The conclusion is that the simplest method of treating premenstrual tension is by means of diuretics such as ammonium chloride, and, if this is not effective, ethisterone or methyltestosterone should be tried.

Summary

The clinical features of premenstrual tension are described and the possible mode of origin of the various symptoms is discussed. Hydration, blood chemistry changes, autonomic nervous system responses, as well as psychogenic and personality factors are believed to play a part in the syndrome. A working hypothesis on which treatment was based is postulated.

Treatment was applied at various aetiological levels, including psychotherapy, dehydration therapy, and administration of progestogens and androgens.

Psychotherapy has an important but limited role. Dehydration therapy was found to alleviate some of the symptoms only. The most effective methods of treatment were administration of a progestogen such as ethisterone or an androgen such as methyltestosterone during the second half of the menstrual cycle.

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BRIGHT'S DISEASE

AN ATTEMPT AT A STATISTICAL ASSESSMENT OF THE CLASSIFICATION PROPOSED BY ELLIS

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[WITH SPECIAL PLATE]

In 1827 Richard Bright described the disease of the kidney to which his name is still attached. His cases included all forms of renal disease unassociated with obvious changes in the lower urinary tract, and so were composed mainly of the three main groups of chronic pyelonephritis, hypertensive nephrosclerosis, and true nephritis. The second of these was clearly separated by Allbutt (1895–6), and the last has been defined by Osler (quoted by Osman, 1939) as "bilateral, non-suppurative inflammatory disease of the kidneys." The importance and frequency of the first group as a cause of Bright's disease has only recently been recognized.

Numerous attempts have been made to subdivide these cases, but none has received general acceptance. Ellis (1942), as a result of a combined clinical and pathological investigation of "about 600" cases, "about 200" of which were fatal, suggested that nephritis could well be separated into only two groups, which he called types I and II.

Type I nephritis is characterized by an abrupt onset with haematuria, and is often preceded by an acute infection such as a sore throat. In most adequately treated cases recovery is complete. Death may occur in the acute stage, and some cases follow a progressive course ending in death in a few months. Other cases appear to recover completely, yet after many years develop renal failure and hypertension.

Type II nephritis, on the other hand, is of insidious onset and usually presents as oedema with albuminuria. There is no history of preceding infection, and haematuria is uncommon. The course of the disease is steadily downhill, and if renal failure and hypertension supervene the oedema tends to abate. Many of these patients die from secondary infection and the remainder from hypertension or uraemia.

Ellis further suggested that the two types were not only clinically distinct but were associated with specific renal histological changes and were unrelated aetiologically. Davson and Platt (1949) supported his conclusions with the exception of the last, upon which they reserved judgment.

The present communication describes a similar investigation of a comparable group of cases, with the difference that, where possible, clinical and pathological diagnoses were made quite independently. There were two main objects. The first was to examine the lesions found in fatal Bright's disease, and the second was to see what support analysis of a further group of cases lent to Ellis's views.

Material and Method

In all, 154 cases were examined. They were those that had been indexed as Bright's disease in the necropsy records of this hospital during 1931–51. No non-fatal cases were investigated, so no comprehensive survey of the natural history of these diseases can be offered.

Twenty-seven of these cases have not been included in the analyses: 8 of them because there was a history of toxaemia of pregnancy, 2 because of industrial exposure to heavy metals; 1 was a typical case of polyarteritis nodosa, and 1 was associated with scleroderma; in the remaining 15 cases the clinical or pathological material was incomplete.

The clinical diagnoses were made on the ward records, which were examined by C. L. J., only very few of the patients being seen by either of us during life. The original necropsy reports were consulted, where necessary, only for the macroscopic observations, the histological data presented being derived from an examination of preserved material by J. B. E. At first these investigations were entirely independent, and extreme care has been exercised to avoid either diagnosis being influenced by evidence proper to the other. In all cases in which clinical and pathological diagnoses differed a re-examination was made, the only additional information at each observer's disposal then being the fact that a discrepancy had been encountered. It was rarely found possible to alter a diagnosis at this stage, and if agreement was not reached the available information was discussed and a definite decision on grouping was made. In only 9 cases (7.1%) was agreement then reached, leaving 19 cases of disagreement. All analyses, except where otherwise stated, have been based on the assumption that, whereever pathological and clinical conclusions conflicted, those drawn from the latter material were the correct ones.

Nomenclature

The classification proposed by Ellis has been used throughout, and his criteria for the diagnosis of nephritis of

types I and II have been strictly adhered to. He does not include chronic interstitial nephritis in his table, but follows the recent trend of referring to these cases as chronic pyelonephritis (Ellis and Evans, 1933; Longcope and Winkenwerder, 1933; Weiss and Parker, 1939; Platt and Davson, 1950), of which condition they are thought to represent a healing or healed stage. In our diagnoses we have depended largely on the descriptions quoted, and since the lesions closely resemble those of the healing stage of "ascending" renal infection we shall, for convenience, call them "chronic pyelonephritis."

The term "nephrosis" is restricted to a predominantly tubular condition in which there is no evidence whatever of inflammatory disease and of which we have encountered only one case.

In the diagnosis of nephrosclerosis the descriptions of Allbutt (1895–6), Ellis (1938), and Platt (1948) have been followed.

The other conditions in Ellis's table have been seen in numbers too small to merit further discussion (e.g., amyloidosis), or not at all (e.g., focal nephritis), or have been excluded (e.g., toxaemia of pregnancy).

Results

Of the 154 cases examined 15 had been admitted in terminal uraemia, and, although clear histological pictures were seen in 13 of them, no further clinical diagnosis could be made. In the remaining two of these and in seven other cases in which the diagnosis could be established clinically, no more precise conclusion than "contracted kidney" (see below) could be reached from the pathological material. In 16 cases renal lesions were unexpected necropsy findings to which there were no relevant clinical notes. In the remaining 116 cases clinical and pathological diagnoses could be made.

TABLE I.—*Distribution of the Cases by Their Clinical Diagnoses*

	This Series	Platt and Davson (1950)*
Acute type I nephritis	9	23
Chronic type I nephritis	21	
Type II nephritis	24	25
Pyelonephritis	12	26
Benign nephrosclerosis	14	16
Malignant hypertension (primary)	13	24
Amyloid disease	2	3
Lipoid nephrosis	1	
Clinical uraemia (Table IIIA)	15	
Incidental necropsy findings of Bright's disease (Table IIIB)	16	
	127	
Toxaemia of pregnancy	8	2
Exposure to heavy metals	2	
Scleroderma	1	1
Polyarteritis nodosa	1	15
No diagnosis	15	
	154	

* These authors made their diagnoses on all available clinical and pathological information; in our series this procedure would have doubled the number of cases of pyelonephritis.

From Table I it will be seen that the distribution of the cases in the major groups is roughly the same as that found in the similar table of Platt and Davson (1950), but the ratio of type I to type II cases is higher than in the fatal cases of Ellis's (1942) series.

Table II shows those 89 cases in which a definite diagnosis of a specific condition could be made on both clinical and pathological grounds, together with the agreements between these diagnoses. In this analysis the number of cases in which agreement has been reached is also expressed as a percentage of the number of clinical and of pathological diagnoses alone and in combination (lines 2, 3, and 4). The total number of cases in line 4 is greater than that in lines 2 and 3 by the 19 cases in which agreement was not reached, for these appear in it twice.

The degree of accuracy of the clinical and pathological diagnoses individually considered is somewhat variable among the different conditions. The lowest figures are seen for the clinical and combined diagnoses of chronic type I nephritis, but only the latter differ significantly from the accuracy of diagnosis of the rest of the inflammatory group (difference 26%, standard error (s.e.) ± 14%; difference 30%, s.e. ± 13%). Significantly greater accuracy was achieved in both clinical and pathological diagnoses of degenerative as opposed to inflammatory disease (differences 21%, s.e. ± 8%), for the clinical diagnosis of chronic pyelonephritis (difference 35%, s.e. ± 7%), and for the pathological diagnosis of acute type I nephritis (difference 32%, s.e. ± 6%). All other differences may have arisen by chance. In the group of cases in which the clinical or pathological diagnosis was chronic type I or type II nephritis agreement was reached in less than two-thirds of them (63%).

Significant agreements between the clinical and pathological diagnoses combined are seen in all the large groups, but they are not all of the same order. Thus those for degenerative disease (93%, s.e. ± 4.7%, = 20 × s.e.) and for acute type of I nephritis (78%, s.e. ± 12%, = 6 × s.e.) are better than those for the chronic inflammatory conditions (47–57%, approximately 5 × s.e.), all three of which are closely similar.

In the first two groups in Table III renal disease was unsuspected until it became manifest at routine necropsy or by the onset of uraemia. About half of the recognizable cases in these groups—that is, excluding the two cases of contracted kidneys—are chronic pyelonephritis (52%), a significantly higher incidence than in the whole series (difference 23%, s.e. ± 10%). The remainder of the cases are distributed among the other forms of renal disease in proportions similar to those shown in Table II (3).

In the remaining group the pathological findings were widespread extreme glomerular hyalinization with interstitial fibrosis and arteriolar hypertrophy. They probably represent patients with two or more pathological processes, such as chronic pyelonephritis superimposed upon chronic type I nephritis, with the subsequent addition of benign hypertensive sclerosis. No definite diagnosis was made, and they are referred to as contracted kidneys.

TABLE II.—*Cases Which Were Both Clinically and Pathologically Diagnosed as Specific Conditions*

	Acute Type I Nephritis	Chronic Type I Nephritis	Type II Nephritis	Chronic Pyelonephritis	Acute Microscopical Form of Periarteritis Nodosa	Total No. of Cases of Inflammatory Disease	Benign Nephrosclerosis	Primary Malignant Hypertension	Amyloid Disease	Lipoid Nephrosis	Total No. of Cases of Degenerative Disease	Combined Totals
(1) Diagnosis agreed	7	9	16	11	0	43	12	12	2	1	27	70
(2) Diagnosed clinically	9	17	23	11	0	60	13	13	2	1	29	89
% (1/2)	78	53	70	100	—	72	92	92	100	100	93	81
(3) Diagnosed pathologically	7	11	21	20	1	60	14	12	2	1	29	89
% (1/3)	100	82	26	55	0	72	86	100	100	100	93	81
(4) Diagnosed either clinically or pathologically	9	19	28	20	1	61	15	13	2	1	30	89
% (1/4)	78	47	57	55	0	70	80	92	100	100	93	81

TABLE III

A. Histological Findings in Cases Clinically Diagnosed as Uraemia	
Chronic type I nephritis	2
Type II nephritis	2
Chronic pyelonephritis	7
Benign nephrosclerosis	1
Contracted kidneys	2
Kimmelstiel-Wilson lesions	1
B. Forms of Bright's Disease Discovered Incidentally at Necropsy	
Acute type I nephritis	2
Chronic type I nephritis	1
Type II nephritis	5
Chronic pyelonephritis	8
C. Clinical Diagnoses in Cases with "Contracted Kidneys"	
Chronic type I nephritis	4
Type II nephritis	1
Chronic pyelonephritis	1
Benign nephrosclerosis	1
Uraemia	2

Table IV gives the details of the discrepancies between clinical and pathological diagnoses of types I and II of nephritis. An illustrative case history for each important line of this table is given below, each being illustrated by a photomicrograph of a typical lesion (Cases 1–7; Plate, Figs. 1–7).

TABLE IV.—*Cases in Which a Diagnosis of Either Type I or Type II Nephritis was Made Either Clinically or Pathologically and in Which Agreement was Not Reached*

Case No. in Appendix	Clinical Diagnosis	Pathological Diagnosis	No. of Cases
1	Acute type I nephritis	Type II nephritis	1
		Polyarteritis nodosa	1
2	Chronic type I nephritis	Type II nephritis	4
3	,, ,, ,,	Chronic pyelonephritis	3
4	,, ,, ,,	Benign nephrosclerosis	1
5	Type II nephritis (probable)	Chronic type I nephritis	2
	,, ,, ,,	,, pyelonephritis	3
6	,, ,, ,, (definite)	,, ,,	2
7	Benign nephrosclerosis	Type II nephritis	1
	Total		18

There were 11 cases in this series diagnosed clinically or pathologically as acute nephritis of type I. Nine of these were diagnosed pathologically as acute type I nephritis, and one, which was of about 14 days' duration, as early type II nephritis (Case 1). The acute microscopical form of polyarteritis nodosa (Davson *et al.*, 1948) was seen only once, but it was noted that acute inflammatory arterial lesions are not uncommon in diseased kidneys. They were seen in three cases in the present series (2%); in one diagnosed pathologically as type II nephritis and clinically as type I, in one of primary malignant hypertension, and in one in which that condition was due to chronic pyelonephritis. The diagnosis of polyarteritis was, however, made only when there was adequate supporting clinical and pathological evidence. Thus, while it seems possible that there is some relationship between nephritis and polyarteritis nodosa, as suggested by Davson *et al.* (1948), our cases are more in keeping with the classical view that acute glomerulonephritis is a clinico-pathological entity that may be fatal in the acute phase.

Case Reports

Case 1.—A woman aged 38 was admitted complaining of swelling of the legs for two days. One month previously she had had tonsillitis and had been sent to bed and given sulphonamides, with much improvement. Two weeks before admission she had a second attack, with slightly swollen ankles. This persisted and became much worse two days before admission. On examination her temperature was 102° F. (38.9° C.) and there was facial and ankle oedema. The blood pressure was 150/80 mm. Hg, and there were some tender enlarged lymph nodes in the anterior triangles of the neck. The urine was much reduced in amount and was of normal specific gravity. It contained red blood cells and epithelial casts with some protein. The blood urea was 100 mg. per 100 ml. A week after admission she became very drowsy and confused, and remained in this state until her death five days later. *Clinical diagnosis:* acute type I nephritis.

At necropsy there was pallor and oedema of the extremities. Death was due to bronchopneumonia. The heart was normal and weighed 300 g. There were a few petechiae in the stomach. The kidneys were enlarged (right 280 g., left 260 g.), soft, and pale, but their pattern was normal. *Pathological diagnosis:* early type II nephritis.

Case 2.—A boy aged 13 was admitted complaining of swelling of the face and legs for a week. At the age of 9 he had had an attack of tonsillitis followed by an illness in which he had headache and pyrexia. He remained in indifferent health, and three months later was admitted to Queen Mary's Hospital, where nephritis was diagnosed. He remained in hospital for six months and then spent six months at home. After this he was again in Queen Mary's for a further six months, and was then admitted to Guy's. He improved greatly on a high-protein diet and was discharged after ten months' treatment. He remained in reasonable health for about a year, although he had several attacks of oedema, each lasting a few days. A week before his last admission he had an attack of macroscopical haematuria, for which he was referred to hospital. On examination he had marked facial, abdominal, and peripheral oedema. His urine contained 10 parts (Esbach) of protein per litre and blood ++. The blood urea was 48 mg. per 100 ml. After a transitory improvement he became progressively worse, and during the next two months he developed pleural and pericardial effusions. At the time of his death the blood urea was 96 mg. per 100 ml. *Clinical diagnosis:* chronic type I nephritis.

Necropsy revealed gross oedema and pallor. The heart weighed 200 g. and was normal, and there was bilateral bronchopneumonia with empyemata. Both kidneys were enlarged, each weighing 225 g. The pattern was preserved and there was yellow streaking of the renal substance. *Pathological diagnosis:* type II nephritis.

Case 3.—A man aged 34 was admitted, having had headaches, vomiting, and blurred vision for two months. When aged 20 he was admitted to Guy's with a story of symptomless haematuria on three occasions, each lasting a week, and of a fourth attack associated with malaise. On examination then he was slightly pyrexial but had no oedema. The blood pressure was 130/80 mm. Hg. His urine (sp. gr. 1025) was bright red with blood, and contained 4 parts (Esbach) of protein per litre. He remained in hospital in bed for the next 50 days, during which time his urine was tested daily. It showed a gradual return to normality except for the persistence of a trace of protein. On only one occasion were pus cells noted. The level of the blood urea on admission was 45 mg. per 100 ml. A diagnosis of acute nephritis was made, and he stayed in hospital for 12 weeks. He remained in good health for the next 13 years, when, two months before his last admission, he was found on examination to be drowsy, with a brown dry tongue and a blood pressure of 240/145 mm. Hg. Papilloedema was present. His urine contained 3 parts (Esbach) protein per litre, the specific gravity varied only from 1008 to 1010, and a few red cells were present, but no casts or pus cells. Urine cultures were sterile and the blood urea was 150 mg. per 100 ml. When he died in a coma one month later the urea had risen to 300 mg. per 100 ml. *Clinical diagnosis:* chronic type I nephritis.

At necropsy the body was pale and there was marked cardiovascular hypertrophy, the heart weighing 550 g. The kidneys were very small (right 30 g., left 50 g.) and the pattern was completely destroyed. The surface was coarsely granular and scarred, and numerous cysts were present. *Pathological diagnosis:* chronic pyelonephritis.

Case 4.—A man aged 58 was admitted complaining of breathlessness and haemoptysis, later shown to be due to pulmonary tuberculosis. He died four months later. He had acute nephritis when aged 23, and at 37 was admitted to St. Olave's Hospital with oedema of the

face, eyes, and legs. A diagnosis of chronic nephritis was made, but unfortunately the records of this admission were destroyed by enemy action. When aged 56 he was admitted to St. Olave's for indigestion, which was shown to be due to a gastric ulcer. Pulmonary tuberculosis and chronic nephritis were also present at this time. His blood pressure was 240/130 mm. Hg. The urine (sp. gr. 1010) contained protein, a few epithelial casts, and some red cells. On admission to Guy's his blood pressure was 245/110 mm. Hg; protein was still present in his urine, but its specific gravity was 1020. The level of blood urea was 66 mg. per 100 ml. *Clinical diagnosis* of the renal condition: chronic type I nephritis.

Necropsy showed advanced bilateral chronic respiratory tuberculosis, and death was due to two large cerebral haemorrhages, one in the right basal ganglia and the other in the pons. Marked cardiovascular hypertrophy was present, the heart weighing 517 g. The kidneys were slightly reduced in size and unequal, although not greatly so (right 97 g., left 107 g.). There was slight capsular adhesion to the finely granular surfaces. The pattern was substantially normal and there was an increase in pelvic fat. The renal arteries were atheromatous. *Pathological diagnosis:* benign nephrosclerosis.

Case 5.—A man aged 30 enjoyed good health until 18 months before his admission, when he developed headaches and vomiting. He had never been ill in his life before, and there was no family history of hypertension. On examination his blood pressure was 220/170 mm. Hg and papilloedema was seen. His urine had a fixed specific gravity of 1010 and contained protein +++ with some granular and hyaline casts and a few pus cells. No red cells were seen. The blood urea on admission was 75 mg. per 100 ml., and rose to 111 mg. per 100 ml. before his death in coma one month later. This man was in the malignant phase of hypertension. In the absence of any family history the occurrence of the condition at this age was probably due to renal disease. There was no history of this, but the urine was more typical of type II nephritis than of primary malignant hypertension. *Clinical diagnosis:* probable type II nephritis with malignant hypertension.

At necropsy pallor and slight oedema were found, along with cardiovascular hypertrophy and bilateral basal bronchopneumonia. The kidneys were of normal size, and each weighed 150 g. The pattern was completely destroyed, the cortex was very thin, and there were some yellow streaks in the renal substance. *Pathological diagnosis:* chronic type I nephritis.

Case 6.—A man aged 30 was admitted complaining of loss of energy and cramps in the calves for about six weeks. For some time he had noticed puffiness of the face and had had nocturnal frequency. The illness started with a coryza and nasal catarrh, and later he developed a sore throat, caused, he thought, by energetic nasal douching with strong saline. This persisted while the other symptoms developed. On examination he was sallow and his face was puffy. His tongue was brown and his mouth dry. The blood pressure was 120/80 mm. Hg, and the urine, which always contained protein, was acid in reaction and had a specific gravity of 1011. Red cells were seen microscopically on only one occasion. The blood urea was 393 mg. per 100 ml. Ten days after admission parotitis developed on the right side and he died a week later. *Clinical diagnosis:* type II nephritis.

At necropsy parotitis was present and there was some oedema of the brain and meninges. The cardiovascular system was normal. There was extreme hydronephrosis on the right side and some large cysts were present in the kidney substance, which was less than a centimetre thick. The left kidney was very small, and had a granular surface and some small cysts. *Pathological diagnosis:* Chronic pyelonephritis.

Case 7.—A woman aged 60 was admitted with a right-sided hemiplegia. She had suffered from mental deterioration for three years, and for three weeks before the stroke she had been treated in bed for heart failure. Before this she had enjoyed good health. Her mother had also had senile mental deterioration and had died from a stroke. On examination her blood pressure was 195/95 mm. Hg; her urine contained protein ++, granular and cellular casts, some red cells, and had a specific gravity of 1025. She died without regaining consciousness. *Clinical diagnosis:* arteriosclerosis with benign nephrosclerosis.

At necropsy there was marked cardiovascular hypertrophy and sclerosis, the heart weighing 500 g. The kidneys were slightly enlarged, and there was some increase in pelvic fat. The pattern was obscure. Some scarring was seen, and there were cysts in the cortex and slight capsular adhesion. *Pathological diagnosis:* type I nephritis.

Clinical Discussion

No difficulty was experienced in the diagnosis of type I nephritis, and clear cases of type II nephritis were also seen. There were, however, cases in which there was abundant information in the notes, but which did not fit easily into this classification. Thus in five of the cases in Table IV—that is, those recorded as Type II (probable)—there was no record of oedema although the clinical story and laboratory findings were otherwise typical.

In his Table I Ellis (1942) showed that his type I nephritis of abrupt onset was clinically distinct from the insidious type II. Similar tables could, however, be constructed for rheumatism of abrupt onset (acute rheumatic fever) and the insidious form of that disease (chronic rheumatic heart disease), but would not be held to separate them aetiologically. Further, in distinguishing type II nephritis from chronic type I no mention is made of the possibility of mild attacks of acute nephritis passing unnoticed, although the paucity of signs and symptoms in some cases that are so recognized and the frequency of acute tonsillitis make the common occurrence of subclinical attacks highly probable. The incidental discovery of three cases of type I nephritis at necropsy during a period in which 30 cases were diagnosed clinically (10%) supports this possibility. Moreover, the clinical features of type II nephritis are much less precise than those of the clearly recognizable type I, so that the former diagnosis may well be applied to various different renal conditions having in common the absence of an acute mode of onset.

Thus it seems that Ellis's contentions must depend for their support upon the existence of a characteristic individual and mutually exclusive histology in his two types.

Pathological Discussion

The main argument against any classification of nephritis is that there is often considerable overlapping of the features presented by a series of cases. There is no doubt that the separate histological pictures described by Ellis do occur in pure form, and the sections here studied contain some unequivocal examples of each. In many kidneys, however, we have found some of the features of several lesions. In particular, the subendothelial hyalinization of the capillaries, characteristic of the type II lesion, was encountered in some degree in ischaemic and other kidneys unassociated with a history of nephritis, and in a case of clinically typical acute nephritis the earlier stage of "bland focal necroses" (Ellis, 1942) was seen. Somewhat similar changes have also been seen in routine necropsy sections from apparently unrelated conditions such as scleroderma and exfoliative dermatitis (one case of each). Seven of our cases also confirm Davson and Platt's (1949) finding of both type I and type II lesions in the same kidney. Agreement had, however, been reached in all these cases, three being diagnosed as type I and four as type II on the basis of the numerically predominant lesion in a glomerular count.

Ellis's method of subdividing these cases pathologically was not, therefore, found entirely satisfactory; neither was the apparent simplicity of the classification paralleled by a comparable ease of microscopical diagnosis.

General Discussion

Highly significant agreements have been found between clinical and pathological diagnoses made on Ellis's criteria after great care had been exercised to avoid collusion. We must conclude, therefore, as did Davson and Platt (1949), that his classification does fit the observed facts. This does not, however, necessarily imply that the features of types I and II require acceptance in every particular. Indeed, it may mean no more than that the less dramatic clinical stories are associated with the less remarkable pathological pictures. Thus while the percentages for the chronic inflammatory diseases shown in Table II (4) represent significantly more agreements than would be obtained by chance, they clearly do not in our hands imply a very high degree of diagnostic accuracy. The independent operation of personal factors may be very important here and cannot be allowed for other than by examining an adequate series with sufficient care. It is of course common experience that any case of more than ordinary complexity may, in the hands of different clinicians and different pathologists, give rise to widely differing opinions as well as to discrepancy between the *ante mortem* and *post mortem* diagnoses; but we believe that the nature of these 154 cases and the materials on which our diagnoses have been based are such that our conclusions are unlikely to be very different from those of other observers making use of the same criteria. If this is so the figures quoted strongly suggest that these criteria may not have been exclusive enough.

It seems to us, therefore, that Ellis's types I and II of nephritis are not mutually exclusive entities, but in his Croonian Lectures little attention was paid to this possibility. The more guarded conclusions of Davson and Platt (1949), the fact that in their investigation clinician and pathologist found it necessary to confer before agreement was reached, and their statement that pathological diagnosis was often made by deciding which was the predominating lesion make it clear that in their series there was some evidence in its favour. In particular, their Case 11, in which the changes of malignant hypertension were found in a type II kidney, may be noted—a combination that we also saw in one case.

We think it more probable, therefore, that the different appearances of type I and type II nephritis represent variations in the response of the renal parenchyma to a single disease rather than to entirely different processes. If this be so, and in view of the difficulties experienced in diagnosis, it seems to us that this classification has little to recommend it from a descriptive point of view. We present no new aetiological evidence, but others have already suggested (Davson and Platt, 1949) that some criticisms may be levelled on these grounds. The pathogenesis has not been elucidated, and it is not necessary to specify the pathological lesion present before giving a prognosis to a patient diagnosed clinically. It may be considered preferable, then, to await some aetiological indications, since these are the only basis for a lasting classification, before accepting a new terminology for nephritis.

Conclusions

In our series of 154 fatal cases of Bright's disease we found chronic pyelonephritis to be about as common as all other forms of inflammatory disease. Acute glomerulonephritis occurred fairly often, but we have no data from which to calculate a fatality rate for this condition. We regard it as an entity separate from and probably commoner than the acute microscopical form of polyarteritis nodosa.

We have obtained significant agreements between independent clinical and pathological diagnoses in all the major groups in the classification suggested by Ellis. Those for degenerative disease and for acute type I nephritis were much better than those for chronic inflammatory conditions. Considerable overlapping was discovered between types I and II of nephritis, and only 63% of them were accurately diagnosed.

From these figures we conclude that the Ellis classification does fit this series of cases, but in its most important aspect, the separation of types I and II of nephritis, it so lacks precision that its adoption hardly seems justified.

We found type II nephritis to be renal failure of insidious onset which may be associated with a variety of pathological conditions. These included acute nephritis, pyelonephritis, and hypertensive and polyarteritic renal lesions. Kidneys showing more than one histological feature said to be individually characteristic were common.

For the present, therefore, it seems that there is little reason for departing from the time-honoured procedure of classifying nephritis into acute, subacute, and chronic forms on clinical grounds. Recognition of nephritis—the acute form of which can usually be distinguished—chronic pyelonephritis, and vascular lesions is probably all that can be accomplished on morphological grounds alone.

Summary

A series of 154 fatal cases of Bright's disease occurring in Guy's Hospital between 1931 and 1951 have been examined independently by us, one making a clinical and the other a pathological diagnosis. The diagnoses were compared and an attempt was made to analyse the results statistically.

Of the lesions responsible for fatal Bright's disease, chronic pyelonephritis accounts for a large proportion, and acute nephritis is not uncommon.

Significant agreement between clinical and histological diagnoses was discovered in all groups when the Ellis classification was applied to these cases. Those for chronic inflammatory conditions were, however, less close than for any of the other major forms of renal disease. Considerable overlapping was encountered and diagnosis was often difficult.

Type II nephritis as diagnosed clinically may be due to several different pathological lesions.

For the present it is suggested that the classification of clinical nephritis into acute, subacute, and chronic forms be continued, and that pathological subdivision be restricted.

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During the twelve months to November 30, 1952, 4,858 children came into the care of the L.C.C., while 4,169 were discharged. On that date there were altogether 8,027 children in care. This represents an increase of about 700 over the previous year, and of 2,300 compared with three years ago. The numbers in the various age-groups were as follows: Under 2 years, 681 children; 2 to 5 years, 1,411; 5 to 15 years, 4,964; and over 15 years, 971. Of the children taken into care during this period, 45 had no parents, 269 were abandoned, 2,637 were taken into care owing to the illness or infirmity of their parents or guardians, and 631 came as a result of "fit person" orders. These figures were given at a meeting of the Children's Committee of the L.C.C. on April 27.

J. B. ENTICKNAP AND C. L. JOINER : BRIGHT'S DISEASE

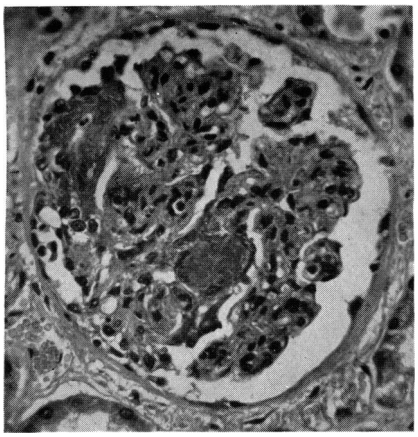


FIG. 1.—Early focal-necrosis stage of type II nephritis in a kidney from a case clinically diagnosed as type I. (H. and E. ×210.)

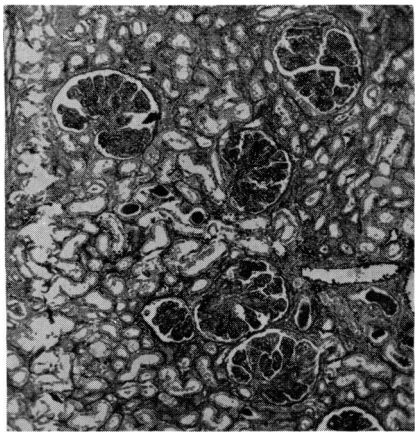


FIG. 2.—Type II nephritis in a case clinically diagnosed as type I. (Mallory. ×50.)

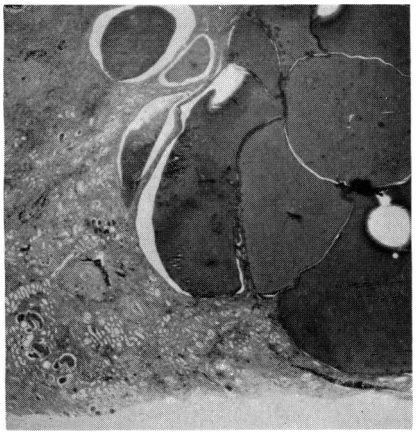


FIG. 3.—Chronic pyelonephritis in a case of type I nephritis. (H. and E. ×16.)

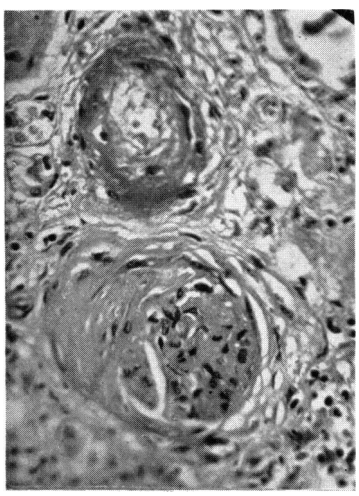


FIG. 4.—Benign nephrosclerosis in a case of type I nephritis. (H. and E. ×200.)

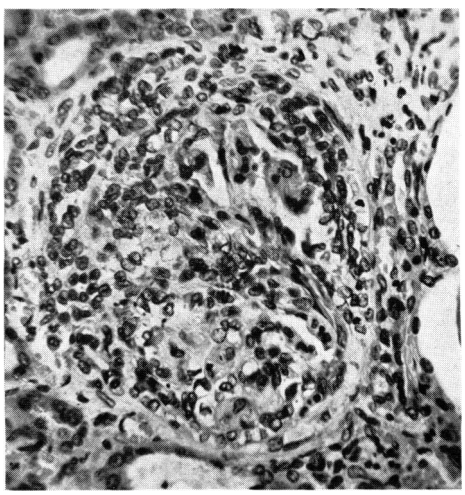


FIG. 5.—Type I glomerular lesion in a case of type II nephritis. (H. and E. ×180.)

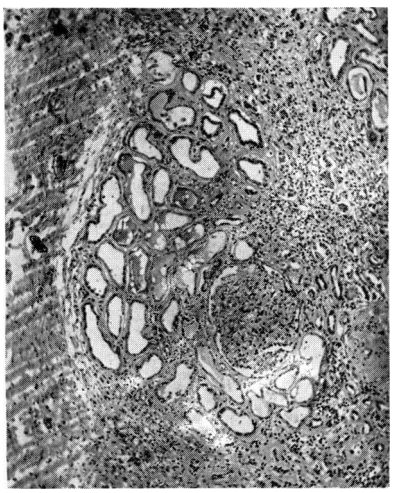


FIG. 6.—Chronic pyelonephritis in a case of type II nephritis. (H. and E. ×70.)

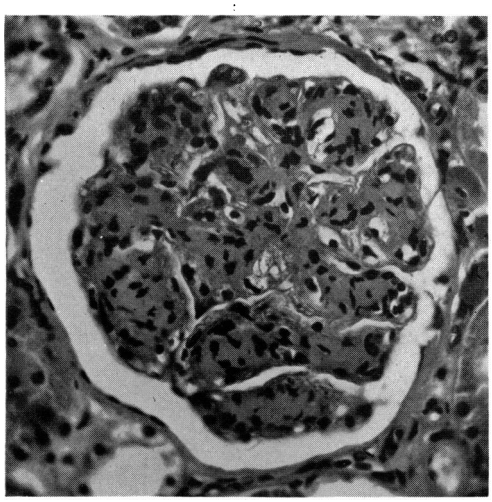
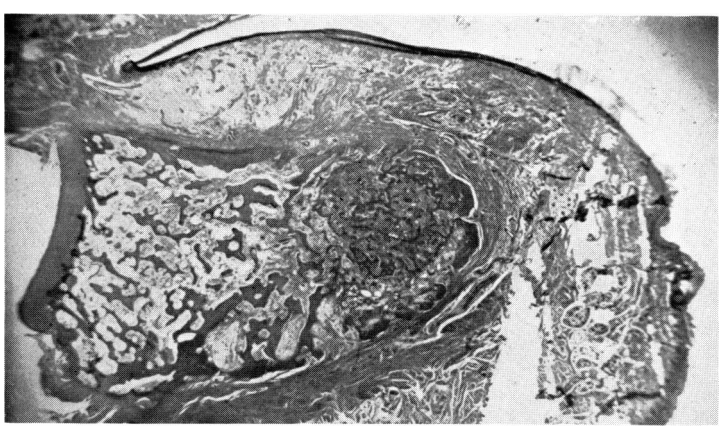


FIG. 7.—Type II glomerular lesion in a case of benign nephrosclerosis. (H. and E. ×200.)

E. H. STRACH : OSTEOID OSTEOMA



Sagittal section of the terminal phalanx showing the osteoid osteoma at the tip. (Low power.)