lung, it is well established, I think, that it will go through its regular stages, and in their regular order. Now, of course, the plan of treatment is not essentially different from that of lobar pneumonia, except that there is less need of the active energetic treatment pursued in such cases, because the area of lung involved will be smaller, and because the disease is less sthenic in character. The patient should remain in a temperature that is absolutely uniform, and not far from 70°. A certain degree of moisture will enable him to respire with more ease and with less pain. If sepsis is present the air may be charged with some disinfectant, and for this purpose I have employed listerine very advantageously. Ipecacuanha has no sphere in the pneumonia of adults, if used as an emetic, but as a sialogogue it has desirable qualities, though I use it less than formerly. Chiefly do I depend upon the muriate and carbonate of ammonia suspended in a mucilaginous liquid. I am inclined to think ammonia has a larger field for use than alcoholics, and for two reasons. The first is that it can be employed with advantage for a longer time continuously; and, secondly, because it has a positively favourable action upon the renal secretion, which is a matter to be considered in every form of pneumonia. In addition, I always use the oil silk jacket lined with ordinary cotton wadding, and I keep it in place during the continuance of the disease. The patient is now bathed constantly in perspiration, and there is no likelihood of his taking cold. The bronchitis, if present, is likewise improved.

In some instances I use poultices, which can easily be slipped in under the oil silk. They are to be renewed every four to six hours. A moderate use of calomel in 1-20 grain doses repeated every two or three hours I find an excellent remedy to relieve congestion of the liver and deplete the engorged lung. In uræmic cases we cannot be too prompt in detecting its early symptoms so as to direct our efforts vigorously in that quarter. In some cardiac cases digitalis or some heart stimulant may be imperatively necessary. I am inclined to think that under these circumstances digitalis continues to be our safest and most efficient remedy if administered with caution, and only when necessary.

The following are illustrative cases:—

BRONCHIAL PNEUMONIA IN CONNECTION WITH ALCOHOLIC CIRRHOSIS AND ABDOMINAL DROPSY.

CASE I.—S. C., carpenter, 35 years of age, was admitted into the Presbyterian Hospital in January of 1886. He had previously suffered from varioloid, intermittent and yellow fevers, and had contracted syphilis five years before admission, and was in addition a confirmed victim of the tobacco and alcoholic habits.

Symptoms pointing to thoracic aneurysm, the patient was put upon Tufnell's treatment. At the end of a month it was thought best to order a regular diet, as symptoms of weakness became apparent. Two weeks later a chill occurred without apparent cause. It lasted thirty minutes, the temperature rising suddenly to 103° F., and an hour later to 105° F. The fever was at first reduced by antipyrin in 30-grain doses. On the second day the temperature rose to 105° F., but was this time reduced to 102° F. by antipyrin (grains xv) in repeated doses with thallin (grains iv). The patient then began to expectorate blood, and on the same day bronchial breathing was detected in the left lower lobe. The patient died on the fourth day of his pneumonia, and apparently of heart failure. At the *post-mortem* examination it was found that the heart was somewhat hypertrophied and that its cavities were dilated. Weight of organ 15 ounces. The aorta and large vessels were quite free from apparent disease. Both lungs were firmly bound down by old pleuritic adhesions, and were congested and oedematous, the right weighing 30 ounces; the left, 27. Both lungs contained areas of lobular pneumonia consolidations. The liver was enlarged, cirrhotic, and pigmented. The peritoneal sac contained a large amount of serous fluid. Here there was a case of alcoholic cirrhosis with abdominal dropsy, where the cause of death was due to bronchial pneumonia. To be sure the patient had gone through many of the phases of constitutional syphilis, but none of its lesions were detected at the necropsy. In such an instance what produced the pneumonia? An answer will only be conjectural.

BRONCHO-PNEUMONIA IN CONNECTION WITH DIPHTHERIA.

CASE II.—The following case is interesting as showing how broncho-pneumonia may be the final lesion in diphtheria. In March, of 1887, I made a *post-mortem* examination in the presence of Drs. J. E. Janvrin, E. H. Peaselee, W. H. Porter, and C. E. Billington, on the body of a lady who had been suffering for about

a week with diphtheria, taken presumably from the grandchild that she had been nursing. She died suddenly with suffocative symptoms, and the following lesions were found. In the trachea from immediately below the thyroid cartilage to the smallest divisions of the bronchi, a membranous exudation, diphtheritic in character. It was thick and tolerably adherent to the mucous membrane, forming a veritable cast of the tubes. No membrane in the buccal or nasal cavities, or in the larynx. Here there was one of those very rare examples of truly diphtheritic bronchitis. The lungs both right and left were studded with zones of bronchial pneumonia in the stage of red hepatisation. The heart was enlarged, fatty, and dilated. These areas were so small and so scattered, that they could hardly have been detected by physical signs. The pneumonia in this case might not have been a very serious affair, had there not been fatty heart, and profound blood infection from the diphtheria.

THE HISTOLOGY OF BRIGHT'S DISEASE.

Read in the Section of Pathology and Physiology at the Annual Meeting of the British Medical Association, held in Leeds, August, 1889.

By GEORGE F. CROOKE, M.D.,

Physician for Out-Patients to the Queen's Hospital, Birmingham, and lately Pathologist to the General Hospital, Birmingham.

THE material examined consists in considerably over a hundred individual cases of nephritis of all forms, and represents the work of ten or twelve years; but the subject of the paper includes only Bright's disease proper; all the suppurative and other forms of kidney mischief, due to local causes, are purposely excluded.

The examples of acute nephritis number sixty, of which only three were instances of an idiopathic affection, the rest being associated with other diseases mostly of a specific febrile nature.

While rejecting the term parenchymatous in Virchow's strict meaning of it, I am disposed to retain it for those cases where the parenchymatous changes undoubtedly predominate. Interstitial changes are undoubtedly also present, but in so slight amount that they might in places easily be overlooked. I have met with a number of such cases where the parenchymatous changes constituted the main anatomical features. I quite subscribe to Cohnheim's classical dictum that glomerular changes alone may be responsible for the cardinal symptoms of Bright's disease, and in a few cases these changes may be so prominent and extensive that the nephritis merits the title of acute glomerular nephritis.

The term acute diffuse nephritis I apply to those cases of nephritis where the parenchyma, the stroma, and the vascular apparatus are almost equally affected, and the extent of the changes indicates the intensity of the inflammation. Cases of acute nephritis of a hæmorrhagic or septic character usually show these changes.

Then there are cases where the stroma is principally affected by a round-celled infiltration, and these I term acute interstitial nephritis. Cases have been described by Dr. Coats (our President), by Biermer, Wagner, Klein, myself, and others in scarlatina.

Coming, now, to chronic nephritis, I take the large white kidney first. As regards its histogenesis, it develops as a chronic nephritis usually in connection with diseases of a subfebrile nature, such as phthisis and chronic suppuration, and it is in these examples that amyloid changes are found associated with the parenchymatous.

Regarding the relation of the two, I coincide with the view recently expressed by Dr. Saundby in his book, that the two processes are concomitant or co-ordinate, and traceable to the same cause. In other words, so far as my observation leads me, the extent of the amyloid changes found in some of these cases is so slight in comparison to the extent and intensity of the parenchymatous, that the two cannot be regarded in the relation of cause and effect. This view has been more recently expressed by Orth in the first part of his second volume on *Pathological Anatomy*. Cases, however, are met with where the organ of the chronic parenchymatous nephritis is distinctly traceable to an acute attack, or to the effects of, perhaps, several intervening attacks. Other noteworthy histological features in cases of subacute or chronic parenchymatous nephritis are the glomerular and interstitial changes present, which have previously attracted the atten-

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 published¹ 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 coincidentally 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.