of the burns and scalds; but in only two cases has sepsis occurred of a sufficiently severe nature to call for the admission of the patients to the wards, and in both these cases cleansing and re-application of the tannic acid dressing has led to an uneventful recovery.

I venture to think that this simple method of compress may be of universal application both by the general practitioner, in the out-patient departments of hospitals, and as a first-aid treatment in works and in the home, presenting, as it does in its simplicity, great advantages over the more complicated method of reinforced compresses, recently advocated by Messrs. Hunt and Scott¹² and Wilson.¹²

CONCLUSIONS

I have endeavoured to trace the progress of the treatment of burns and scalds during the past forty years, and to show that, although improved social conditions have done much to reduce this mortality, yet improved treatment has done more—especially the introduction, or should I say rediscovery, of the value of tannic acid in this relationship.

I have described a simple method of using this tannic acid. Even if you do not consider it suitable for universal application, as I hope you may, yet you must admit that its use in reducing the mortality of severe burns and scalds to 4 per cent. and 1.7 per cent. respectively at least redounds to the credit of the local treatment, for social conditions remain the same as in the preceding five years, when the mortality was considerably higher (15.5 per cent. and 7.5 per cent.).

Further, I would urge the necessity of separating the figures relative to the treatment of burns from those of the treatment of scalds in articles dealing with this subject.

I wish to tender my thanks, in addition to those already mentioned, to my successive house-surgeons, Messrs. R. W. Butler, R. O. Lee, T. H. Wilson, R. H. Franklin, and T. W. Mimpriss, and especially to Miss Routh (sister of the septic wards) to whose patient care and continued enthusiasm so much of the success of this treatment has been due.

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RECENT CONCEPTIONS OF BRIGHT'S DISEASE AND ITS TREATMENT*

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My intention is to attempt to summarize the trend of opinion and lines of advance in our knowledge of Bright's disease since the beginning of this century. An enormous mass of information has been added to the subject of kidney disease in the last twenty-five years, but it must be admitted that most of the problems are still unsolved, and this knowledge has not yet had the simplifying effect which comes when theories are proved and hypotheses become facts. It is only possible to touch upon a few of the problems, and I therefore intend to discuss only questions with a direct practical bearing, and to omit reference to the numerous theories and more academic problems.

*Extract of paper read at the Leeds and West Riding Medico-Chirurgical Society, October, 1932.

My main impression as a student twenty-five years ago was that the clinical teaching was entirely based on the end-results of kidney disease. One thought only in terms of gross structural changes, and every endeavour was made to correlate the clinical picture with the various forms of anatomically altered kidneys. All albuminuria was regarded seriously, though so-called physiological albuminuria was mentioned as a real, but rare, occurrence. Degenerative kidney disease, as distinct from inflammatory infections, was not recognized. Nor was the possibility of the occurrence of a reversible reaction considered. The infective origin of nephritis (apart from scarlatinal nephritis) was scarcely mooted at that time, while the conception of a kidney affection as "a disease of the body in which the kidneys suffer" was unknown.

One of the most important advances has been the change in attitude towards albuminuria *per se*. Up to the time of the war there never had been any attempt on a large scale to find out the incidence of albuminuria in adults, although several casual investigations on inmates of institutions had revealed a surprisingly large number of albuminurics. During the war an investigation was carried out by MacLean on 60,000 men, physically fit to undergo military training; 5 per cent. (3,000) of these showed albuminuria—2 per cent. marked, and 3 per cent. slight in degree. It is also to be noted that the investigation was carried out on early morning samples of urine. If specimens had been collected after exercise the percentage of albuminurics would probably have been doubled. Albuminuria must therefore be regarded as much commoner than previously supposed. Moreover, the after-history of these men while on active service revealed no higher incidence of nephritis or of other affections than in the 57,000 free from albuminuria. Doubtless a considerable proportion of these cases were due to affections of the lower urinary tract, while many were of an age at which functional albuminuria was probable. But the investigation goes to show that albuminuria should not be regarded seriously in the absence of definite signs of organic disease, especially of the cardiovascular system, and where excess of casts has not been demonstrated in the centrifuged sediment of urine.

The attempted treatment of albuminuria is worthy of brief mention. Frequently patients are referred to the out-patient department because a small amount of albumin persists in spite of all efforts "to clear it up." The deleterious effect of a loss of a small amount of albumin is of no moment—even the loss of much larger quantities is no great drain on the protein of the tissues. A urine of average output which is coagulable on heating means merely the daily loss of the amount of protein contained in one egg—approximately only one-tenth of the minimal total protein intake. Moreover, as Geoffrey Evans puts it, the residual albumin from a previous kidney affection may have no more significance than the scar of a perfectly healed wound in the skin. Such albuminuria often disappears if and when the general metabolic disturbance clears up and the renal tissue has not been so irrevocably damaged that recovery is impossible. Otherwise, in spite of all therapeutic measures, the proteinuria will become permanent and persistent. It is not uncommon to see cases of chronic albuminuria with a persistent slight general oedema which disappears within a week when the patient is recommended to take a diet containing an average protein ration. Such oedema is in fact a starvation oedema, due to the individual having been restricted to a low protein diet for months in a mistaken and futile attempt to "clear up" the slight persistent albuminuria.

SECRETORY CLASSIFICATION

Perhaps one of the greatest advances has been the change of conception of Bright's disease as a condition of altered kidney structure in response to a disorder of the body chemistry of which the kidney lesion is a local manifestation. In recent years much attention has been focused on this biochemical conception. Before the war a certain amount of work was done on this aspect of the question by the French school, notably by Widal and Ambard, but little practical application had resulted. Since then, as a result of the introduction of tests of renal functional efficiency, and of the relative ease of estimation of the blood urea content by newer and simpler methods, considerable attention has been devoted to a secretory classification of Bright's disease in contrast to the anatomical one, and the nomenclature of the French school has come into vogue, namely:

1. *Hydraemic Nephritis (or Water Retention Type).*—Profuse albuminuria; marked oedema; retention of chlorides; normal blood urea; normal blood pressure; no cardiovascular changes.

2. *Azotaemic Nephritis (or Nitrogen Retention Type).*—Slight or no albuminuria; no oedema or salt retention; increased blood urea and blood pressure with cardiovascular changes.

But it was soon found that the clinical problem was far too complex for such a rigid biochemical classification. The percentage of nephritics who fall definitely into one or other group is relatively small, with the result that a third class—and this by far the largest class—"mixed nephritis," had to be introduced. It is now recognized that this classification has marked limitations, and that it represents only one aspect of the problem. It has, however, introduced a useful, practical conception, especially in its bearing on treatment. If there is no nitrogen retention, as shown by a normal blood urea content, then there is no reason why the protein of the dietary should be unduly restricted. Again, it also helps to clear up some of the confusion as to the indications for a salt-free diet. Such a diet is most valuable in the treatment of the oedema of hydraemic nephritis; on the whole, it is practically useless in cases of azotaemic nephritis, and indeed may do positive harm if adhered to for more than three weeks at a time. Its insipid nature diminishes the appetite, while even more important is the fact that its long-continued use leads to a condition of chronic ill-health probably from uncompensated acidosis.

Brief mention of renal function tests is perhaps appropriate here. The first and most popular was MacLean's urea concentration test, which has been in vogue for fifteen years. There have been various attempts to increase its delicacy, notably Calvert's range of function test, while more recently there has been introduced van Slyke's range of clearance test. There is no doubt that these tests have been of considerable value in certain types of cases—for example, in the recovery stage of acute nephritis, especially if recovery is delayed—in estimating the degree of residual kidney damage. Perhaps they have been of most value to the surgeon, who merely requires an opinion as to whether renal function is so grossly impaired as to contraindicate or delay operation. But from the physician's standpoint they have marked limitations. They give no indication as to the nature of the morbid change which is affecting the functioning tissue; and it is the nature of the lesion, and not its extent, in the early stages of nephritis at least, that is one of the physician's chief guides to prognosis. Moreover, these tests give equivocal results in the very type of case in which the clinician also is in doubt—

for example, functional albuminurias, or early cases of suspected granular kidney. This is scarcely surprising when we recollect that experimentally two-thirds of the renal tissue can be removed before any gross defect in renal function can be detected by biochemical means. Finally, a word of caution must be uttered, for certain dangers seem to lurk in the very attractiveness of these tests. More attention is apt to be paid by students to the estimation of the blood urea, etc., than to the simpler procedures which can be carried out by himself—for example, the detection of tube-casts, or the estimation of specific gravity in nocturnal and diurnal samples of urine; while again, if these tests are once carried out and found satisfactory in the presence of a lesion too early to be detected by such methods, its gradually progressive character may be ignored by the practitioner. The albuminuria of pregnancy is perhaps a case in point.

Attention must be drawn to the usefulness of blood urea estimations in cases of latent uraemia (apart from the more acute type associated with anuria). In conditions with a long history of progressive languor, easily induced fatigue, loss of appetite, perhaps anaemia, but no albuminuria, and probably no cardiovascular changes, a blood urea content of 100–200 mg. per 100 c.cm. may be present, and such an estimation may be the only means of confirming the diagnosis, which otherwise would merely be a matter of surmise. Although the blood urea content is, in this way, of definite value in determining the presence of a uraemic state, yet it is the consensus of opinion that the retention of urea is not the cause of uraemia. Indeed, this neurotoxic condition appears to be a morbid state due to various disturbances of the body chemistry of which the manifestations of renal disease are themselves only a part. Although the problem of uraemia is still unsolved, yet there has been an advance of practical importance in recent years in the differentiation of the group of cases due to cerebral oedema. It is a well-known fact of clinical experience that the convulsions and amaurosis of acute nephritis are more alarming, though much less serious, than the quieter type of neuro-toxaemia seen in cases of chronic nephritis. The prognosis is much better if only the patient survives the convulsions. These appear to be of mechanical origin, associated with cerebral oedema. Lumbar puncture has been found to be dramatic in its relief of such convulsive seizures, whereas the evacuation of cerebro-spinal fluid is worse than useless in the treatment of the milder convulsions and twitchings of chronic uraemia, which is a neuro-toxaemia, in contrast to the neuro-oedema of the former condition.

As for acute Bright's disease, the most important practical point which has been emphasized in recent years is the clear-cut distinction in children and young adults between acute diffuse nephritis and acute haemorrhagic nephritis or so-called "essential renal haematuria." The latter is much the more common affection; malaise, pyrexia, and oedema are all very slight, while the blood pressure is unaffected—indeed, except for the definite haematuria (often recurrent) the absence of physical signs and symptoms is a distinctive feature. These cases are practically always streptococcal infections—tonsillitis, sore throats, antral infections, middle-ear disease. The condition clears up after treatment of the septic focus, and usually no permanent damage results. Acute diffuse nephritis is a much less common affection, associated with definite pyrexia, dropsy, oliguria, profuse albuminuria with probably a raised blood pressure. Its aetiology is doubtful, although probably also infective, and it is a much more serious affection both as regards immediate and ultimate prognosis. It is the failure to differentiate

these two types which has led to the diverse views frequently expressed as to the prognosis of acute Bright's disease in children.

THREE GROUPS OF CASES

Although it is my intention to avoid any attempt at classification of renal disease (and for this reason have adopted the non-committal title of Bright's disease), yet the recognition and differentiation of three reasonably distinct groups of cases is probably of help both as to prognosis and as to treatment.

1. *The Septic Group*.—Perhaps the best example is the acute haemorrhagic nephritis discussed above. But chronic sepsis is no doubt also an important aetiological factor, the type of lesion resulting therefrom depending on the renal element upon which the brunt of such sepsis falls. Hence in the heterogeneous group of conditions called chronic nephritis every effort must be made to eradicate any foci of latent sepsis.

2. *The metabolic group*, of which the best example is the chronic interstitial nephritis, or red granular kidney of classical description. Although the textbooks make much of gout, lead, diabetes, etc., as important causal factors, yet there seems little doubt that nowadays the basal pathology of the condition is best expressed by the term "goutiness" of Leonard Williams. "It is that condition of ill-health due to insufficient or perverted metabolism leading either to the formation of material which is foreign to the healthy economy or to the inadequate discharge of normal excreta." In short, it is due to a want of balance between intake and output. This metabolic group is usually associated with high blood pressure, and probably both the kidney lesion and the hypertension have the same aetiology. But further discussion of this subject is outside the scope of the present paper.

3. *The extrarenal group* is worthy of recognition, if only to draw special attention to that type of generalized affection of which the kidney lesion is merely a local expression. During the war the occurrence of famine or starvation oedema in Central Europe focused attention on the extra-renal origin of oedema as distinct from the simple mechanical theory of renal obstruction—nephritis being the cause of anasarca—a view which had persisted since the time of Bright. The trend of recent opinion is that so-called renal oedema is not a direct consequence of renal injury, but is merely a local manifestation of widespread changes in the tissues, the cause of which is still unknown.

In recent years the literature of Germany and America abounds in discussions on nephrosis, as distinct from the disease usually recognized as chronic parenchymatous nephritis, but the introduction of this term has not been received very favourably in this country. It was first introduced by Müller in 1905 to designate those cases in which the main feature is degeneration of the tubules as contrasted with inflammatory conditions of the glomeruli. The distinguishing features of nephrosis are largely negative in character—namely, (1) absence of renal inadequacy and of tube-casts in the urine, although there is a profuse albuminuria; (2) no increase in blood pressure; and (3) no cardiac enlargement—while the most interesting positive characteristic is the great increase of cholesterol in the blood. Further, to quote Evans, "their striking feature is the complete recovery that is made for no apparent reason after months of persistent oedema, marked albuminuria, and a general condition of severe illness. Nothing like this is seen in any other form of Bright's disease." Many such cases in children and young adults have been recorded, mostly of severe type, while there are probably numerous milder affections which clear up without their relation to this group of lipoid nephrosis being recognized.

Apart from this sharply defined clinical entity, degeneration of the renal tubules is usually associated sooner or

later with glomerular inflammatory changes, and therefore much confusion has resulted from the use of the term "nephrosis" by foreign workers to denote the condition generally recognized in this country as chronic parenchymatous nephritis. It should be added, however, that the introduction of this conception of nephrosis has been useful in directing attention to the importance of widespread tissue changes in affections associated with general oedema and profuse albuminuria, while it also emphasizes the fact that an apparently gross kidney affection may be coterminous with its extrarenal cause.

TREATMENT BY DIET AND ALKALIS

Very little space is left for the discussion of treatment. Some of the main advances have been already indicated in the discussion on albuminuria and uraemia, on the biochemical types of nephritis and the importance of the role of sepsis in special groups of cases. In the last few years perhaps most attention has been paid to the effects of alkalis and of high basic diets in nephritis. The beneficial effects of alkalis and the deleterious action of acids on the kidney have long been recognized. Bright himself advocated the use of an alkaline mixture, and for generations physicians have prescribed alkalis, doubtless empirically, in the treatment of kidney disease. Nowadays they are given in three different types of case. It is now the practice of many fever hospitals to give alkalis to all cases of scarlet fever, since it is considered that by this regime the incidence of post-scarlatinal nephritis has been considerably reduced. The usual dosage ranges between 120 and 240 grains per day, the aim being to give just sufficient alkali to keep the urine about the neutral point. In cases of hydraemic nephritis Osman has emphasized the value of large doses of alkalis—700 to 800 grains per day. This line of treatment may sometimes be beneficial, though in cases of mixed nephritis the results are disappointing. Moreover, there are decided disadvantages. In many patients these large doses cause much vomiting, while there is a very real danger of alkalosis. Such intensive alkaline therapy has very definite limitations, and in my opinion should never be attempted unless under hospital conditions of supervision.

Perhaps a more useful part is played by alkalis in the metabolic group of cases as already defined. They may be administered in a mixture of the usual alkaline salts or in the form of the so-called "high base diet." The acid or base forming properties of food depend on the inorganic constituents or "ash" remaining after the organic constituents have been oxidized in the body, and, according to the type of food ingested, this ash may be neutral, acid, or alkaline. On a high base diet, namely, fruit, vegetables, bread, and milk, even advanced cases of chronic interstitial nephritis seem to improve. It is possible to arrange a high acid and high base diet containing the same amount of protein, and recent carefully controlled work by Lyon and his co-workers shows that cases do much better on the high base diet. The practical deduction is that it is not the type of protein in the diet which matters, but the actual acidity or basicity of the diet used. In other words, dietetic restrictions are of minor importance so long as the diet given is sufficiently "covered" by doses of alkalis. In practice, however, the highly acid character of certain proteins makes their adequate covering with alkalis a matter of some difficulty. Certain restrictions are required, but a varied diet of meat, fish, and eggs may safely be given to patients with chronic interstitial nephritis to their physical benefit and mental contentment if alkalis in ordinary doses be also administered.