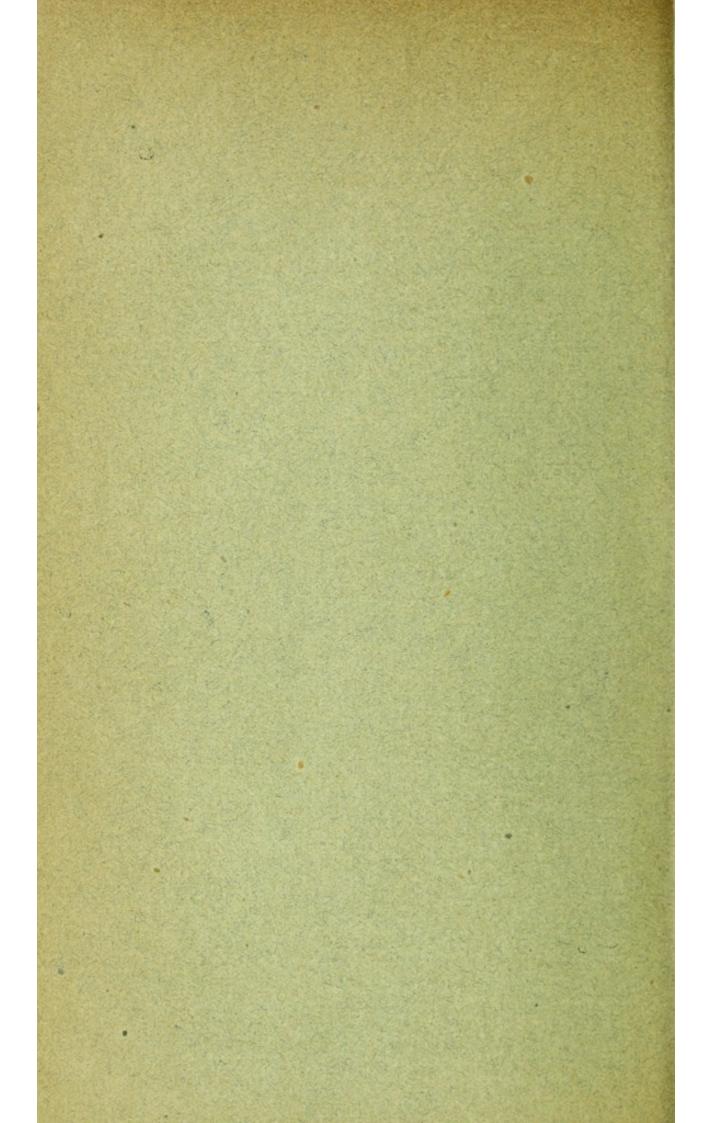
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The physical factor in conical cornea.

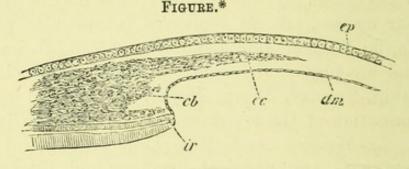
By JOHN TWEEDY.

AT a recent meeting of the Society, in the course of a discussion on a case of conical cornea, I ventured incidentally to offer an explanation of the physical cause of this Though I have during the past ten to twelve disorder. years often referred to this explanation at University College and at the Moorfields Hospital, and described it in an article on "Conical Cornea" in 'Heath's Surgical Dictionary,' published about six years ago, it seemed novel to most of those present. I have been asked to make it the subject of a special communication. In doing so I wish it to be understood that I do not intend to consider the various ætiological conditions of conical cornea, but only that which I regard as the constant physical basis upon which all other causes and conditions operate and depend.

On referring to the text-books in common use I find the various causes given of conical cornea may be arranged into four classes, viz. (1) increased intra-ocular pressure (Graefe, de Wecker and Landolt, 1883); (2) malnutrition and atrophy of the centre of the cornea (Lawson, Nettleship, Swanzy, Berry, Noyes, Meyer, and others); (3) diminution of the resistance of the cornea (Soelberg Wells, de Wecker and Masselon, 1889); (4) inherent weakness, and deficient firmness and thickness of the cornea (Macnamara and H. W. Williams, of Boston). All these explanations are necessarily only conjectural and hypothetical, as is also the explanation I propose. Now it has been laid down by the logicians that the true test of an hypothesis is its conformity and agreement with observed facts. Tried by this gauge, it seems to me that only the fourth of the above hypotheses is tenable. I take it that it will generally be allowed that though there may be in a few cases an increase of intra-ocular pressure,

this state of increased tension cannot be regarded as an essential factor, inasmuch as it is absent in the majority of cases throughout their course. The assumption of a localised malnutrition and atrophy of the cornea seems rather a pathological Deus ex machinâ than a well-founded induction from observed facts. It would be as reasonable to assert that an ordinary ventral or an inguinal hernia is due to malnutrition and atrophy of the abdominal walls. The fourth of the above hypotheses is the only one that agrees with all the clinical facts of conical cornea; namely, an inherent weakness, and want of firmness and thickness of the cornea. If so, what is the cause of this inherent weakness and want of firmness? Neither Mr. Macnamara nor Dr. Williams offers any explanation. This is the precise point to which I invite the attention and criticism of the Society.

I propose that the constant physical factor in conical cornea is an imperfect embryological development and growth of the centre of the cornea; just as the predisposing cause of many cases of inguinal hernia is a faulty development and growth of the abdominal walls and inguinal apparatus.



Section through the eye of a fowl on the eighth day of development, to show the iris and cornea in the process of formation (after Kesseler). *ep.* Epiblastic epithelium of the cornea. *cc.* Corneal corpuscles growing into the structureless matrix of the cornea. *dm.* Descemet's membrane. *ir.* Iris. *cb.* Mesoblast of the iris (this reference letter points a little too high). The space between the layers *dm.* and *ep.* is filled with structureless matrix of the cornea.

There are at least two embryological conditions which may tend to leave the centre of the cornea weak, and are

* Taken from Balfour's 'Comparative Embryology,' vol. ii, p. 408, by permission of the publishers, Messrs. Macmillan & Co. therefore favorable to the occurrence of conical cornea. The earliest rudiment of the cornea consisted of epiblastic cells covering the summit of the primary optic vesicle. These cells elongate, and then the layer folds inwards, forming a pit which constitutes the rudimentary crystalline lens. The involution and subsequent detachment of the lens from the epiblastic cells which are the prototype of the corneal epithelium involve a temporary breach in the continuity of the epiblastic corneal layer. This is a first possible source of weakness of the centre of the When the lens has completely separated from cornea. the cornea a thin homogeneous or sparsely cellular layer is found lying immediately adjacent to the corneal epithelium and in contact with the lens. The origin of this layer is in dispute; some believing that it is a cuticular deposit from the corneal epithelium, and therefore epiblastic in origin, while others believe it to be of mesoblastic descent. Be this as it may, it is not disputed that early in embryonic life this layer is gradually split into two layers by the intrusion into it from the periphery of the mesoblast which forms the choroid and sclerotic. The mesoblast, which then becomes the foundation of the fibro-cellular element of the cornea, encroaches centripetally, so that eventually the apices of the advancing columns of cells meet in the centre of the cornea and coalesce. In the normal course of a development the central portions of the cornea become firmly consolidated, and attain due proportionate thickness and strength. If a check occurs at this stage the centre of the cornea is incompletely evolved, and is therefore left more or less permanently weak. This constitutes a second element of weakness of the centre of the cornea, and it is this element which I believe does obtain in all cases in which conical cornea supervenes, and is the essential predisposing factor. The earlier and greater the check, the more imperfect will be the development and growth of the centre of the cornea, and, consequently, the earlier will the manifestation of conical cornea appear. In some cases conical cornea is even congenital. If the check to development and growth be later and less, the centre of the cornea, though preternaturally thin and weak, may not yield for months or for years until the strain of accommodation is called forth, or until the cornea is further relatively weakened by its increasing expanse in the growing eyeball. A third element of weakness of the centre of the fœtal cornea is to be found in the course and distribution of the blood-supply from the circumference to the centre.

In support of the hypothesis I propose, it may be observed that conical cornea is often associated with other developmental defects, such as congenital lenticular opacities, dental anomalies, coloboma of the optic nerve and sheath, and of the choroid, congenital peculiarities or other morbid states of the skin, such as ichthyosis, psoriasis, acne, eczema. My teacher, the late Mr. Wharton Jones, pointed out that there is often associated with conical cornea a characteristic appearance of the skin, which is dark, coarse, semi-cicatricial, and having large sebaceous follicles.* Further, conical cornea is often inherited, and specially affects certain families.

Three sisters, all of whom have conical cornea, have been under my care from time to time at Moorfields during the past few years.

I submit that this hypothesis of the origin of conical cornea not only agrees with clinical facts, but it is the only one which satisfactorily accounts for the seat and shape of the conicity. It also indicates the mode of treatment. The indications are to flatten and strengthen by cicatrisation that part of the cornea in which the summit of the cone is found. These are the conditions obtained by von Graefe's method; though they are probably more easily and more effectually secured by the modern plan of using the galvano-cautery than by Graefe's original procedure. (March 10th, 1892.)

^{*} A few days after making the above communication I saw a young lady who had conical cornea. Her lateral upper incisors are extremely small, and the uvula was bifid. The other teeth were of natural size and shape. Her mother also has conical cornea.

