no important structures are pressed upon the diagnosis may be most difficult. Especially if pain be absent, the true state of affairs may easily be overlooked. In some of such cases, the only symptoms are cough (which may not have the characteristic ring) and increasing dyspnoea; on auscultation some râles may be heard at the left pulmonary base. Such symptoms and physical signs may easily be ascribed to other causes. The lungs may be emphysematous, still further complicating the difficulties of diagnosis. When examining patients exhibiting these symptoms and signs, the aortic second sound should be carefully noted. Then, if an aneurysm be present, the characteristic ringing second sound is heard accompanied by a distinct jog to the ear, when a wooden stethoscope is employed. The presence of this phenomenon should always give rise to the probability of aneurysm being present. In the cases to which I am now alluding the diagnosis may be corroborated by the presence of tracheal tugging, or the conduction of tracheal sounds to the margin of the manubrium sterni. If the aneurysm is situated on the descending thoracic aorta the diagnosis will be still more difficult. Indeed, if pain be absent, as not infrequently occurs, the exact morbid condition may remain entirely unrecognized. Then we only have the cough, dyspepsia, and râles at the base of the left lung to guide us, and these phenomena may, as we know, be due to many causes. The *x* rays may then lend valuable aid in diagnosis; the shadow yielded by an aneurysm being plainly visible, although errors are easily made, unless the shadow cast by a healthy aorta is fully known.

I have laid stress on the above points in order further to accentuate my contention that these râles may be indicative of grave organic disease, and may be the only physical sign to warn us that some obscure lesion is present.

I will here relate a case which shows the difficulty which arises in cases of this kind.

A man, aged 56, had been in the Middlesex Hospital, having been admitted for hæmoptysis, but at that time there were no definite physical signs. He subsequently attended under my care as an out-patient. I found a faint diastolic murmur over the aortic area, and numerous râles just above the angle of the scapula on the left side. He complained of no pain whatsoever, and was thought to be suffering from aortic regurgitation. Some time afterwards he was again admitted into the wards with profuse hæmoptysis, and died in about three hours. At the autopsy an aneurysm of the descending thoracic aorta was found, which had ruptured into the lung at a spot exactly corresponding to that over which I had heard the râles.

This case is a striking instance of the uncertainty which attends the occurrence of this disease, and the importance that may be attached in certain cases to the appearance of these râles.

Closely allied to aneurysm is the existence of other tumours in the chest indicating their existence only by pressure on a bronchus or vessels at the root of the lung. Carcinoma affecting a lung usually commences at the root, and may give rise to the physical sign I have been considering.

I may, perhaps, be here allowed to interpolate the warning that dyspnoea out of all proportion to the physical signs points to the possibility of carcinoma of the lung; the other indications, especially the wasting, only becoming marked at a later period of the case.

In children, also, enlarged glands may give rise to similar manifestations. In such instances, paroxysmal attacks of dyspnoea, closely resembling asthmatic attacks, may be observed.

The difficulties of diagnosis in such cases is obvious. As I have already indicated, râles at the base of one lung are present in many varying conditions. I will now briefly refer to one or two others in which they cannot be ascribed to persistent oedema of the lungs, but these diseases may easily be mistaken for some of those I have already pointed out. Râles at the base of one lung, not necessarily the left, may be the first indication of early basic pulmonary tuberculosis. I have seen several instances of patients attending in the out-patient department of Brompton Hospital complaining of cough, expectoration, and loss of flesh, the only physical sign being the existence of such râles, the subsequent course of the illness indicating its tuberculous nature. The examination of the sputum is, under such circumstances, of the greatest value, and I may insist on the importance of an examination for

tubercle bacilli in all cases where the diagnosis is obscure. Pulmonary tuberculosis commencing at the base instead of at the apex is uncommon, but this initial aide must not be forgotten.

Another lesion that may give rise to similar physical signs is bronchiectasis. The signs in this disease are frequently obscure. Râles may be present over the affected area of the lung, and if this happens to be the lower lobe we again have the sounds to which I am referring. In many cases there is no obvious impairment of the percussion note or alteration of the breath sounds. The râles, however, have a peculiar character, being sharply crackling and high-pitched. By some physicians they are known as "bronchiectatic râles." The odour of the breath, especially after coughing, and the peculiarities of the sputum may corroborate the diagnosis. The condition may, however, be confounded with pulmonary abscess; but in the latter disease the sputum is not so copious, and the physical signs are often very slight. Abscess of the lung affecting the base is, I believe, not common, but in other areas the differential diagnosis between gangrene of the lung, bronchiectasis, and localized empyema is often very difficult, but scarcely enters into the range of my subject to-day. I would only add that in bronchiectasis and abscess of the lung, the former especially, treatment by means of the creosote bath has given excellent results.

I do not suggest that I have exhausted all the causes which may give rise to temporary oedema; collapse of the lung may, for instance, give rise to temporary oedema, but is difficult to distinguish from other conditions. I trust, however, that I have said sufficient to show that the greatest care is necessary in arriving at a conclusion when signs of persistent oedema are found to exist.

Remarks

ON

ACUTE NEPHRITIS (MORBUS BRIGHTII),

WITH SPECIAL REFERENCE TO TREATMENT.*

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ALTHOUGH anasarca and ascites were well known to the ancients, and are referred to in the writings of Aretaeus and Celsus amongst others, and much later Contugno and Cruikshanks in the eighteenth century noticed that dropsy is often attended with the presence of serum in the urine, yet it was left to our renowned countryman, Richard Bright, to discover and establish the fact that many cases of dropsy were dependent on well-marked lesions of the kidneys, and that the urine in such cases was albuminous. His *Reports of Medical Cases*, published in 1827, containing a lucid description of 23 renal cases, with the *post-mortem* reports in some instances, is a lasting monument to his acumen and skill as a physician. Following Bright came many workers, notably Osborne, James Gregory, Christison, Barlow, Rees, and Wilkes in this country, and Rayer, Professor Frerichs, Jaccoud, and Bartels on the Continent. These men were divided into two schools, one led by Bright and supported by Rayer, which contended that the primary disease was situated in the kidney; and the other led by Graves, who in 1831 propounded that the condition of the blood was the primary disease, and the condition of the kidney which resulted from the irritation of the albumen passing through it was secondary. And there was truth in both of these views, as acute and chronic renal disease had not yet been differentiated, and Richard Bright himself, under his third heading, described "contracted kidney." It was Rayer in 1840 (*Traité des Maladies des Reins*) who first positively recognized the changes in the kidneys to be inflammatory, and stated that they could be acute or chronic. This view was proved conclusively by the histological investigations of Reinhardt and Frerichs in 1850 and 1851. Anticipating both these writers, Dr. George Johnson pub-

* A paper read before the Liverpool Medical Institution, February 15th, 1906.

lished a series of memoirs, followed by a large work in 1852 on *Diseases of the Kidneys*, in which he endeavoured to classify the diseases from the results of his investigation into the minute pathological anatomy of the organ. These investigations were strikingly important in one particular, namely, they were the first that recognized the changes which the blood-vessel walls undergo. And these observations were thus precursors of those well-known researches of Grainger Stewart, Gull, and others. Before leaving this brief historical note I must mention the names of Virchow, Bartels, Bradford, and last, but not least, Dickinson, to whom we owe much of our present knowledge of Bright's disease.

In this paper I deal with acute nephritis entirely from a clinical aspect, and not from its pathological side.

Etiology.

Acute Bright's disease is undoubtedly a complaint of the temperate zones, and though occurring in the tropics much less frequently, is practically unknown in the arctic regions. It therefore appears that cold, to produce this disorder, is more likely to do so under a varying temperature than in an equable one, and as if the active combustion required for the maintenance of animal heat in the polar regions consumes matters which in other circumstances would be thrown on the kidneys to their detriment. The disease is more common in the male sex, even in childhood, and this difference is decidedly more marked among adults. Thus Dr. Tripe, writing on scarlatinal dropsy, found in 99 cases—60 males and 39 females; and of 105 cases of nephritis from all causes in

children under 12 years of age at the Hospital for Sick Children, 58 were in boys and 47 in girls.² In later life the preponderance is on the same side; thus, Dr. Dickinson recalls that of 54 cases under his own observation in adults (that is, over the age of 16), there were 33 males and 21 females.³ My 42 cases show a very similar proportion—25 males and 17 females. This is not surprising when it is remembered that man throws more work upon the kidney in eating and drinking than woman, and is more liable to exposure to cold and wet and changes of temperature, and also the poison of lead. There is one cause of renal disease which affects females only, that is pregnancy.

As regards age, probably no age is exempt. It is, however, rare in the first year of life, and is stated to be rare after 40, but not unknown after 50. The two decades in which most cases are said to occur are up to 10 and up to 30. My cases, as far as they go, seem to show that, in proportion to the population, all the decades are affected pretty uniformly after the first. For, considering that they are taken chiefly from admissions to a general hospital, the number in the first decade is probably below the average.

The cases (42) are as follows:

Up to 10, up to 20, up to 30, up to 40, up to 50, up to 60, 60+
9 7 7 8 6 2 3

The youngest patients were 1 year 2 months and 2½ years, and the oldest 62, 63, and 65 years respectively. And as not very infrequently acute nephritis occurs in a subject suffering from chronic nephritis, and is then often

Summary of Cases.

No.	Case.	Age.	Sex	Month Disease Commenced.	Occupation.	Cause of Disease.	Duration in Hospital.	Result.	Remarks.
1	G. J.	8	M.	November	Schoolboy	Scarlatina	9 weeks	Cured	—
2	M. G.	38	F.	July	Housewife	(c) Cold and wet	15 weeks	Cured	—
3	R. W.	62	M.	June	Dock labourer	(c) Cold and wet	11 weeks	Cured	—
4	A. H.	17	F.	January	Confectioner	Cold and wet	18 weeks	In statu quo	Had suffered from the disease 7 months prior to admission.
5	A. M.	32	F.	October	Housewife (washerwoman)	Cold and wet	13 weeks	Cured	—
6	M. H.	26	F.	July	Domestic servant	Scarlatina	23½ weeks	Improved	This patient recovered 5 months after leaving hospital.
7	W. P.	5	M.	April	Schoolboy	Cold and wet	10 weeks	Cured	—
8	M. E.	18	F.	October	At home	Cold and wet	3½ weeks	Cured	Was an O. P. for 2½ months.
9	F. P.	17	M.	July	Nil	Doubtful	10 weeks	Cured	—
10	G. B.	1 year 2 mos.	M.	January	—	Doubtful	7 weeks	Cured	—
11	C. W.	54	F.	January	Housewife	Cold and wet	7 weeks	Cured	Dilatation of left ventricle and mitral regurgitation.
12	R. T.	51	F.	November	Housewife	Doubtful	8 weeks	Left at own request, in statu quo	Ascites, 5 pints withdrawn.
13	E. D.	18	M.	September	Apprentice	Burns	7 weeks	Cured	Mitral stenosis.
14	H. B.	42	M.	May	Painter	Lead, previous syphilis	12 days	Improved	Left at own request.
15	J. B.	10	M.	August	Schoolboy	Scarlatina	5 weeks	Cured	—
16	R. C.	34	M.	July	Dock labourer	(c) Cold and wet	16 weeks	Cured	Dropsy after scarlatina when 3 or 4 years of age.
17	R. H.	39	M.	March	Porter	(c) Cold and wet	9 weeks	Cured	—
18	F. P. W.	2½	M.	November	—	Scarlatina	2 weeks	Cured	Marked dropsy disappeared very rapidly.
19	D. W.	13	F.	October	Schoolgirl	Cold and wet	5 weeks	Cured	—
20	J. W.	17	M.	January	Stable boy	Cold and wet	3 weeks	Greatly improved	Left at own request.
21	N. K.	37	M.	November	Dock labourer	Rheumatic fever	14½ weeks	Improved	Finally passed into chronic nephritis.
22	E. F.	6	F.	February	Schoolgirl	Cold and wet	8 weeks	Cured	Dropsy after scarlatina when aged 3.
23	A. T.	3	F.	January	—	Scarlatina	4 weeks	Cured	—
24	T. S.	37	M.	February	Labourer	Cold and wet	14½ weeks	Cured	A previous attack 12 years before.
25	T. S.	39	M.	August	Labourer	Cold and wet	9 weeks	Cured	Same patient as Case 24. Third attack.
26	B. M.	42	F.	May	Housewife	Pregnancy	13 weeks	Improved	Ninth time pregnant.
27	M. M.	10	M.	May	Schoolboy	Doubtful	7 weeks	Died	Ascites, very marked oedema, and died of cardiac asthenia.
28	H. T.	43	M.	May	Saw-grinder	(c) Doubtful	2 days	Died 48 hours after admission	Complicated by cardiac dilatation.
29	J. H.	49	F.	August	Domestic servant	Cold and wet	21 weeks	Greatly improved	Complicated by dilatation of heart and mitral regurgitation.
*30	J. D.	30	M.	February	Engineer	Diphtheria	8 weeks	Cured	—
*31	R. T.	25	M.	April	Doctor	Diphtheria	6 weeks	Cured	—
*32	M. L.	21	F.	January	Housewife	Pregnancy, Cold and wet	12 months	Cured	Abortion occurred.
*33	J. R.	46	M.	November	Gentleman	(c) Cold and wet	4 months	Cured	—
*34	F. T.	17	F.	January	At home	Cold and wet	Several months	Cured	Uraemia, convulsions and coma for 24 hours.
35	T. L.	6	F.	February	Schoolgirl	Scarlatina	10 weeks	Cured	—
*36	J. R.	27	M.	November	Clerk	Cold and wet	7 weeks	Cured	—
*37	M. T.	63	M.	November	Publican	Excessive drinking	12 weeks	Death	Uraemia, coma.
38	G. F.	24	M.	December	Baker	Chill	10 weeks	Greatly improved	Still in hospital.
39	W. C.	46	M.	December	Clerk	Jaundice	5 weeks	Cured	—
40	R. C.	30	F.	January	Housewife	Chill	7 weeks	Cured	—
41	A. Ch.	21	M.	March	Bricklayer	Cold and wet	12 weeks	Cured	—
42	E. N.	65	F.	January	Housewife	?	4 weeks	Cured	Also suffered from myxoedema.

* Indicates cases seen in Consultation, all the others are Hospital cases. (c) Indicates decided history of alcoholism.

fatal, it must be admitted that it is not as rare after 50 years of age as is sometimes supposed. Among the other predisposing causes, mental and nervous exhaustion, heredity, and alcoholism are important factors, the latter probably being the most important. Many cases of acute Bright's disease date from a chill contracted during or immediately after a drinking bout, six at least of my cases being accounted for in this way. Lastly, as a predisposing cause, I would include a *previous attack of acute nephritis*. Thus, in four of my cases there was a distinct history of a previous attack:

CASE XVI.—A dock labourer, aged 34, stated "that he had had dropsy following scarlet fever at the age of 3 or 4."

CASE XXII.—A girl, aged 6. Mother stated "that the child had had dropsy and inflammation of kidneys at the age of 4, but she had quite recovered after being in bed for a few weeks." This patient had all the signs and symptoms of acute nephritis, and she left the hospital after eight weeks, cured.

CASE XXIV.—A labourer, aged 37, stated he had had a similar illness twelve years before. He left hospital after fourteen and a half weeks cured.

CASE XXV.—The same labourer, aged 39, after a chill was readmitted two and a half years later, with his third attack of acute nephritis; he was discharged cured after nine weeks, but had a relapse when an out-patient, from which he has now completely recovered.

The exciting causes may be grouped:

1. Those affecting the skin, and thus throwing extra work on the kidney; of these, cold and wet is much the most common, and is much more effective if associated with alcoholism. Cold probably acts in producing nephritis as an indirect cause, just in the same way as it acts on the lungs in producing pneumonia.

2. Toxic substances produced by micro-organisms, that is, the specific fevers which evidently produce a renal irritant, notably scarlatina, which is the most fertile source of this disease in childhood. Dr. Dickinson, in his recent article,¹ puts a query after acute rheumatism as a cause, but in Case XXI acute nephritis undoubtedly began whilst the patient was in bed suffering from rheumatic fever.

3. Toxic matters which act as renal irritants, present in the blood—that is, turpentine, cantharides, alcohol, lead, arsenic, and as a subgroup in this class may be added "bile" in cases of jaundice, and sugar in cases of diabetes mellitus. Case XXXIX, a man aged 46, was suffering from jaundice, and had blood, albumen, and casts in his urine.

4. Severe burns which act under both Group 1 and 3.

5. Pregnancy, which excites the disease by interfering with the renal circulation and throwing greater work on the organ. Probably, however, many of these cases are dependent on a chill as well.

The following were the exciting causes in 42 cases:

Cold and wet	...	...	...	21*
Scarlatina	...	...	...	6
Diphtheria	...	...	...	2
Pregnancy	...	...	...	2
Acute rheumatism	...	...	...	1
Burns	...	...	...	1
Excessive drinking	...	...	...	1
Jaundice	...	...	...	1
Lead	...	...	...	1
Doubtful	...	...	...	6†
Total	...	...	...	42

* Including 6 in which there was a history of drinking and chill. Four cases were known to have had previous attacks of acute nephritis.

† One of these, Case XLII, was a woman aged 65, who was also suffering from myxoedema, and it is interesting to note that experimental removal of the thyroid in animals produces albuminuria.

Twenty-seven cases commenced in the winter half of the year (October-March) and 15 in the summer portion (April-September).

The onset of acute nephritis is very varied. In some cases, especially those which are due to a chill (as in Case XIX), the affection may commence with severe febrile phenomena, even a rigor, and the temperature may rise to a high level for a short time, even as high as 104° F. This condition we do not often see in hospital cases, as it has usually passed before they seek admission, but the history of "a shiver and feeling cold" is often given. Thus Case XXIX stated that he had to return home from work as he had a shivering attack. This pyrexia in uncomplicated cases is always of short duration, and is often associated with a coated tongue, headache, dyspepsia, and sometimes vomiting and pain in the loins. Other attacks due to the same cause begin more gradually, the patient notices his urine is scanty and perhaps contains blood. If the patient persists in going to work the

symptoms all become aggravated, especially the dropsy, which becomes general, and soon he is compelled to seek advice. Other cases, especially those which are accompanied by the symptoms of the primary disease are usually first recognized by either of two signs: First, oedema of the face and eyelids or of the body (this occurred in Cases XXI, XXX, and XXXV), with perhaps a slight rise of temperature; or, secondly, by the appearance of blood in the urine. This happened in the case of a medical man who, when suffering from diphtheria, was surprised one morning to find his urine bright red (Case XXXI). Oedema appeared shortly afterwards. However the complaint may begin, at first the amount of urine secreted in twenty-four hours is far below normal in nearly every case, and this condition often exists prior to the dropsy, and, according to Bartels,² even before albumen appears. As a rule, the more scanty the urine passed the more serious the outlook, especially if the condition be maintained. In those cases, fortunately rare, in which total suppression occurs, death usually ensues in a few days. Even, however, if very small quantities of urine are passed, recovery may take place; thus, in Case X only a few drachms, and in Case XXXV, whom I saw in consultation with Dr. Morris, only a few ounces of bloody urine were passed for several days, and yet recovery occurred. Diuresis in the course of an attack is a good sign, and frequently accompanies or precedes the disappearance of the albumen. This condition was recorded in several of my cases. It was most marked in Case XVI, in which the quantity of urine passed per diem suddenly rose from 40 to over 100 oz., and on one day it amounted to 169 oz., and on the following day to 173 oz.; the specific gravity at the same time fell from 1023 to 1012. This polyuria lasted for several weeks whilst the albumen was disappearing. For two weeks before this patient left hospital his urine was quite normal in character and amount. The explanation for this large increase of watery urine is to be found, I think, in the fact that only a portion of the kidneys has recovered and the other parts are still disabled—a condition analogous to that brought about by Bradford's experiments, in which, after the removal of two-thirds of the total renal substance, a greatly-increased flow of dilute urine results. In the clinical conditions, however, the functionally recovered area steadily increases till it finally extends through the whole kidney.

CHARACTER OF URINE.

The urine contains blood, renal debris, and urates. The reaction is invariably acid, the specific gravity usually high, 1024 to 1028, rarely 1030. The total quantity of urea excreted per diem by my patients has never been found to reach the normal quantity. All the other solids are proportionately diminished.

Albumen was found in all these cases of nephritis, but there was one patient in whom it was absent for a time. Case X, my youngest patient, aged 1 year and 2 months, was a well-nourished child, and was admitted suffering from severe general anasarca. His face was swollen and white, eyelids so distended he could not open them; his limbs were distended and swollen, the skin being pale, shiny and pitting readily. Pulse 100 to 120; tension raised. Heart sounds loud and pure at all areas. Respirations 40. There was some oedema at the base of the right lung, and a little fluid in the abdominal cavity. The day following admission, as he had passed no water, 5 drachms were drawn off with the catheter; this urine contained no albumen or blood, and microscopically urates only. It was not until four days after admission that albumen and casts appeared in his urine. All this time the quantity of urine was very scanty, only a few drachms per diem. After the appearance of the albumen the urine became more copious, and the child ultimately recovered. This condition is certainly rare, and, if the appearance and symptoms of the child had not been so typically those of real dropsy, the absence of albumen might have shaken the diagnosis. Bartels³ records that he has met with a few cases in which dropsy set in after scarlet fever, although the urine contained no albumen. In his cases very little urine was excreted; in one only two tablespoonfuls in twenty-four hours. He says: "Very soon afterwards a more abundant excretion of bloody urine set in, whilst the dropsy increased." In these latter points my case differs from his, as the dropsy passed off, and the quantity of urine

increased as the albumen appeared, and at no time was there any haematuria. I agree with his conclusion "that the kidneys of the little dropsical convalescent from scarlet fever must have been in a condition of inflammation at the time when the urine was scanty, although it was free from albumen." Henoch⁷ and Dickinson⁸ have published solitary cases which prove the correctness of this view. One must conclude that this urine, free from albumen, is secreted by a portion, perhaps a small portion, of the kidneys which is not affected by the inflammation. So emphatic is Bartels, that he says: "I have never seen dropsy after scarlet fever that was not due to nephritis."

Haematuria is often present in acute Bright's disease, and was found in 19, that is, 45 per cent. of my cases. Sometimes it disappears in a short time; at others it remains in greater or lesser amounts for weeks. In the former cases it usually disappears much earlier than the albuminuria, and in the latter and less common cases it may be accompanied by only a small quantity of albumen, as in Case XXXVIII, where haematuria was present with only 0.05 per cent. of albumen, and this usually departs shortly after the blood.

Microscopically, casts can nearly always be found. Hyaline and epithelial are the usual variety at the onset, with blood casts if haematuria is present. Later in the history of an attack, the casts usually appear more granular and even fatty, and as the case proceeds towards recovery they diminish in size and number, and finally disappear. If the acute inflammation is limited to the interstitial tissue, there are no casts in the urine from first to last. These cases are rare, and are said to be fatal.

OEDEMA.

The oedema of acute Bright's disease is most characteristic. In Case XXVII the skin and subcutaneous tissues were so distended that quantities of serum exuded through the sweat ducts. This was accompanied by sloughing of the skin. In other and minor cases, the oedema may be more local, being confined to the eyelids and face, where it is especially noticeable in the morning; but probably the best site to find it in patients recumbent in bed is in the loose skin over the sacrum, where it forms the "sacral cushion." In all my cases some oedema was present. But instances are recorded in which most intense acute nephritis with typical scanty bloody urine occur, without any dropsy. Such cases are rare, and often end fatally. This renal dropsy was formerly supposed to be due to the diminished excretion, but Dr. Rose Bradford, in his admirable Croonian Lectures of 1904, has shown that the dropsy is not merely due to renal inefficiency. He proves that experimentally ligation of renal arteries, removal and destruction of the kidney, do not cause dropsy, nor is it produced clinically in the case of nephritis just mentioned, or in complete calculous obstruction, or in thrombosis of the renal arteries. He comes to the conclusion that the dropsy is due to the renal inefficiency plus a hydraemic condition of the blood brought about as a result of the partial or complete suppression of the urinary excretion. There is also experimental evidence in support of this view. And we know that clinically renal dropsy is always associated with marked anaemia, which is so conspicuous in acute Bright's disease, and

is developed so rapidly. This anaemia, though often said to be due to the loss of blood and albumen in the kidneys, appears to me to be quite out of proportion to these causes, and I would suggest that it is either due to (1) a loss of internal secretion from the damaged kidneys which is necessary for the anabolism of the blood, or (2) to harmful products retained in the blood which should be excreted by the kidneys, and which destroy the corpuscles.

CARDIO-VASCULAR CHANGES.

Increased action of the left ventricle of the heart can, I believe, be detected from the onset of the attack in most cases of acute nephritis; and the hard, high tension pulse is evident from the very first. As yet I have only been able to take the blood pressure by the sphygmomanometer in a few cases, and these show a distinct fall in pressure as recovery advances. Case XXV demonstrates in a most striking manner the high blood pressure, and how rapidly it fell, its fall coinciding with the diminution of the albuminuria (vide chart).

OTHER SYMPTOMS.

The coated tongue, anorexia, occasional vomiting, and morning headaches are symptoms which are often present, especially at the onset, or preceding uraemia in severe

cases. The skin in this disease is usually dry and harsh. In one or two of these cases it was ichthyotic in some parts and sweating was induced with extra difficulty. When it is found to be soft and moist from perspiration recovery can be guaranteed.

Lumbar pain is a symptom which was present in twelve of my cases, all adults, and males with the exception of two. Occasionally, as in Cases XXIV, XXV and XXXVI, it persisted into convalescence.

DURATION.

The duration of nephritis is very varied, and it is impossible to predict the course or ultimate result of any

case with certainty. The younger the patient, however the more rapid the course to recovery or death. Thus in my 9 cases of children up to 10 years of age, the duration in Cases XVIII and XXIII was only two and four weeks respectively, and the average length of complaint in the nine cases was just under seven weeks, which, as a glance at the table will show, is very much shorter than that of the adults. Fortunately, many patients steadily progress towards recovery.

The shortest attack among my cases was by no means the mildest. It was a small boy, aged 2½ (Case XVIII). He had had scarlatina some weeks prior to admission. He was pale, very oedematous, and presented the typical picture of acute nephritis. Under the influence of diaphoretics, etc., his skin perspired freely. On the sixth day a large quantity of urine, free from albumen, was excreted, and the dropsy then rapidly passed away. On the other hand, it frequently happens that cases which have steadily, or even rapidly, improved for a time come to a standstill, and the urine is found to contain about the same amount of blood and albumen day after day for long periods, this amount varying from a considerable quantity of both blood and albumen to a trace, or to a trace of albumen alone. Thus, in Case VI, after a steady improvement, the urine contained from 2 to 1 part per 1,000 of

albumen, and was tinged with blood for seventeen weeks, notwithstanding a stringent diet and all the usual methods of treatment. And in Case xxxix, after a steady and rapid improvement, the urine tinged with blood contained three-quarters to one-half part per 1,000 of albumen for weeks. In this case, as the albumen is so slight, I expect it will disappear as soon as the blood ceases. I cannot agree with the statement made by several writers "that haematuria is a good sign because it relieves the congestion." It is an indication of severe inflammation, and, as far as I have been able to observe, those cases have progressed better and more rapidly in which there has been no haematuria or in which it persisted for a very short time. Case xxi of my series supports this contention; for the first four and a half weeks after admission the patient steadily improved, the albumen falling from 7 to 1 part per 1,000, but the haematuria remained, and in spite of the most active and rigid treatment it persisted till he left the hospital, and it was present for many months afterwards. This patient ultimately passed into a state of chronic parenchymatous nephritis. This is an example of the small and rare minority of cases of acute nephritis which passes into the chronic condition, and also coincides with Bartel's experience that true rheumatic nephritis, of which it was an example, tends to become chronic.⁹ Dr. Dickinson truly says:

Of those who completely recover, the vast majority do so within a year; of those who die, the majority do so in six months.

But I would strongly urge that some cases require long and patient treatment; thus, Case vi recovered after nearly six months in hospital and five months at home, where she continued treatment in bed and on a milk diet. Case xvi required nearly four months, Case xxiv over three months, and Case xxxii twelve months before they were completely cured.

MODES OF TERMINATION.

The causes of death in this disease are numerous; in children it usually results from some inflammatory complication of the lungs or pleura, or uraemia. In adults uraemia is the more common cause, and then dropsy and its effects. But secondary inflammation of the serous cavities—notably pericarditis and peritonitis—may also be causes of a fatal termination, and pneumonia and pleurisy, though these are much less common than in childhood. Heart failure from dilatation of the hypertrophied left ventricle is also a cause, and was present in 2 of my fatal cases (Cases xxvii and xxxviii).

Of my 42 cases the results are as follows:

- 30 cured.
- 2 left *in statu quo*, and probably passed into a chronic condition.
- 6 left improved, of whom 1 (Case vi) recovered.
- 1 (Case xxi) passed into chronic nephritis, and the others have been lost sight of.
- 1 (Case xxxviii) has greatly improved, and is still in hospital.
- 3 died—2 of cardiac failure, one of which (Case xxvii) was associated with ascites, marked oedema, and some sloughing in the cellular tissues and skin.
- 1 of uraemia.

Total 42

RECURRENCES AND RELAPSES.

I believe that the more accurate the history of cases of this disease the more frequently will it be found that we are dealing with a recurrent attack. Thus, in Case xvi a recurrent attack occurred after thirty years, and in Case xxiv after twelve years.

Relapses occurring during the illness or convalescence are only too common, and are the cause of considerable anxiety. I will refer to this condition again under the heading of "Treatment."

There is, however, another condition which might be called subacute nephritis, as it is a very mild condition of nephritis, in which a patient who has previously had acute nephritis, and from which he has completely recovered, develops albuminuria, lumbar pain, and headache when unduly exposed to a chill or fatigue, and which passes off after a few days if judiciously treated by rest in bed, purgatives, light non-nitrogenous diet, and perhaps

mild diaphoretics. These attacks I have seen in Case xxxi.

I have tested the urine on several occasions during these attacks, and have found albumen, urates, and debris, but on only one occasion some hyaline casts. There is usually no dropsy. It is needless to say that the urine is quite normal between the attacks. Perhaps it is noteworthy that he is also subject to psoriasis, but the onset of the disorder is quite independent of any change in the skin lesion, and always follows some definite chill. For instance, once it followed a difficult instrumental confinement, in which he exerted himself for some hours, having doffed his coat. Probably this condition is not so rare as it appears, and some cases of those disorders called "lumbago" and "biliousness" may in reality be such a subacute nephritis in a person who has previously, at some time or other, suffered from acute nephritis; and some of the cases of functional albuminuria, or the albuminuria first detected at a life insurance examination may be of this nature.

TREATMENT.

The indications for the treatment of acute nephritis are: (1) To secure a rest for the inflamed organ, and to thereby diminish the risk of an increase of the inflammation; (2) to counteract the dangers which result from the interference with the excretory action of the kidney by encouraging the other organs—namely, skin and bowels—to act freely; and (3) to relieve the hyperaemia of the kidneys if possible.

The patient should be kept in bed between blankets, and clothed in a woollen nightgown, and he should not be allowed to get out of bed on any pretext whatever. His skin is thus more ready to act, all risk of a chill avoided, and his body is in as complete a state of rest as possible, metabolism being thereby diminished.

Diet.

1. To secure rest for the kidney the diet should be limited to one which will throw as little work on the excretory organs as possible, and undoubtedly the best is one which consists of milk only. For an adult in bed 50 oz. in the twenty-four hours is quite sufficient. It is best given in small quantities—4 oz. every two hours—and taken warm. If milk is objected to, or causes nausea or dyspepsia, it may be diluted with water, soda water, or potash water. When given in this way I have never seen any digestive disturbance arise, and all my patients have been treated on exclusively milk diet. Butter milk and skimmed milk might be substituted if desired, but they are not so nutritious. It is advisable to continue the use of milk far into the convalescence, and to very gradually return to a normal diet, by adding first a little carbohydrate, such as milk pudding, followed by bread and butter, and then a very little fish. Very frequently, however, relapses are brought on by adding to a milk diet; thus in Case ix the urine had been free from blood for nine days, but on the tenth it reappeared, the patient having eaten a few biscuits smuggled in by friends the previous day. Albumen recurred after six days' absence in the urine of Case xiii from the same cause, and Case xxx had a return of albuminuria and oedema the day following that on which he was ordered a little fish, and it was found that he had eaten heartily of the biggest sole that could be procured. Also in the convalescent stage great care should be observed to prevent a relapse, and for this reason my hospital cases attend the outpatient department weekly for about two months, and have their urine tested. In this way Cases xiii and xxv were discovered soon after their discharge to be suffering from albuminuria. They admitted having eaten freely of meat.

Because I have seen so much injury done by even slight additions to a milk diet I am very strongly averse to the views given in many textbooks on this subject. One writer says, "light animal broths should be included, thin beef tea or thin broth of other kinds"; another, "when the patient's appetite will permit, a more solid diet, consisting of easily-digested meats, light vegetable food, and good bread, may, of course, be adopted"; and another, though advising milk diet, says, "if necessary, beef tea and chicken broth." I cannot too strongly deprecate such views. I have yet to see the patient suffering from acute Bright's disease or any other complaint who

cannot live in bed for many weeks on a milk diet, and even thrive on it. And I am confident that if we desire to relieve the kidneys we cannot be too particular in avoiding a nitrogenous food. If the patient complains of thirst, Imperial Drink may be allowed. If the urine is scanty and very acid, 1 oz. of water containing gr. x of sodium bicarbonate and gr. x of potassium citrate may be added to each quantity of milk, as recommended by Sir William Roberts, or this may be given every four hours.

Drugs.

2. To counteract the dangers which result from interference with the excretory action of the kidneys, (a) purgatives and (b) diaphoretics are very beneficial.

The best *purgatives* are the hydragogue purgatives, which act by reducing the renal hyperaemia, lowering the blood pressure, relieving the oedema, and removing from the blood some of the toxic constituents. A good evacuation should be obtained at least twice a day. I generally find 1 drachm of compound jalap powder at night, or 3 or 4 drachms of magnesium sulphate or sodium sulphate in 1 oz. of water first thing in the morning most advantageous. Improvement under this treatment is noticeable. Mercurials should rarely be used, and then with the greatest care and followed by a saline. Bright, who used them at first, was the first to deprecate their use.

Diaphoretics, which by making the skin active, reduce probably to a slight extent the hyperaemia of the kidneys, diminish the oedema, and assist in removing from the body some of the waste products. Of these the tincture of jaborandi in 3ss to 3j doses, or the liquid extract of jaborandi in m̄v to m̄xv, are most useful. Also the liquor of ammonium acetate in doses of 2 to 6 drachms, and potassium citrate are recommended; but there is one drug which appears to be of the greatest benefit in all cases of acute nephritis, and that is sodium benzoate, and I always use it from the commencement in doses of 10 to 20 gr. It probably acts as a mild diaphoretic, a very mild diuretic, and most effectually as a disinfectant to the urinary tubules; for we know how useful it is in cases of cystitis, and what strong disinfectant properties the compound tincture of benzoin possesses. It is also believed to have the property of favouring the conversion of urates into less irritating and more easily excreted compounds. I prefer the sodium salt, though I have seen good results from ammonium benzoate; for the base of the latter must be excreted partly as urea and thereby throw slightly more work on the kidneys. I frequently prescribe:

R Tincture of jaborandi m̄xxx
Sodium benzoate gr. xv
Chloroform water 1 oz.
1 oz. every four hours.

If diaphoresis does not follow this treatment, hot air baths or the warm pack are most useful; the former is the safer, as there is less danger of the patient contracting a chill. When a poor result has been obtained, $\frac{1}{16}$ gr. of pilocarpine may be given hypodermically on the next occasion, and if necessary the dose may be gradually increased to $\frac{1}{8}$ gr. or more. As this drug also stimulates the bronchial glands, its effect on the lungs should be watched, and it should be given with caution in cases of uraemia, and never in any case in which lung complications are present.

Digitalis or other stimulating diuretics should never be used during an acute attack of nephritis, except only in cases of cardiac failure. In most uncomplicated cases they can only do harm by increasing the already high blood pressure and the hyperaemia of the too congested kidneys. Indeed their use is often followed by haematuria.

Counter-irritation.

3. To relieve the hyperaemia of the kidney, besides the methods already expounded, direct counter-irritation over the loins may be employed. This mode of treatment is especially useful in the early stages of acute nephritis, and it should always be used in cases in which there is suppression of urine. The various ways in which counter-irritation can be applied are (1) mustard poultices, (2) dry-cupping, and (3) wet-cupping. The two former are the ones now usually adopted.

Dry-cupping is the more preferable, and should be applied over both kidneys, and followed by hot poultices.

In this way counter-irritation may be maintained regularly with beneficial results, and in many of my cases the blood and albumen have considerably disappeared after a few days of this treatment. This also greatly relieves any lumbar pain. The cups I prefer are the large ones having a circumference the size of an ordinary teacup or glass.

Wet-cupping and leeches applied to the loins are methods not often used.

Sometimes, in cases of acute nephritis, in spite of the use of the usual therapeutic measures, certain symptoms persist and require special treatment. Thus, if headache, restlessness, and sleeplessness are troublesome, the administration of bromides and chloral are extremely serviceable, and can be given with safety if the pulse tension is good.

Morphine and opium, however, are contraindicated in all cases of acute nephritis, since they are not readily excreted by the damaged kidneys, and therefore produce a greater toxic effect, and, moreover, they may be followed by dangerous suppression and uraemia. Hyoscine hydrobromide and hyoscyamine sulphate are, however, free from these dangers, and may be given subcutaneously in doses of $\frac{1}{32}$ to $\frac{1}{16}$ gr. Persistent vomiting, except as an indication of uraemia, is not usually present in patients who are carefully dieted. If it should occur, a little bismuth is generally sufficient, and some physicians recommend dilute hydrocyanic acid. When the anasarca of the limbs is very great, it may be relieved by numerous small punctures or by the introduction of Southey's tubes through the skin. Every aseptic precaution must be observed; also, dropsy into the serous cavities should be removed by tapping. In the case of ascites this is frequently followed by a greater flow of urine; such occurred in Cases XII and XVII. During convalescence anaemia requires mild preparations of iron. They must be given with great care and the astringent preparations should be avoided. It is important to warn a convalescent patient to avoid all risks of chill, undue exertion, and a too nitrogenous diet.

URAEMIA.

Uraemia, the actual cause of which we do not yet thoroughly understand, is undoubtedly a series of symptoms depending on a toxæmia due to an auto-intoxication dependent on the loss of function of the kidney. Its onset may be sudden, but it is often indicated by such symptoms as dull headache, drowsiness, vomiting, and convulsions; and these must be immediately opposed by vigorous measures, that is, free purgation, free diaphoresis, and counter-irritation over the loins should at once be practised on the lines already indicated. If, however, the uraemia be well established, and coma and convulsions, etc., are present, the bowels must be moved by half to one minim or more of croton oil applied to the tongue, and sweating may be brought about by pilocarpine injected under the skin, assisted by hot blankets and bottles to the body, and hot mustard poultices over the loins. Such treatment relieved the coma and convulsions of Case XXXIV, though they had persisted for over twenty hours, and the patient had passed only a few ounces of highly albuminous and very bloody urine.

Treatment on these lines also relieved the uraemia in Case XXXVII on several different occasions, but in the end he succumbed. Undoubtedly, from the many recorded cases, uraemia is often expelled by the withdrawal of 10 to 20 oz. of blood from a vein, and the injection of some warm normal saline solution; but before giving the credit of any instance of recovery to a particular method of treatment we must remember that cases with very severe symptoms of several hours of unconsciousness and the almost total suppression of urine do sometimes spontaneously recover. If it were not for this fact I would give the credit of the recovery of Case XXXIV to the use of pilocarpine. Nevertheless, judging from the facts that this condition is a toxæmia, the analogy of the treatment of diabetic coma, and the beneficial results already attained, this method of saline injection into the veins is certainly one of the many measures of treatment which should always be adopted where possible. The inhalation of chloroform is specially useful in relieving those cases in which asthma or convulsions are the predominating symptoms, and bromides may be given if the patient is not comatose. In conclusion, I would reiterate the importance of

prolonged treatment in the tedious cases of acute nephritis.

REFERENCES.

¹ *British and Foreign Medico-Chirurgical Review*, 1854. ² *Pathology and Treatment of Albuminuria*, by W. H. Dickinson. ³ *Allbutt's System of Medicine*, vol. iv, p. 358. ⁴ *Ibid.*, p. 359. ⁵ *Von Ziemssen*, vol. xv, p. 284. ⁶ *Ibid.*, p. 277. ⁷ *Berliner klinische Wochenschrift*, 1873, No. 50. ⁸ *Allbutt's System of Medicine*, vol. iv, p. 368. ⁹ *Von Ziemssen*, vol. xv, p. 258.

Presidential Address ON FOOD AND FOOD PRESERVATIVES:

THEIR INFLUENCE ON THE INCREASED
PREVALENCE OF CERTAIN DISEASES.

DELIVERED BEFORE THE ANNUAL MEETING OF THE
NORTH WALES BRANCH OF THE BRITISH
MEDICAL ASSOCIATION.

By RICHARD JONES, M.D. EDIN., D.P.H. CAMB.,
PRESIDENT OF THE BRANCH.

GENTLEMEN,—There can be no doubt that the health of the public suffers by the enormous consumption of tinned and preserved meats now offered to the public. This injurious effect may be due to one of two causes, or, probably a combination of both.

1. Meat that is diseased or becomes diseased before consumption.

2. The adulteration or the addition of preservatives to the food, either to preserve or to disguise its diseased or unwholesome character.

It does not follow that all adulterated foods are injurious to health—for example, butter may be adulterated with margarine—the same may be said of milk and water, wheat and potato starch; such adulterations are nevertheless, a fraud, and punishable by certain Acts of Parliament.

But it is seldom that we find butter adulterated with margarine without the addition of a preservative and colouring matter. This is commonly boric acid or borax and annatto. It is the addition of the preservative that makes it possible (in many instances) to place diseased meat, milk, or fruit upon the market.

Preserved peas are often, if not always, adulterated by the addition of copper sulphate, both as a preservative and a colouring reagent. Here we have an adulterant that is also poisonous.

The revelations connected with the Chicago tinned meat trade have been brought prominently before the public; possibly too much is being made of this, and it would be well to bear in mind what the late Sir J. Simon, the pioneer of public health in Great Britain, said in the Eighties, when referring to a report of Professor Gamgee on meat inspection in this country:

Horned cattle affected with pleuropneumonia are much oftener than not slaughtered on account of the disease, and when slaughtered are commonly (except their lungs) eaten, and this even though the lung disease has made such progress as notably to taint the carcass; that the presence of parasites in the flesh of an animal seldom influences the owner against selling it for food; that carcasses too obviously ill-conditioned for exposure in the butcher's shop are abundantly sent to the sausage makers, or sometimes pickled and dried; that some sausage makers will utilize even the most diseased organs which can be furnished them; that the principal alternative, on a large scale, to the above-described human consumption of diseased carcasses is that, in connexion with some slaughtering establishments, swine are habitually fed on the offal and scavenger of the shambles, and devour, often raw and with other abominable fifth, such diseased organs as are below the sausage-maker's standard of usefulness.

Many changes have since taken place, all tending to minimize the possibilities of such practices being resorted to in these days, with what success is often questionable.

Diseased meat has its danger, and is undoubtedly the cause of much illness and death; but I much doubt whether the possibility of the contents being diseased is the only danger we have to face when consuming preserved and tinned meats.

To-day I propose bringing before you the large question of food and food preservatives, and their influence on the increased prevalence of certain diseases.

In 1899 a Departmental Committee was appointed to inquire into the use of preservatives and colouring matters in the preservation and colouring of foods. In the report of this Committee, published in 1901, it is stated that "we cannot overlook the danger to which the uncontrolled use of drugs in the food of the population may be likely to give rise."

In dealing with the use of preservatives in milk they come to the conclusion that no preservatives or colouring matter whatsoever should be allowed. They state that:

The farmer or producer sometimes applies it; so does the wholesale purveyor; so does the retail dealer; lastly, the domestic use of preservatives is increasing, and has become very general, and hence the milk may receive a fourth dose before it reaches the unsuspecting consumer.

And further that

One of the considerations which renders it expedient to prohibit the use of any preservative in milk offered for sale—namely the large quantity which may be taken into the system of the consumer—places, in our opinion, wine, cider, and temperance beverages upon a very similar footing.

Moreover, while by far the greater proportion of preservatives used in the dairy industry consists of compounds of boron, it is otherwise with fermented and temperance drinks. The usual preservatives in these articles are salicylic acid and formaldehyde, and although the quantity of each actually required is very small, it is often largely exceeded.

The report also states that it is

Highly undesirable to use copper sulphate, even in the smallest quantity, to preserve and colour peas and other vegetables.

The extent to which the use of preservatives and colouring matters become possible will be seen from the following table relating to the importation of food and foodstuffs into the United Kingdom during 1905.*

Imported Foodstuffs, 1905.

	Quantity.	Value in £.
Butter	4,147,866 cwt.	21,586,622
Cheese	2,442,682 "	6,339,811
Cocoa butter	685,720 lb.	28,544
Fish (canned)... ..	667,450 cwt.	1,800,126
" (uncanned)	"	1,339,328
Peas (not fresh)	2,015,876 cwt.	725,104
Beans (all kinds)	1,444,939 "	535,519
Wheat (meal and flour)	22,576,430 "	6,044,845
Corn and grain (of all kinds)	207,930,766 "	70,057,290
Fruit (dried and preserved)	2,152,339 "	2,375,336
" (other than preserved)	"	10,270,584
" (canned—no sugar)	525,139 cwt.	326,286
" juice	783,000 gal.	74,645
Margarine and margarine cheese	967,793 "	2,735,785
Meat (frozen, salted, and preserved)... ..	18,679,507 "	38,487,409
Fresh milk	49,640 "	100,517
Mineral waters	1,410,330 doz.	308,619
Oils (refined and edible)	471,080 cwt.	500,000
Oleo-margarine	254,312 "	495,212
Poultry and game	"	1,000,000
Chicory	1,914,572 lb.	38,606
Cider	385,044 gal.	15,188
Cocoa	63,936,974 lb.	2,256,389
Ice	345,332 tons	165,030
Stearine... ..	1,822,819 cwt.	2,369,386
Albumen	"	37,792
Sauces and condiments	342,907 lb.	40,044
Spices	"	783,004
Spirits (tested)	7,630,765 gal.	1,821,297
" (not tested)	59,441 "	131,342
Sugar (refined and unrefined)	29,352,549 cwt.	19,471,811
Molasses	2,537,999 "	502,324
Glucose... ..	1,410,557 "	608,553
Caramel, cherries Chutney, confectionery (hard and soft)	160,000,000 lb.	2,779,757
Canned, bottled		
Crystallized fruit		
Condensed milk	280,002 cwt.	290,164
Preserved fruit of all kinds		
Vegetables (preserved, canned) (raw)	2,536,280 gal.	3,889,74
Pickles	302,486 "	120,262
Vinegar... ..	309,034,959 lb.	17,671
Tea	12,731,050 gal.	9,307,235
Wines	18,662,078 lb.	4,072,199
Coffee	56,605 bbls.	2,578,371
Beer	"	153,430
Caseine... ..	"	34,362
Eggs	18,814,293 h'ds.	6,812,436
Lard	2,206,390 cwt.	3,961,671
Methyl alcohol	466,051 gal.	54,262
Kernels... ..	51,522 tons	771,900

Living animals, destined to be used as food, are not included; these numbered 748,373, and were valued at £9,904,879.

The value of the condensed milk imported during 1905 was over £1,000,000.

* Board of Trade returns.