Remarks ON ACUTE NEPHRITIS.*

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INCIDENCE.

It is generally believed that acute inflammation of the kidneys is a rare disease. In civilian practice it is certainly rare, except as a complication of scarlatina. The figures given by Herringham in his book on kidney diseases (1912) are often quoted. He states that only 166 cases were treated at St. Bartholomew's Hospital out of about 63,000 admissions during a period of nine years.

Reference to the Registrar-General's reports shows that in the five years 1922 to 1926 inclusive the total number of deaths from all causes registered in the Irish Free State was 314,829. Of these, the total number registered as from acute nephritis was 1,362, of which 645 were in females and 717 in males. These figures include all deaths from acute nephritis in adults and all deaths from nephritis in children under 10 years of age, but do not include deaths from eclampsia. These figures show that one death in every 231 registered is reported as due to acute nephritis. The deaths registered as due to chronic nephritis during the same period numbered 6,143.

In England and Wales during the same years (1922-26) the total number of deaths from all causes registered was 2,231,445. These included 6,964 classified as acute nephritis, of which 3,288 were in females and 3,676 in males. This gives one death from acute nephritis in every 334. The deaths registered as due to chronic nephritis during the same period numbered 56,146. If we assume that there is an immediate 10 per cent. mortality from the acute disease (and this is probably higher than what actually occurs) the number of cases treated in any one year in either country can be easily calculated.

Owing to the comparative infrequency of the disease no systematic study of groups of cases was possible until the great war. Then, as is universally known, nephritis occurred in epidemic form, and under conditions where close observation, both of immediate symptoms and remote consequences, was possible. The result is that our knowledge of the disease has greatly increased of recent years, and we now perhaps realize more fully than formerly that many deaths recorded as due to chronic nephritis are the late sequelae of acute nephritis.

PROPHYLAXIS.

The best way to avoid an attack of acute nephritis is to shun those conditions which render the individual specially liable to it; but in spite of every effort many people will continue to suffer from scarlatina and other infective conditions, and many will be far from anxious to avoid pregnancy. The question, then, arises as to whether anything can be done to decrease the incidence of nephritis in such conditions. It is quite unnecessary to refer to the common-sense precautions which ordinary clinical experience will dictate, and which should, above all, be observed by those already suffering from chronic renal disease.

The recently acquired knowledge that septic tonsils and septic nasal sinuses may be the source of toxins which can produce acute damage in the kidneys suggests at once that the removal of such tonsils and adequate treatment of such nasal diseases may be looked upon as a preventive measure. In scarlatina it was the fashion some twenty years ago to prescribe urinary antiseptics of the hexamine type, and many statistical records were published to show that the incidence of nephritis was lessened in cases in which these drugs were given. The passage of time has not confirmed these records, however, and few clinicians now believe these antiseptics to be of any value.

A new impetus to preventive measures has been given by the researches of Osman, who, as a result of careful study in collaboration with others, has stated recently that the incidence of nephritis after scarlatina is greater in those whose urine is unduly acid and in whom the alkali reserve of the blood is diminished. He and his co-workers also claim to have reduced the incidence of nephritis after scarlatina to 0.6 per cent. in 620 cases treated by alkalis, as compared with a 5.5 per cent. incidence in 316 cases not so treated. If these results are confirmed it may be advisable to employ alkalis in future in all cases of scarlatina, and to extend Osman's observations to other diseases, not excluding pregnancy, in which nephritis is a frequent incident. Large doses are, however, required, and it is possible that many patients will object to their consumption. At present Osman's conclusions must be received with hope, but with caution. Peters, in an analysis of nearly 8,000 cases treated between 1910 and 1927, found no significant difference in the onset of complications in those treated with and without alkalis respectively, and Stieglitz goes so far as to say that excessive intake of alkalis may actually predispose to nephritis instead of the reverse.

The value of scarlatinal antitoxin is still *sub judice*, but the reports up to date are fairly satisfactory. Gardner Robb states that in 100 cases of average severity treated by serum no case of nephritis occurred, and there is no reference in his paper to even the occurrence of albuminuria. On the other hand, Dr. Burns, a former pupil of mine, tells me that the experience at the City Hospital, Fazakerley, Liverpool, is that scarlatinal antitoxin does not diminish the incidence of complications.

CLASSIFICATION.

Innumerable attempts have been made to classify the different forms of nephritis, but fortunately the limitation of my subject to acute nephritis renders it unnecessary to attempt anything but the very simplest; indeed, from my point of view, much time has been wasted in the attempts that have been made to divide and subdivide, instead of recognizing the unity which underlies the varying pathological manifestations of the acute condition. It is, however, necessary to state that under the term "acute nephritis" I include not merely acute non-suppurative inflammations of the kidney, but also acute non-suppurative degeneration. In short, I do not intend to use the word "nephrosis" as indicating a condition distinct from acute nephritis. The term "nephrosis," indeed, has not been generally adopted by English writers, though it is employed largely in Europe and the United States to designate a clinical and pathological entity of acute origin, characterized by primary degeneration of tubular epithelium, unaccompanied by inflammatory phenomena. Even if this condition does exist in the pure state, I am convinced that it is wiser, both for the sake of clarity of thought and also for diagnostic and teaching purposes, to include it merely as a variety of acute nephritis. This remark, of course, applies to the condition known as toxæmic kidney, a term which is becoming increasingly frequent in the English literature to describe a state identical with nephrosis.

Classifications have been attempted on the basis of etiology, pathology, clinical symptoms, and on that of disordered function. Provided, however, that we remember that the kidney is composed of four elements—glomerular, tubular, interstitial, and vascular—and that each element may be involved in a varying degree according to the cause of the inflammation, its severity, and its duration; and provided we remember that the functional changes are likely to vary also with the degree of involvement of each of these structures, and the severity of that involvement, we will do better as clinicians to recognize one form of acute nephritis only. The only exception that I can see to this view is the acute nephritis which is met with as a complication or symptom of ulcerative endocarditis. It must, of course, be remembered that acute inflammation may attack not merely a healthy kidney, but also a kidney damaged to a greater or lesser extent by previous disease.

[3533]

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SYMPTOMATOLOGY.

The symptoms are too familiar to require enumeration. They vary in severity from transitory febrile albuminuria to the most acutely fulminant manifestations. It is customary to distinguish a variety characterized mainly by oedema from a variety in which haemorrhagic urine is the pre-predominant symptom, and it is even contended that parenchymatous lesions with resultant oedema are more particularly likely to be produced by staphylococcal toxins and glomerular lesions with haemorrhage by streptococcal toxins. The evidence for this is, however, far from convincing, and my own experience is that oedematous and haemorrhagic cases are by no means invariably clear-cut. A rise in blood pressure was at one time regarded as almost certain evidence of old-standing nephritis. It is now, however, universally recognized that even in primary acute attacks of every variety the blood pressure may rise rapidly. As a fall in blood pressure accompanied by diminution in size of the heart is a favourable sign, it is important to take systematic sphygmomanometer readings throughout. Diagnosis can hardly ever be in doubt, except in the very rare cases of complete anuria with palpable swelling of one or both kidneys, and with delayed appearance of oedema. This was met with in the cases described by Reginald Harrison in which surgical treatment was undertaken under the impression that cases of acute hydronephrosis were being dealt with. One case of this type was that of a lady, aged 35, whom I saw in consultation some years ago. Her history was as follows:

After a day of active exercise she woke in the middle of the night with severe pain in the back and much restlessness. The next morning she was slightly feverish, was unable to pass urine, and presented a distinct tumour in the right renal region. I saw her two days later in conjunction with her medical attendant and a surgeon. In the forty-eight hours since the beginning of her illness not a single drop of urine had been passed, nor could any be obtained by catheter. She was feverish and restless, and on her right side a tumour, diagnosed as an enlarged kidney, was easily detectable, and was extremely tender on pressure. X-ray examination was out of the question; the diagnosis reached was acute calculous hydronephrosis with reflex cessation of function of the left kidney. As no improvement had taken place next day, and as no further symptoms had appeared, operation was agreed to; but on exposing the right kidney, no stone and no hydronephrosis was found. The kidney was much enlarged, and on incising the capsule bulged out as if under extreme tension. On the next day a small quantity of urine was passed; it was found to be loaded with albumin, and contained blood and granular and epithelial casts. After a hesitating course, uraemia developed ten days later, and the patient died. Prior to death she developed generalized oedema and other ordinary nephritic symptoms. It was only possible to remove the right kidney for examination, and this was found to show pronounced glomerulotubular nephritis.

CHEMISTRY.

In a case of average severity the condition of the urine is familiar. It is scanty, probably owing to the diminished blood flow brought about by the general vascular congestion, and contains blood, casts, and abundant albumin. It is generally assumed that the albumin is entirely composed of blood protein filtered through the glomerular membrane, but a recent study by Kellaway, Davies, and Williams suggests that when the cells are severely damaged much of the protein present consists of kidney substance. Further work on the proteins of urine is desirable. The quantity of albumin diminishes as the patient improves. Acute nephritis is, indeed, one of the few conditions in which regular estimation of the amount of albumin passed is of value. Incidentally, however, it may be stated that estimations by the Esbach albuminometer are even less reliable than is generally believed. In a recent study Geoffrey Bewley has reminded us that the readings of that instrument vary widely according to the temperature. Thus, the same specimen of urine on two successive days will vary as much as 25 per cent. according to the temperature of the day. In fact, the ordinary heat and acetic acid test in most cases enables one to judge just as satisfactorily as does Esbach's instrument. The urea present is diminished in the early stages, and chlorides may be reduced almost to vanishing point. Diastase also is lessened, except in the nephritis of eclampsia.

In conformity with the condition of the urine certain blood changes are found. Urea is almost invariably increased, and in cases which do not progress favourably very high levels of blood urea may be reached. As improvement sets in and diuresis occurs the urea rapidly returns to normal. It is admitted that the progress of the case and the immediate prognosis may be better determined by blood urea estimation than by any other laboratory examination.

The chloride in the plasma is probably increased in the early stages, although opinions vary greatly on this matter, and the whole question of chloride excretion and chloride retention is one that must be approached with much humility. Some writers believe that chloride retention in the body is due to a definite renal failure to excrete chlorides. Others regard it as merely secondary to the failure on the part of the kidney to excrete water. Still others believe that chloride retention arises in a totally different way. They hold that in acute nephritis the permeability of the capillaries throughout the body becomes increased as the result of the presence of toxins in the circulatory system, and possibly as a result of diminished surface tension of the serum, and that this increased permeability, combined with the hydraemia resulting from oliguria, leads to a great outpouring of fluid into tissue spaces. This fluid must contain its normal quotient of sodium chloride, and hence there results a locking up of chlorides in the body, due to tissue demand rather than to renal failure. The actual chloride is of importance, and many now feel that the sodium ion is more important than the chlorine ion. In favour of this view the beneficial effect of calcium chloride and ammonium chloride in producing diuresis is often referred to.

Through these tangled views it is hard to find one's way, but the fact remains that there is considerable chloride retention in the body as a whole. Other blood changes include the existence of a slight acidosis due to a diminution of the sodium carbonate blood content. The existence of this was foreshadowed long before the days of biochemistry by clinicians, who found that it was more difficult to make the urine alkaline in acute nephritis than in health, and who believed that once it was rendered alkaline the danger of the onset of uraemia was almost certainly averted.

Fischer's theories may here be referred to briefly. He believes that local alterations in the acidity and alkalinity of tissues produce changes in hydration, inasmuch as a changed reaction alters the capacity of the body colloids to hold water. Many of the actual changes found in the kidney cells are explained by him in this way, but the theory, though attractive at first sight, has been proved untenable. In my hands the treatment of renal cases by Fischer's strongly alkaline solution has not proved satisfactory.

Blood phosphates are also increased, and are stated to be closely associated with the development of toxic symptoms. A return to normal is of favourable significance. On the knowledge that a deleterious phosphate retention exists is based the treatment by calcium salts, and von Noorden's special recommendation that large quantities of calcium should be given to all nephritic patients who are taking much milk. Milk, as is well known, is rich in phosphoric acid.

Maxwell has found that cholesterol also is increased in the blood in acute nephritis, and although other observers have stated the contrary, his work seems sufficiently definite to establish the fact. He asserts that, as the patient improves, oedema first disappears, then excessive cholesterol, and then albumin. From the practical point of view, however, cholesterol estimations are at present unimportant.

If the patient progresses from an acute into a subacute stage, further blood changes in the nature of anaemia and diminished protein content, with possible changes of albumin-globulin ratio, may take place; but these do not require discussion.

Coincident with the changes in the blood and blood pressure, various changes occur in the cerebro-spinal fluid. The actual pressure of the fluid is increased, as evidenced by the fact that when a lumbar puncture is done it squirts out under considerable pressure. Analysis of the fluid will

show increase of urea, increase of protein content, and, according to most writers, a considerable increase of chlorides, especially if uraemia is threatened. It is even said that a high chloride level is diagnostic of the onset of uraemia.

FUNCTIONAL KIDNEY TESTS.

Since the days of Bright progress in our knowledge of the nephritides has been steady, but comparatively uneventful. Gradually improving microscopical technique has added much bulk to the literature, and has succeeded in differentiating fairly satisfactorily, and with almost painful accuracy, the stages and varying types of nephritis. With the coming of biochemistry a new chapter has been opened, and with the ever-increasing progress of this science we are now in a position to make not merely a guess at the anatomical state of the kidneys in any given case, but to estimate with some certainty the degree of impairment of their physiological function. It is, however, important for the clinician to remember that functional tests should not displace either clinical examination or judgement based on careful observation and examination of the patient. In acute nephritis, indeed, most of these tests are of little importance till the acute stage has passed; but in the recovery stage, particularly if recovery is delayed, they may be of much value in prognosis and in estimation of residual kidney damage. The tests must be repeated again and again, and found to give consistent results, before they can be relied on. A single functional test is valueless, and may be misleading. Further, these tests do not always give us information concerning one only of the four parts of the renal mechanism. For example, the urea concentration test may show defective function either because too little water is absorbed from the tubules, or because the glomerular membrane and the tubules do not permit the passage of a normal amount of urea. On the other hand, the dye tests probably afford information concerning the epithelial cells alone, and when benzoates are given with the object of testing how much hippuric acid will be formed, we are determining the power of the interstitial tissue to form that substance, and the power of the cells to excrete it.

COURSE AND PROGNOSIS.

Provided no complication shows itself by far the largest number of cases of acute nephritis pursue a favourable course, though not necessarily to complete recovery. In an average case, after a few days, the original oliguria gives place to diuresis, and, coincidently with this, the oedema begins to disappear. In my own experience in adults those cases which present the greatest amount of blood in the urine at the beginning recover most rapidly. In the most favourable cases within a month no trace of the disease may be left. The actual percentage of cases in which permanent damage—either stationary or progressive, and either mild or severe—is left has been estimated differently by various observers. De Wesselow, for example, estimated in war nephritis that 70 per cent. had completely recovered after the lapse of a year; while Hume and Natrass, in a recent study, refer to the fact that in France 77 per cent. were found normal after the same period. On the other hand, these last-named state, as the result of their own observations on 281 cases seen during the years 1922-25—that is, over a period commencing four years after the termination of the war—that only 45.5 per cent. had no evidence of renal disease, 9.5 per cent. had advanced chronic nephritis, and 2.5 per cent. had died, while 43.5 per cent. showed evidence of some permanent damage to the kidneys, and were believed to be developing advanced chronic nephritis.

In watching cases one of the most important things to consider is whether the subacute or chronic process is or is not going to supervene on the acute. It has probably been the experience of everyone to meet with cases in which albumin has completely disappeared from the urine after persisting for as long as twelve months or more; but this is exceptional, and in general it may be assumed that if albumin is still present after the lapse of six months, complete restoration to health is unlikely. In estimating prognosis we must of course consider whether we are or are not dealing with an exacerbation of a pre-

existing nephritis. It is in these cases that the functional tests are of so much value. Some writers go so far as to say that, provided such tests show a normal response, the presence of albumin is of no consequence whatever. With this view I cannot agree. My experience is that no matter what response patients may show to tests, if albumin is persistent they remain below par, and are particularly liable to intercurrent infections.

TREATMENT.

In the early stages rest is imperative, and should be insisted on as long as haematuria persists. In cases with oedema and fairly abundant albumin I have often found improvement follow getting the patient up. Each case must be judged on its merits, but in general I am opposed to overdoing the period of rest in bed.

As regards diet, most patients at the beginning of their illness suffer from anorexia, and some from nausea and vomiting. This automatically limits the intake of food. As von Noorden long ago pointed out, even water should not be pressed at this stage, and whatever theories we may hold, it certainly seems advisable to limit also the intake of chlorides. Small quantities of milk and water to which a little calcium carbonate has been added satisfy all necessities. As improvement sets in, carbohydrates may be added fairly freely, always having regard to the digestive powers of the patient. Grapes and fruit juices, including fruit jellies, are appreciated, and gradually proteins may be increased. The revolt against the consumption of large quantities of milk in this stage, on the ground of its large protein and water content, took place during my student days, and at once appealed to common sense. Even when almost complete recovery has taken place it is well to maintain some restriction regarding proteins, though one admits with Wordley that no direct increase of albumin results from an increased protein intake. In later subacute stages the problem may alter, and Epstein's diet be required.

As regards other measures, the old-fashioned hot pack and the more newly devised radiant heat bath seem temporarily to be under a cloud. In stating this I cannot help recalling the old-fashioned suggestion to rub the skin with bacon fat in order to prevent sweating, and so to leave more fluid in the body for the purpose of flushing the kidneys. Indiscriminate sweating is, I am sure, weakening, but when headache and other uraemic symptoms threaten, I have not the least doubt that a good radiant heat bath is beneficial. It is seldom that in any stage direct measures for the relief of anasarca will be required, but, if they are, Southey's tubes are undoubtedly preferable to multiple punctures.

No drug treatment, apart from the giving of mild diaphoretics and purgatives, is required at first. All stimulants of the kidney must, of course, be rigidly barred. Later, with hydraemia, and with excess of urea in the blood, these may be tried, in the hope of promoting diuresis. I have tried ammonium chloride, so strongly recommended from the Mayo Clinic, but without good results, and the equally strongly recommended novasurol is unquestionably a substance to be used with caution. In my experience it is a much safer remedy in cases of cardiac than of renal oedema.

Decapsulation.

In 1896 Reginald Harrison put on record three cases of acute nephritis in which cure had followed incision and stripping back of the capsule. Later he reported further cases, and stated that operation was indicated (1) in cases of acute nephritis in which convalescence is delayed, and in which albumin and casts do not disappear from the urine; (2) in cases where suppression of urine occurs; (3) in cases where marked cardiac and circulatory disturbances arise in the course of inflammatory renal disorders.

In 1903 Edebohls suggested a similar operation in more chronic cases of nephritis, with the object of preventing compression of the kidney and of establishing collateral circulation; and more recently Roosing (1922) has recorded 69 cases of nephropathy grouped under four headings, for which decapsulation was performed with apparently good results.

The reasons for such an operation are quite different in an acute and in a subacute stage. In an acute stage, when the capsule is incised the kidney bulges out, and therefore it might be claimed that relief from tension is afforded. In practice, however, the operation seems to have been generally abandoned, and inasmuch as most cases pursue a favourable course on ordinary lines such a serious procedure is never justified. In the subacute cases the capsule has adapted itself to the increased size of the kidney, and operation can only be justified on the plea of establishing collateral circulation, and thereby arresting a progressively downward course. Most physicians who realize that recovery from an operation does not always mean cure of a disease, and who realize the many variations in course taken by subacute nephritis, will hesitate long before recommending surgical intervention. In a recent paper, however, Fawcett urges the value of the operation in a specially selected group of subacute parenchymatous cases, the sequelae, for the most part, of acute inflammation.

When uraemic coma develops, either subsequent to or independent of epileptiform convulsions, and when all ordinary measures such as hot packs, the use of pilocarpine in small doses, and purging fail to produce rapid improvement, venesection should be performed. I well remember the first case upon which I performed venesection, some twenty-five years ago.

A boy, aged 18, was admitted suffering from acute nephritis. After a few days he was well on his way to recovery. At this stage his friends managed to bring in a large supply of food, and the boy enjoyed a surreptitious but hearty meal. That evening he had a severe convulsion and passed into a state of coma. During the next day the coma persisted and there was almost complete anuria. The next morning, thirty-six hours after the beginning of the coma, he was still deeply unconscious, and apparently going downhill. I bled him to the extent of about 15 oz.; a few hours afterwards he opened his eyes, and that evening he again became conscious, and ultimately made a good recovery.

When bleeding a serious case, if the blood flows badly, it is worth remembering Lauder Brunton's tip that inhalation of oxygen will make the blood less treacherous and greatly facilitate procedure. It is curious that some writers, such as Tweedy, regard bleeding as dangerous in eclampsia, but most gynaecologists are in favour of moderate venesection.

During the war I practised lumbar puncture in addition to venesection for the repeated convulsions that were so frequently encountered. The most recent writers seem to regard lumbar puncture as a mistake, but I am convinced that no harm ever resulted in my own cases, and, judged as a clinical procedure, it appeared to have a beneficial result. It seems, indeed, to be a common-sense method of treatment. The cerebro-spinal fluid, as already stated, is under high pressure, and contains an excessive amount of urea and of chlorides. Removal of fluid is often good for the headache of chronic renal disease, and I therefore find it hard to believe that relief of tension will not be beneficial in acute renal disease accompanied by nervous phenomena.

The giving of saline injections intravenously, subcutaneously, or into the colon in uraemia is of doubtful value. For a couple of years I employed Fischer's solution both intravenously and by the rectum, but could never satisfy myself that it was of value, and the rectal injection of the highly alkaline solution which he recommends, containing as it does 10 grams of sodium carbonate per litre, is extremely irritating. The injection of plain sodium chloride and of sodium citrate solution can be justified only as a diluent of retained toxins, and may be useful in this way, even though it is realized that the kidney finds difficulty in excreting both chloride and sodium ion. On the whole, I am inclined to recommend their use in the form of colonic injections after thorough evacuation of the colon.

As regards drugs, morphine, so long under suspicion in uraemic cases, is now recognized as both safe and satisfactory in checking repeated convulsions. It is hard to realize why this remedy was so long under suspicion, having regard to the fact that in the allied condition of eclampsia it has been employed for many years.

ACUTE NECROSIS OF THE PANCREAS.*

BY

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THE term "pancreatitis" as the subject of this discussion was deliberately chosen so that it might cover the wide range of conditions usually included under that heading, many of which are but little understood: in order that those taking part in the discussion should not be tied down to any particular type, but might be able to give their views and experience of the many varieties of the condition, and especially of the less regular types or "formes frustes" of this somewhat rare disease.

In the few remarks that I have to offer, largely based on six cases of which I have had personal experience in the last two years, I propose to limit myself to the condition known as acute necrosis of the pancreas, which is a better term than acute haemorrhagic pancreatitis, as it does not beg the question of its primarily inflammatory origin, a view which is not held by a very considerable number of observers. Nor is the term "haemorrhagic" entirely satisfactory, as in a certain proportion of cases haemorrhage is not always strikingly in evidence.

THE PECULIAR FEATURES OF DISEASE OF THE PANCREAS.

The pancreas has been well described as a laboratory of chemical physiology. In addition to its internal secretion, it forms the most powerful proteolytic ferment, the fat-splitting and the amylolytic, in addition to others of less importance. Another feature of this gland in which it differs from other secreting glands is that it possesses two ducts, sometimes in anastomosis, sometimes not, and of which, to use an Irishism, the lesser—the accessory duct of Santorini—is sometimes the greater. The latter duct has no valve at its entry into the duodenum, a fact the importance of which will be seen later. The main duct of Wirsung in the majority of cases joins the bile duct before it pours its contents into the duodenum, the distance of this junction from the intestinal opening varying from zero to 11 mm., averaging about 4 mm. All these facts have a bearing on the canalicular theory of pancreatic necrosis.

In the view of many writers on the subject the varying forms of acute pancreatitis as described in the textbooks are simply stages of the same process, and are described by Brocq¹ as acute oedematous pancreatitis, acute haemorrhagic pancreatitis, and gangrenous and suppurative pancreatitis, the rarity of the first being due to the fact that the surgeon seldom sees cases at this early stage.

As regards the suppurative varieties, probably some are primarily infective, whether the infection is duct-borne or by way of the lymphatics, but many are simply secondary infections following on the necrosis of the gland.

In studying the various papers and case reports of acute pancreatitis one cannot but be struck by the varying types of the disease which they describe, the great difference in the mortality of different clinics, the diverse views held as to its etiology, and in some degree also as to the appropriate treatment.

This is perhaps not to be greatly wondered at, when, in a condition relatively uncommon, it can only fall to the lot of any individual surgeon to meet with a limited number of cases. Further, in many of the necropsy reports there is no account of a minute examination of the ducts which would throw some light on the etiology. In reading the papers of Deaver and other supporters of the theory of lymphatic extension from the gall-bladder it would appear that, as is well put in a paper by Fiske Jones,² they are describing a condition different altogether from that of acute pancreatic necrosis, and that whilst that theory may well account for chronic pancreatitis and some of the suppurative varieties, it is not applicable, at any rate, to the majority of cases of the former condition.

ETIOLOGY.

The immediate cause of the production of the *drame pancréatique* is the setting free of the secretion of the

* Read in opening a discussion in the Section of Surgery of the Annual Meeting of the British Medical Association, Cardiff, 1928.