immunity from recurrence for three years after operation as being practical evidence of a cure is based on the statement that a large majority of the recurrences occur within three years—and, indeed, 80 per cent. are said to recur in the first year after the incomplete operation. Now in Tables A. and B., which may be taken to be an average class of cases, local recurrences took place in 26 out of the 35 cases—i.e., 74 $\frac{2}{3}$ per cent. Of these 8 recurred within a year=30 $\frac{1}{3}$ per cent.; 18 in all had recurred in three years=69 $\frac{3}{8}$ per cent.; leaving 8 cases, or 30 $\frac{1}{3}$ per cent., which recurred after three years. Now in these cases the operation was one which is now regarded as incomplete; it is therefore reasonable to conclude that the radical operation at present advocated would be followed by a larger proportion of cases recurring after three years.

To show the possible effect of such a time limit on statistics I place side by side the total percentage of cures and failures in Tables A. and B., reckoned first without and secondly with a three years' limit, on the presumption that after three years' immunity the cases had been lost sight of or not followed up.

Without any limit.	With a three years' limit.
29 failures out of 35 cases. = 82 $\frac{2}{3}$ % failures. 17 $\frac{1}{3}$ % cures.	20 failures out of 35 cases. = 57 $\frac{1}{3}$ % failures. 42 $\frac{2}{3}$ % cures.

With the time limit Nos. 2, 4, and 8 in Table B., and Nos. 11, 14, 16, 23, 24, and 25 in Table A., would have to be put down as cures.

Stratford-place, W.

INDIGESTION AND BRIGHT'S DISEASE.¹

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THE association of symptoms of indigestion with acute or chronic Bright's disease is constantly coming under observation. Very commonly the persistence of the symptoms of indigestion or the accompanying attacks of vomiting are the immediate causes of an examination of the urine, and they may thus be the first warning notes which lead to the establishment of a diagnosis of Bright's disease.

Vomiting, which is often a premonitory symptom of acute uræmia, may in more chronic cases continue for weeks or months in spite of all that is done to arrest it. It may defy every dietetic precaution and it may resist all forms of medicinal treatment. Mostly it is associated with troublesome constipation, but it may occasionally be accompanied by diarrhoea of an equally intractable description. Sometimes these symptoms may give rise to a suspicion of malignant disease affecting some part of the alimentary canal. Some twelve years ago when attending a gentleman who had exhibited well-marked symptoms of chronic kidney disease, an inter-current attack of vomiting led me to seek the advice of a leading consultant, and I had the greatest difficulty in persuading him that transient fulness and sense of resistance in the region of the cæcum were not permanent conditions and evidences of malignant disease. In view, therefore, of the great liability to error I venture to lay stress upon the importance of extreme caution when confronted with vomiting, diarrhoea, or both, in connexion with albuminuria. A little delay while watching the progress of a supposed tumour may prevent the anxieties attaching to a diagnosis of malignant disease. The tumour may shift its position and under appropriate treatment may disappear entirely, as indeed it did in the patient I have already referred to. True uræmic vomiting very frequently occurs in the early morning, before food has been taken, and in old standing cases the vomit has

often an alkaline reaction and an ammoniacal odour. It may, however, occur at any time during the day without any relation to the time at which food has been taken, and, as in a patient lately under my care in King's College Hospital, the vomited matters may be little else than deeply bile-tinged mucus, sometimes of a vivid green colour. In the case in question the vomiting continued for some four or five days before the end, and the clinical features were in every respect those of advanced chronic kidney disease from alcoholism.

The condition I have described is usually a prelude of more serious uræmic symptoms, but the digestive system commonly shows signs of impaired activity, which do not necessarily lead up to actual vomiting. Patients with chronic kidney mischief frequently exhibit a thickly furred tongue and suffer from anorexia. They find by unpleasant experience that they have to be careful in their mode of living, that many common articles of diet disagree with them, and that they are prone to flatulent discomfort, eructations and other evidences of chronic gastric catarrh which sometimes leads to considerable prostration. The relationship of these two conditions is not, however, always easy to follow. Sceptics are not wanting who urge that many persons are dyspeptic without having Bright's disease, and that the two conditions may run side by side in the same case without any necessary connexion. Although this is undoubtedly true for some cases, true renal dyspepsia seems to be marked by an intractable character which lends a distinguishing feature, and most authors admit that chronic dyspepsia and vomiting form part of the phenomena of chronic uræmia; but, when from other indications we are convinced that Bright's disease has long existed in the case before us, and when the nature of the pulse, of the sounds of the heart, of the skin, and, perhaps, of the retina, all point in one direction, we may still find great difficulty in stating definitely in what way Bright's disease has caused the apparently consecutive dyspeptic trouble. For practical purposes vomiting and dyspepsia may be considered together, the former being regarded as the most obvious symptom and evidence of the latter. It has been suggested that in these cases the wall of the stomach partakes of the general œdema and that, as a consequence, its functions are impaired. This suggestion is one which it is most difficult to prove and almost equally difficult to disprove. The condition described can scarcely be determined on the post-mortem tables, since it is quite conceivable that œdema of a mucous membrane sufficient to produce symptoms during life might leave no trace after death; further, it must be noted that the dyspepsia of chronic kidney disease is often, as I have already said, the first symptom to attract attention; it frequently precedes any indication of œdema or dropsy in other parts of the body. Even if we admit that symptoms of dyspepsia might and should result from œdema of the wall of the stomach, there appears to me to be much more against the œdema theory than in its favour. As generally recognised, œdema either affects the whole of the body when it results from some altered state of the blood, or it is localised when it is consecutive to some alteration in the blood current, whether produced by alterations in the vascular wall, or by some disease of the heart or lungs leading to diminished rate of movement of the blood. Now, an œdema of the stomach without indication of other dropsical signs would not fit into either category, and it appears to require too great a stretch of the imagination to be considered at greater length.

Another suggestion, which is also difficult to prove, although it appears more probable, is that the vomiting and dyspepsia are due to some eliminative effort by which the deficient work of the kidney is to be performed vicariously. I have already referred to the alkaline reaction and ammoniacal odour of the vomited matters in chronic and persistent cases of renal vomiting. These certainly seem to point to eliminative action, and it is quite conceivable, I think, that in milder cases the eliminative processes may produce a form of gastritis. It is generally admitted that certain drugs may cause vomiting during their elimination owing to a local irritative effect upon the mucous membrane of the stomach. For example let me remind you of the three-fold emetic action of tartar emetic; how it produces an early act of vomiting owing to its direct action upon the wall of the stomach, how it is speedily absorbed and then again, by its influence on the medulla, causes vomiting, and how it may yet cause a third or prolonged bout of vomiting and nausea during the time of its

¹ A paper read before the Nottingham Medico-Chirurgical Society on April 29th, 1896.

excretion by the mucous membrane of the stomach. That these are not fanciful theories has been shown by Magendie's conclusive experiment of replacing the stomach by a bladder and injecting the tartar emetic into the blood. In further support of this idea of the influence of elimination in determining indigestion and vomiting, stands the clinical fact of the relief of symptoms which may occasionally result from free purgation. This relief is seen most markedly in cases of acute nephritis, as for example in those occurring after scarlet fever, when several watery evacuations are commonly followed by a general improvement, the headache and insomnia pass away, the oedema diminishes, and after a time the kidney resumes its previously checked functions. The relief afforded by purgation is not, however, limited to these cases of acute scarlatinal nephritis, it is sometimes seen in cases of chronic kidney disease, and it accounts for the frequency of the routine administration of the compound jalap powder. In such cases, however, we must not assume too hastily that the benefit of this purgative action is solely or even mainly due to vicarious elimination of nitrogenous matters through the channel of the bowel. It is more probable that the free removal of fluid diminishes the renal engorgement and that, by relieving the strain upon the renal vessels, it produces a condition of temporary physiological rest which enables the damaged kidneys to recover and regain their interrupted functions.

While recognising the important indications afforded by dyspeptic troubles in the course of chronic and acute disease of the kidney, it is well not to overlook the influence of various gastric derangements in the *production* of kidney disease. Dyspeptic troubles are not always the sequel; they may play an important part as a predisposing cause. It is more particularly to this aspect of the association of the conditions "dyspepsia and Bright's disease" that I wish to invite attention.

The term "digestive albuminuria" is frequently met with in literature, and it seems to imply that the albuminuria is due to some interference with the digestive powers. The influence of diet in causing albuminuria in healthy people has been frequently asserted and frequently denied, but doubtless we have all met with cases of albuminuria in young men which have appeared to depend upon some dietetic errors. I remember one such case in a young city man who was trying to live on two meals a day, and who was in the habit of taking enormous breakfasts, which often consisted of chops and always concluded with two or three boiled eggs and then marmalade. The albuminuria entirely disappeared with a more rational dietary. Numerous similar cases have been reported by other observers, notably by Grainger Stewart and Brunton, and the connexion with errors of diet is now so well recognised that recently, in insurance work, a man with tense arteries and renal aspect tried to apologise for his albuminuria on the ground of some hard boiled eggs at breakfast that morning. (It is, perhaps, needless to add that I accepted his apologies and acted upon my own observations.) It cannot be doubted, however, that the existence of oxaluria or of uric acid lithiasis is a very common source of chronic kidney trouble. The frequency with which gout is followed by chronic nephritis has long been recognised, and it is worth bearing in mind that dyspepsia is an extremely common prodromal symptom of gout. The frequent irritation of the kidney by oxalates or by uric acid cannot fail to be followed by permanent changes. Individuals living in large cities, leading an inactive life and taking large quantities of food, are very prone to be troubled in this way, and in these people the foundation of the chronic kidney trouble is undoubtedly to be found in the long-continued dyspepsia.

Again, the association of chronic alcoholism with interstitial nephritis is well recognised, and it must be remembered that one of the leading symptoms of chronic alcoholism is dyspepsia. In our hospital work we all know the probable causation of frequent slight diarrhoea, nausea, and distaste for food, especially in the morning, a collection of symptoms commonly exhibited by Covent Garden porters and sometimes by cabmen; and we are familiar with the deprecatory smile and the final grudging admission of "a tidy drop." Now these cases are laying the foundation of chronic kidney mischief, not so much in irritation of the kidney by the alcohol, since, as you know, a large proportion of the alcohol is eliminated by the lungs, as by the dyspepsia which results from their habits. Most of these men are badly nourished owing to their distaste for food; most of them are melancholic from a similar cause; and a

large number pass urine from which a copious collection of crystals may be obtained. They are subject to repeated attacks of gravel, and in this way, in all probability, Bright's disease is started by the mechanical irritation of the lining membrane of the tubules of the kidney.

That it is not always easy to determine the true course of events may be shown by brief notes of the following cases. A middle aged man told me he had albuminuria, and at the first examination of his water it was very pale and I could only find a faint trace of albumin. He was disposed to date his troubles from an attack of influenza, but he had a thickly-coated tongue, his teeth were very bad, and he said he had frequently had much dyspeptic trouble. His mother died from Bright's disease. In this case I believe the sequence of events to stand something like this: (1) hereditary predisposition; (2) dyspepsia from deficient power of mastication; and (3) consecutive kidney trouble. From the vagueness of the account it is obvious, however, that the position of the last two conditions might be reversed, but the absence of arterial changes and of alteration in the sounds of the heart lead me to think that the albuminuria and the kidney trouble were in an early stage.

To give another example of the production of renal trouble through the digestive system. Two years ago I saw a patient with an intermittent heart and a florid complexion, and at that time there was no albumin in his urine. I thought then that he drank too much alcohol, and my suspicions were confirmed later by the development of an alcoholic aspect, a furred tongue, and by finding a trace of albumin in his urine which had a specific gravity of 1020. This case I think must be recognised as illustrating the evolution of chronic kidney disease produced by the irritative action of alcohol upon the digestive system.

One of the most interesting cases of this nature which has come under my observation is that of a man, whom I have seen at long intervals during the past ten or twelve years. He was frequently troubled with gravel and passed beautiful stellate crystals and often described the irritation they caused. He had a strongly marked family history of renal trouble. In spite of repeated warnings of the dangers incurred he made little or no change in his habits, and of late after sundry well-marked gouty attacks his urine, whenever I have examined it, contains a trace of albumin. Whether the albuminuria is simply due to the continued irritation of the kidney or the pelvis or ureters by the stellate crystals, or whether this irritation has yet led to definite structural alterations in the kidney, I hardly know. He has no retinal changes, his aspect does not present any characteristic pallor, and his pulse does not show any high tension, except during the attacks of gout. Still, one cannot help feeling certain that the future of this case will be marked by the gradually increasing discomforts and dangers of chronic renal disease.

In endeavouring to estimate whether the renal trouble—the albuminuria—is due to true structural disease or only to some transient and possibly unimportant irritation it is well to determine the nature of the albumin, but very often examination of the pulse, the heart, and the eye afford indications of greater value. For example, a woman aged thirty-five years was recently brought to me by Mr. Philip Birch with the following history. Six years ago, during her first pregnancy, she had cystitis, which lasted two years and was accompanied by a trace of albumin which seemed just more than could be accounted for by the pus present. Then she became pregnant again and lost all trace of cystitis and albumin. Last summer she had an intermittent pulse, and in spite of treatment the pulse had not regained regularity. Her digestion seemed fairly good, but she was very pale and had a characteristic subconjunctival tear. The pulse tracing showed marked high tension as well as intermission, and although I could get no reaction with nitric acid or picric acid, a faint line of opacity soon formed with trichloroacetic acid and demonstrated the cause of the intermittent pulse.

A somewhat similar case—a man aged thirty-six years—was sent to me by Mr. Stanford Morton, who described hemorrhages and patches of fatty degeneration in his fundi, which gave all the appearance of albuminuric or diabetic retinitis. The urine in this case had a specific gravity of 1020, gave no reaction for sugar, and no reaction with nitric acid or picric acid, but only a faint cloud with trichloroacetic acid; but the pulse tracing and the sounds at the base left no doubt that we were dealing with a case of chronic kidney disease in the non-albuminuric phase.

Such cases almost speak for themselves, and even in the

relative absence of albuminuria, one has no difficulty in attributing them to chronic Bright's disease. They are in the stage of quiescence—the stage so often recognised of chronic renal disease *without albuminuria*. The absence of albumin does not remove them from the category of chronic renal trouble. I even venture to doubt whether since Bright's discovery of the association of albuminuria with renal changes the true significance of many cases has not been obscured, rather than helped, by the constant search for albumin. To take only one aspect, that of life assurance, I believe that by many medical examiners both these cases might have been passed; and I fear that in testing for albumin we may perhaps be tempted to devote too little attention to the appearance of the patient and the character of the pulse. Further, I think that the true bearings of these cases might readily be overlooked if at a later stage they presented themselves for the treatment of dyspeptic troubles. The dimness of vision of which the man complained might easily be referred to dyspepsia, while an intermittent pulse is, according to many teachers, of so little moment that, if the primary cause of complaint appeared to be dyspepsia, the intermission might probably be regarded as a passing vagary scarcely worth noting.

From what has been said it will be seen that I consider it most important, when dealing with dyspepsia and albuminuria, to endeavour to form an accurate idea of their relation to one another. Not only the prognosis, but also the treatment, depend largely upon a correct recognition of whether the indigestion is the result or the exciting cause. If it is the result the nature of treatment must vary somewhat with the urgency of the symptoms. In any case free purgation is essential, and the use of hydragogue purgatives is to be preferred. We should never attempt to stimulate the action of the kidney directly by diuretics, even when the amount of urine passed is much below the normal quantity. If the nature of the urine indicates acute engorgement our object should be to promote physiological rest, to diminish the amount of work required of the kidney by removing water from the system by other channels. Frequently, in severe cases, the use of the wet pack or the vapour bath becomes an all-important factor in the treatment of renal vomiting. If we recognise at an early stage the true renal nature, our treatment is more likely to be serviceable than if we confine our attention to the condition of the stomach. Regulation of diet and the use of gastric sedatives, although they should not be neglected, are not likely to give good results unless at the same time we treat the underlying cause of the symptoms. On the other hand, if the symptoms are not so urgent, and if we have reasonable cause for suspecting that the albuminuria is the result of the dyspepsia, our course is equally clear. We must endeavour to ascertain whether the trouble is merely dietetic or whether it is oxaluric or lithæmic, but I will not be wearisome by giving any further details of a subject so simple. The main thing is to regulate the diet and by careful study of the individual case to determine what constituent should be removed. Sometimes it is a mere question of distribution, preventing overloading of the stomach on one or two occasions during the day. Sometimes we find it necessary to eliminate eggs or pastry or cheese; in oxaluric cases we remove sugar and reduce the vegetable acids, and so on. The treatment really presents no difficulty when once a satisfactory diagnosis has been made.

To state briefly the conclusions to be drawn from the foregoing observations, although I would by no means underrate the importance of testing the urine in every case of chronic dyspepsia occurring in adult life, so as to run no risk of overlooking an existing Bright's disease, I venture to lay stress upon the frequency with which long-continued dyspepsia stands in the position of predisposing cause, rather than as the result of chronic kidney disease.

Harley-street, W.

KENT BENEVOLENT MEDICAL SOCIETY.—The annual meeting of the members of the Kent Benevolent Medical Society was held last week in the library of the Kent and Canterbury Hospital. Mr. A. Long of Dover presided, and the accounts for the year showed that of £421 13s. 1d. which had been received during the past twelve months (including a legacy of £50 from the late Mr. John Marshall of Dover) a sum of £371 14s. 1d. had been expended. The meeting unanimously resolved that £100 out of the balance in hand should be added to the invested capital of the society, and that the sum of £305 should be distributed amongst the applicants whose names were before the meeting.

A MODIFICATION OF DR. FOWLER'S METHOD OF STERILISING CATGUT.¹

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THE perfect sterilisation of catgut is such an important point in modern surgery that the following method of accomplishing it may be of interest to surgeons. The mechanical details of the process are new as far as I am aware, but the principle was originated by Dr. Fowler, who sterilises catgut by sealing up suitable lengths of it in a strong horseshoe-shaped glass tube containing alcohol, the tube being then raised to whatever temperature is thought advisable either by heating it in some liquid of sufficiently high boiling point or by placing it in a dry steriliser. Catgut, owing to its origin and mode of preparation, is essentially a septic material. It is made by twisting into cords the specially prepared submucosa of the small intestines of the sheep, and the twisting process has the effect of bringing bacteria, which at first were on the outside of the intestine, into the centre of the threads. The fat which is present then glues the different strands firmly together and encloses the bacteria in a sheath which is impermeable to the ordinary means of sterilisation. When, however, the catgut is introduced into the body, during the process of absorption every particle of it is layer by layer brought into contact with the surrounding tissues, so that while bacteria contained in silk or silkworm gut may never come into contact with the tissues every bacterium present in catgut is of necessity obliged to do so, and it is this property of being absorbed which makes the absolute asepsis of catgut so imperative.

The chief methods of rendering anything aseptic are—(1) the removal of germs by mechanical means, and (2) the destruction of them by germicides. The first of these is useless as far as catgut is concerned, and we must rely upon germicides, which can be divided generally into—(1) certain chemical substances and (2) heat in some form. Chemical methods are all placed at a great disadvantage owing to the bacteria in catgut being encased in fat. They mostly depend on first removing as much of the fat as possible by soaking the catgut in ether and then subjecting it to the prolonged action of an antiseptic, but all these methods necessitate very frequent changing of the solution, they are exceedingly expensive, and they are very far from certain, for cultivations have often been grown from catgut which was believed to be absolutely pure. Heat is very much more certain in its operation than chemical antiseptics, for boiling water will destroy anthrax spores in two minutes, while even 1 per cent. solution of corrosive sublimate will require several hours for the same purpose. Boiling water or steam, however, cannot be applied to catgut, as it swells in them and becomes a shapeless mass, while dry heat is difficult to manage and requires very many precautions in its use, else the catgut becomes brittle and useless. In short, a medium is required in which catgut can be heated without destroying its strength, and this is best furnished by alcohol, which, however, boils at 173° F., and as this temperature is not sufficient the alcohol must be super-heated.

By Dr. Fowler's method already described the catgut is raised to 212°, the bacteria are destroyed, and at the same time the gut is kept strong and serviceable, but the surgeon is under the necessity of buying catgut already prepared and sterilised. I venture, therefore, to make known the following slight modification of Dr. Fowler's method, as it enables the sterilisation to be easily and quickly performed by anybody. The catgut, instead of being sealed up in glass tubes, is rolled on glass plates and placed in a special brass box three-quarters full of alcohol, and the whole is then immersed in boiling water for the necessary time. The box is strong enough to stand the required pressure—viz., a force of about four pounds to the square inch. It has a screw-top provided with an indiarubber washer and projecting arms above and below to facilitate screwing it up or opening it. The catgut must be absolutely dehydrated and must not

¹ A paper read before the Obstetrical Section of the Royal Academy of Medicine in Ireland on April 6th, 1896.