tion of pathologists. In my series there are quite gradations in the transition from the large to the small, white, contracted, granular kidney. Examples of a chronic interstitial and glomerular nephritis developing from an acute attack are demonstrated in scarlatina. In a proportion of cases, varying in the experience of different pathologists, the kidney changes are secondary to generalised vascular changes, such as the gouty and the arterio-sclerotic kidney of advancing age; the primary changes are of a chronic inflammatory nature, and limited to the blood vessels and stroma around them; the secondary are degenerative, affecting the glomeruli and parenchyma. In such cases the cardio-vascular changes are primary, and the kidney changes secondary, while in other cases of chronic nephritis, such as the chronic parenchymatous interstitial forms, they are secondary to the disease of the kidney itself.

These cases are the only link disconnected, as it were, from the chain of evidence of the unity in the histogenesis of nephritis as a substantive disease of the kidney. Kidneys the seat of slight interstitial changes of vascular origin, as above referred to, are very prone to intercurrent acute exacerbations; and I have seen three cases where the fatal issue occurred during the acute stage. The kidneys were by no means enlarged.

Regarding the vascular and glomerular changes in Bright's disease, in acute nephritis they are active and proliferative in character, and found in the muscular coat and adventitia of the small arteries, and in the glomeruli, mostly in or amongst the vascular tufts themselves, and in acute interstitial nephritis around Bowman's capsule. In subacute and chronic cases they are found mostly within the capsules in the form of an exudation which compresses or strangles the capillary tufts, or in more chronic cases the latter undergo hyaline degeneration, the intracapsular exudation undergoes organisation, and the tufts gradually become incorporated with the fibro-nucleated thickening of the capsule, constituting the so-called hyaline fibroid or myxomatous degeneration of the glomeruli. Such a degeneration is eminently productive, and not necrotic in character, and expresses a descent from a higher to a lower type of structure. Lastly, we must always bear in mind and carefully distinguish between the productive and degenerative changes in considering the pathology of nephritis in general.

ABSTRACT OF

THE BOWMAN LECTURE

ON

THE PATHOGENY OF CONVERGENT AND DIVERGENT STRABISMUS.

Delivered before the Ophthalmological Society of the United Kingdom.

By Professor EDMUND HANSEN GRUT,
Of the University of Copenhagen.

In endeavouring to understand the origin and true nature of squint, it is of importance to have as definite knowledge as possible concerning the "position of rest" of the eyeballs in emmetropia, and the different kinds of ametropia. The position assumed by a normal eye which is occluded by the hand, while the patient fixes a distant object, is the anatomical position of rest, and is, in the lecturer's opinion, one of slight divergence. The occluded eye may not, however, assume its anatomical position of rest, but may, as a result of habit, remain in the functional position of rest, even though its powers are for the time suspended. This functional position of rest is that which the eyeball assumes in consequence of the desire for distinct vision, and is in emmetropic eyes one of parallelism.

Professor Grut gave the following definitions of his views of the nature of strabismus: 1. Convergent squint originates in, and is maintained as the result of, an innervation which induces in the internal recti a contraction exceeding in amount that which is necessary. When this temporary innervation is temporarily or permanently suspended, the squint disappears. 2. Divergent squint is the expression of a relaxation of convergence innervation, which permits of the eye assuming its anatomical position of rest. It can therefore, in the absence of any paresis or abnormal position of the muscle, only arise when the anatomical position of rest is a divergent one. Thus divergent is the direct antithesis of convergent squint in its mode of origin, as well as in its direction.

Habit and exercise play an important part in the production of the normal movements and positions of the eyes; and their influence in the production of the abnormal positions of squinting eyes is also considerable. The absolute concomitancy of lateral and convergence movements of an occluded eye, to which the ordinary stimuli (fusion of images, etc.) are wanting, is a proof of the power of habit.

Permanent convergent strabismus is capable of explanation by assuming a considerable displacement inwards of the functional position of rest in consequence of the habit of continuous excessive convergence, which prevents complete relaxation of the internal recti. In periodic convergent squint the anatomical position of rest is one of unusual divergence; the squint which occurs on accommodative efforts moves the functional position of rest inwards, so that it nearly coincides with the position of parallelism of the optic axes, and hence, except during strong accommodation, no squint is evident. The necessity for frequent or continuous convergence develops a habit of convergence which cannot be given up; internal squint is, therefore, in a sense a habit, but one which has arisen in the interests of vision, and from which the individual is unable to free himself. Of all conditions inducing this excessive convergence, hypermetropia is undoubtedly that most frequently met with,

In divergent strabismus there is no shortening or contraction of the externi, no innervation leading to excessive action on their part. The condition arises from complete relaxation of the interni, owing to which the functional position of rest gives way to the anatomical position of rest. If the latter is one of parallelism, no absolute divergence will occur. Cessation of convergence does not cause absolute divergence any more than the impossibility of simultaneous divergence (the normal condition) gives rise to convergence. This difference in the pathogenesis of divergent and convergent squint is well shown in the effect of tenotomy upon the two conditions. If divergent strabismus, like its antithesis, were due to increased contraction of the muscle, division of the tendon should give as great an effect as is generally obtained from tenotomy of the internal rectus.

The lecturer concluded with a careful criticism of the various theories which have been brought forward to explain the pathogenesis of convergent and divergent strabismus.

COINCIDENT DEFECTS IN CHILDREN IN ASSOCIATION WITH MENTAL DULNESS, NERVE DEFECTS, AND LOW NUTRITION.

Read in the Section of Diseases of Children, at the Annual Meeting of the British Medical Association held in Leeds, August, 1889.

By FRANCIS WARNER, M.D.Lond., F.R.C.P.,
Physician to the London Hospital.

The object of this paper is to direct attention to the frequency of duplicate defects when the development of a child is abnormal, and the frequent coincidence of mental dulness, nerve defects, and low nutrition.

In 1882 I published[1] 23 cases of gross defects in development, imbeciles and idiots being generally passed over in this group. Among 11 cases of congenital defect of the heart, one had cleft palate, one malformation of hands, one a malformed ear, and three had defects of brain from birth. In two other cases of cleft palate, the head was very small. A case of web fingers showed cerebral deficiency; two intra-uterine amputations (?) showed no known defects. A case of jaundice from birth had double hydrocele. The remaining six cases did not present gross coincident defects; thus 10 cases out of the 23 cases presented obvious coincident defects, the brain being defective in six cases (that is six in 23 cases); it appears then that the brain is often coincidently defective.

I would suggest that it is necessary in a case where some defect in development is found, carefully to examine the heart and the

[1] *Medical Times and Gazette*, January, 1882.