does not seem very ill during the attack in spite of a high temperature.

This type is, I think, fairly well recognized, there is the sudden rise of temperature without apparent cause, subsiding in a week or so; then the patient remains well perhaps for some months, and then without warning the attack recurs. The attacks generally tend to become fewer and less severe after puberty is passed, but I have known them continue till 19 years of age at least. I am inclined to think that children of a neurotic type are the ones most often affected.

Some children, it must be admitted, are more prone to fever attacks from slight causes than others. Another class of child is more prone to vomiting attacks than the ordinary child. Other children are more prone to nerve storms, or in other words to epilepsy, the exciting cause of which may defy our best endeavour. In children who suffer from recurring fever attacks it is probable their heat or vasomotor centre is unstable and easily influenced by toxins or other causes. That bacteria may enter the system through the naso-pharynx, and that the adenoid tissue of the naso-pharynx if excessive may offer a suitable ground for their cultivation is highly probable. Any unhealthy condition of things in these regions should be dealt with; though I confess I have seen cases in which the naso-pharynx has been several times harried and scarified in vain.

Then there is the "jam and bread-and-butter" theory—that is, that the attacks are due to starch and sugar toxins absorbed from the alimentary canal, and it has been asserted that abstinence in great part from these luxuries, has prevented or lessened the violence of the attacks.

But I confess I have had scant success by means of such treatment in the cases above described, for food and diet have seemed to have very little influence. Then, again, uric acid enthusiasts have had their say, and a form of diet in which proteids are but scantily represented may certainly be tried. But we have, I am inclined to think, a lot yet to learn before we can boast of much solid knowledge of these puzzling attacks.

Now, some of the feverish states we have to deal with in older children are plainly due to gastric insufficiency associated with or without a chronic intestinal catarrh. Perhaps the child has from time to time vomiting attacks with feverishness, succeeded by loss of appetite and an evening rise of temperature (101° to 102° F.), which perhaps suggests a subacute tuberculosis to the medical attendant. An examination shows a dilated stomach or one which is in a chronic condition of diastole, with decomposing contents and a constant evolution of gas. This is certainly a condition which will in some children, but not in all, cause an evening rise of temperature, and perhaps more or less wasting and anaemia. Very much the same symptoms may be produced by intestinal dyspepsia: the child loses flesh, has a poor appetite, passes pale-coloured stools with an excess of mucus, and there is an evening rise of temperature, which may go on week after week. It is in this class of case that dieting is clearly important, and when cutting down the amount of starch, sugar, and milk is undertaken, an improvement may be quickly effected. In some children the evening rise of temperature is apparently altogether out of proportion to the amount of intestinal catarrh or indigestion which appears to be present.

But yet again I am inclined to think that most of us are consulted with regard to children who for weeks and weeks may have an evening rise of temperature of a few degrees with nothing at all obvious to account for it. Some of these are children of the "neurotic type," clever children who must be doing something, and who seem to get a temperature whenever they are fatigued. Excitement, tiring muscular exertion, overwork of brain and muscle, and the temperature will go up. Naturally in all these cases we fear some tuberculous process may be in progress; one feels for an offending appendix, and, of course, carefully investigates the stools for either round or thread worms; then, having threshed out the question of the evening temperature in all possible lights, the wise practitioner packs the child off into the country for three months, accidentally breaks the much-used and treasured family thermometer, and supplies the mother with one which "all the king's horses and all the king's men" cannot make rise beyond the modest height of 99° F.

No doubt there can be too much temperature taking and weekly weighing of children who have learned by experience what a lot of anxiety their ailments give their friends; far better for them to hear nothing of disease, to lead a healthy outdoor life with varied occupations, whether they are simply neurotic and dyspeptic, or there are a batch of tuberculous glands somewhere out of sight and touch.

But I should weary you if I were to attempt to discuss all the forms of fever to which children were liable, and which for the time being we were unable to classify or explain. I have said nothing of "gland fever," or the fever which may precede for some days the outbreak of the rash of erythema nodosum. Then, again, I am mindful of the erratic fever, sometimes high, which precedes for some days the classical signs of an anterior poliomyelitis. Two children in the same house are attacked with an unexplained fever; one recovers without a bad symptom, the other, to our intense surprise, is found to have one or more limbs paralysed.

I will make no excuse for having approached the subject from the point of view of the general practitioner, mindful of the difficulties he has often thrown in his way of making a diagnosis, and the lack of opportunity he often finds in obtaining the help that the pathologist alone can give. Happy is the man who finds his pleasure in his daily round of work, and to whom the unsolved problems of the world provide alike an inspiration and a stimulus.

ON THE URINARY MUCOUS TRACT, AND NOT THE BLOOD STREAM, AS THE ROUTE OF INVASION BY PATHOGENIC ORGANISMS UNDER CERTAIN CONDITIONS.

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EVERY surgeon must be familiar with a certain class of cases which clinically reproduce the features usually associated with pyelitis—namely, an acute infection of the pelvis of one or both kidneys, and in which there is a curious absence on superficial observation of any adequate cause for the illness so produced: No passing of a renal calculus, no previous introduction of instruments or injury to the urethra or bladder, no antecedent attack of gonorrhoea or stricture.

Hitherto it has been the custom to explain such cases as originating in a blood infection—that is to say, organisms, it may be the *Bacillus coli* derived from the intestinal canal, find their way under certain circumstances into the blood stream, are excreted by the kidneys, and in their passage through these organs give rise to the symptoms of inflammation in the pelvis of the kidney.

Although Mr. Barnard¹ in England, Leuhartz,² Lennander, and others on the Continent have realized the probably true explanation, yet it is still customary in this country to regard the blood stream as the primary source of infection.

There are certain facts in connexion with these cases which are very suggestive as throwing light on the real method of infection.

First, the majority occur in the female sex; all surgeons are agreed on this point.

Secondly, the trouble is especially apt to arise during the pregnant state. Now, in connexion with this last point, it is of interest to note that in an investigation into the condition of the female urethra in gynaecological cases, Dr. J. Campbell of Belfast found evidence of bacterial infection, purulent discharge, etc., in 30 per cent. of gynaecological and pregnant cases. The kind of organism varied. The *Staphylococcus albus* was present in 94 per cent. of the cases, and the gonococcus or streptococcus in the remainder.

Further, Rebland and Cumston³ have drawn attention to pyelonephritis as a complication of pregnancy, and they associate with it the dilatation of the ureters, especially the right, which occurs in the pregnant state. The uterus, when rising out of the pelvis in the fifth or sixth month, compresses the ureter against the pelvic brim. Lohlein and Olshausen have pointed out the fact that the ureters are found dilated in women dying in labour. Cumston's interpretation of the sequence of events is that the ureteral obstruction leads to a partial

hydronephrosis, and that during this state of lessened resistance on the part of the kidney so distended, virulent organisms of the colon bacillus type find their way into the blood stream and so infect the kidneys, the colon bacillus being found in the urine in most of these cases.

In the Address in Surgery to the British Medical Association, at the annual meeting at Leicester in 1905, I brought forward evidence to show that under certain circumstances a regurgitant mucous stream does occur in the genito-urinary tract capable of conveying inert foreign particles, such as indigo, along the whole length of the tract from the urethra to the upper end of the ureter; indigo granules inserted into the anterior urethra in the male subject having been recovered from a suprapubic opening in the bladder, and in another case powdered indigo inserted into the bladder, was recovered from the upper end of the fistulous ureter, opening on the surface of the loin.

It was also pointed out that the one necessary predisposing factor is that there shall be some temporary arrest, partial or complete, in the normal outgoing flow of the natural secretion, the urine in the case of the kidney. If this arrest occurs continuously or intermittently, then the conditions are present which favour an ascending mucous stream or current in the urinary tract, and with this current the probable conveyance of micro-organisms, should these gain an entrance into the urethra or bladder.

Before putting together the evidence derived from clinical observation on the one side and from experimental investigation on the other, I wish to draw attention to a few salient features in three cases of pyelitis under my own observation.

The first is that of a previously healthy man of 40 years of age, who was seized with acute symptoms of what clinically resembled an attack of cystitis, with frequent micturition and much pain and soreness during the act, referred to the neck of the bladder. In a few days the patient became critically ill, with repeated rigors and a temperature of 104° , and much pain in the right loin. The urine at this time was acid in reaction, and contained some blood cells, pus cells, and a few granular casts. Bacteriologically the urine gave a culture of *B. coli*. The symptoms, local and general, gradually subsided during the next six days, and the patient remained fairly comfortable for three weeks, with a normal temperature and a clear urine. Then another acute febrile attack occurred, with considerable loin pain and increased urinary tenesmus, with more pus in the urine. After a gradual fall in temperature, lasting another twelve days, the urine gradually cleared up, and the patient made a good recovery.

The second case is that of a young man of 28, previously healthy, who, without any previous urinary instrumentation, gonorrhoea, or apparent cause, developed an ordinary rather transient attack of cystitis, with urinary tenesmus and ropy urine. This was succeeded in about a week by infection of the pelvis of the left kidney, which became swollen and tender, the extension upwards of the infection being ushered in by a rigor and a temperature of 103° . The symptoms subsided, but relapses occurred on three occasions, accompanied with a rise of temperature to 103° and exacerbation of the kidney pain. The urine at this time was acid in reaction, and contained pus and blood cells. Then after apparent recovery and an interval of a few weeks the rise of temperature and rigors returned, accompanied this time with pain and tenderness in the right kidney; the increased frequency of micturition also returned with pus in acid urine. The patient gradually recovered, the urine becoming normal.

The third case is that of a young woman, multipara, in the fifth month of pregnancy, who while in otherwise good health was seized with all the symptoms of acute cystitis, with a moderate degree of fever and frequent and painful micturition, and alkaline purulent urine containing also viscid mucus. Seven days later the temperature rose to 103° , the urine became acid, then nearly solid with albumen, and gave a copious deposit of pus without much mucus; the patient complained of great pain and tenderness in the left kidney region. The temperature fell gradually for two days and the pus diminished, when a further rise of temperature occurred to 102° , with an increased quantity of pus in the urine and pain in the region of the right kidney. Gradually the temperature subsided and the urine became normal, and the pregnancy went to full term, ending in the delivery of a healthy male child.

In these three cases there were distinct symptoms at the beginning of the attack, of a definite cystitis, transitory in character, but definitely preceding the kidney symptoms. This does not always occur; thus in Leuhartz's account of 80 cases of ascending infection of the kidney pelvis in adults, the bladder escaped infection in many of the cases. The patients were chiefly females, in whom the drainage and flushing of the bladder is probably

more efficient than in the case of men. The right kidney was affected more often than the left. The infecting organism was definitely shown to be the colon bacillus in 66 cases, while the proteus bacillus and Friedländer's pneumococcus were found in some of the remaining cases. Moreover, in Leuhartz's cases, as in those I have previously recorded, there is no history of any preceding urethritis or traumatism from the passage of instruments.

Putting the clinical, bacteriological, and experimental facts together, it is impossible to resist the conclusion that the infection, whatever the organism may be, enters the body by way of the mucous canal of the urethra, spreads thence to the bladder, and is carried upwards along the ureters to the pelvis of the kidneys.

There are many objections to the theory which regards the blood stream as the primary source of infection, and the kidneys as secondarily involved during the process of excretion of the organisms.

First, in some, at any rate, of the cases in which the colon bacillus is the active organism there is a preliminary cystitis. Moreover, there are no general symptoms pointing to invasion of the circulation by pyogenic organisms preceding the occurrence of definite local urinary symptoms, while the absence of any foci of infection in other organs is remarkable; for if the infection is general and the organisms are brought by the blood stream to the kidneys, it is probable that some would be carried to other organs also.

On the other hand, if we regard the infective organism (the colon bacillus in most cases) as escaping from the intestinal canal and gaining access to the urinary tract by way of the urethra from the surface of the body, it may be urged that the bladder should be first affected in all cases, and that the urethra also should show some signs of inflammatory reaction.

But there are reasons for believing that virulent micro-organisms may be carried along a healthy mucous canal without overcoming the resistance offered by the epithelial cells (if healthy) lining the tube, and that such organisms may be carried upwards (isolated in, and with their growth, possibly temporarily, inhibited by the normal mucus which lines the walls of the canal) to a portion of the urinary tract in which, owing to some stagnation or reversal of normal currents, or to some loss of resistance on the part of the epithelium, or possibly to some abnormality in the character of the protecting mucus, they can commence rapid and virulent growth.

Thus, in the microscopical examination of sections of the Fallopian tube in the case of a female child who died from pneumococcal peritonitis, and in whom the symptoms pointed to the genital canal and the vagina as the route of invasion, the pneumococcus was found on and amongst the epithelial cells lining the villi of the tube, but no desquamation of the epithelium or infiltration by leucocytes, or signs of inflammatory reaction, were visible.

Moreover, there are many examples of infection in which different organisms travel along mucous canals, such as the bronchi or the intestines, and only produce their pathological effect at the site of peculiar susceptibility or seat of election, such as the lower end of the ileum in the case of the typhoid bacillus, and when this organism (as frequently occurs) is excreted by the kidneys, little or slight inflammatory reaction is present locally in the kidney.

The non-motility of the infecting organism in some of these cases of ascending pyelitis may be at first sight regarded as a difficulty on the supposition that the infection rapidly ascends the whole urinary tract, and first causes symptoms at the upper end or kidney pelvis, and in the absence of any evidence of extension of infection by contiguous growth along the ureter. But since the demonstration by the experiments recorded by myself in the Address in Surgery of the comparatively rapid manner in which inert, non-motile particles of a foreign substance are carried up the urethra and ureters, this difficulty disappears.

Moreover, this question of the mucous canal route of invasion as against the blood-stream route, in the case of organs communicating by their ducts with the exterior of the body, is one of general and not merely local interest. The gall bladder and the gall ducts come into the same category, also the pancreatic ducts and salivary ducts, and the true explanation of the manner of infection of the kidney pelvis is probably the true explanation

of the route of infection in the case of the gall ducts also.

It has hitherto been generally supposed that pathogenic organisms find their way into the blood stream from the intestinal canal and so reach the liver, and in the course of their passage with the bile along the liver ducts, after excretion, cause the symptoms referable to inflammation of those canals.

The observed facts, however, do not favour such an explanation. For instance, in all cases in which we know for certain that the organisms are conveyed to the organ in question, the liver or kidney, by means of the blood stream—in cases, in fact, in which the organ is affected as part of a generalized systemic affection—it is the cortical or secreting portion of the organ which first becomes affected and suffers most. Large or small, isolated or multiple, embolic suppurating foci form in the cortex while the excretory canals escape, or only suffer later. This is well shown in the case of the multiple abscesses of the liver occurring in the course of portal pyæmia. On the other hand, the comparative rarity of liver complications and abscesses in cholangitis and cholelithiasis is very remarkable.

I think also other experiments described in the address to which I have already alluded confirm, in the case of the gall bladder and biliary ducts, the conclusions which we have already come to in the case of the kidneys and urinary tract. Indigo taken by the stomach was recovered through a gall bladder fistula, and had evidently been carried up from the duodenum along the common bile duct, and so into the cystic duct and gall bladder.

Where inert powdered indigo is carried, pathogenic organisms can be carried also, provided that the arrest or reversal (temporary or otherwise) of the mucous current in the bile ducts is brought about from pathological causes, such as obstruction at the outlet, in the same way that it is brought about surgically in the case of the gall-bladder fistula.

Moreover, I think we may extend this theory of duct infection to other affections besides those caused by the pus-forming organisms. Take, for instance, the tubercle bacillus and the manner in which it attacks the testicle. Tuberculosis of the testicle commences in the epididymis at the upper or blind end as it were, of the genital duct, the vas deferens, and not in the body of the testis itself, where we should expect it to begin if the organism were brought by the blood stream.

Surely, too, in cases of tuberculous fistula *in ano*, the organism reaches the atraded mucous membrane from the intestinal canal and not from the blood stream, and I am inclined to think that in cases of tuberculous disease of the kidney, the bladder or the testis, the organism will eventually be shown in some cases to have ascended the mucous canal from external contamination instead of having descended it after excretion by, and passage through, the kidney. In fact, I would be inclined to put forward the following suggestion:

Infective processes originating in the excretory ducts of organs or glands or in mucous canals probably depend on these mucous canals as the route of invasion, while infective processes originating in the parenchyma or cortical portion of the same glands or organs depend on the blood stream for the entrance of pathogenic organisms.

This view is also in harmony with the observed phenomena of toxic processes. Cytotoxins circulating in the blood exert their cytolytic action primarily and chiefly on the epithelial cells of the cortex of an organ—the tubules in the case of the kidney, the secretory epithelium in the case of the liver—while the epithelium of the excretory ducts at first escapes injury, thus showing that the resisting power of the latter is at least as great as, and probably greater than, that of the secretory epithelium. Whereas in all the cases we have been considering of pyelitis, cholangitis, etc., due to mucous invasion, this duct epithelium has been the first to suffer.

In thus attempting to focus attention on the mucous canals as routes of invasion by micro-organisms I am hopeful that the importance of safeguarding the orifices of these canals opening on the surface of the body will become more manifest. Frequent bathing, rigid cleanliness, and personal hygiene will do much if regularly practised among hospital patients, indeed, among all classes of patients, to prevent infection of these canals.

It may be urged that tuberculosis of the kidney or of

the epididymis occurs only in persons with a lowered resistance to the tubercle bacillus, and, in most cases, in those who are already the subjects of tuberculous disease in other organs which may serve as foci of infection. This, of course, is only saying in other words, in the language of the mucous canal invasion hypothesis, that a manufactory exists in these patients for the multiplication and liberation of the tubercle bacillus, and the pouring out of numbers of these organisms on the surface of the body by way of the mouth or the anus. Organisms so liberated may easily reach the urethral orifice, male or female, by direct contact, or by contaminated fingers, while the diminished resistance of epithelium is necessary in either case, in order that the organisms so conveyed may grow and flourish.

To return to the analogous case of the gall bladder: organisms, probably of special virulence, must first be grown in the bowel during some attack of local intestinal catarrh or indigestion. These are carried up the common bile duct, and only produce symptoms or set up a cholangitis in those cases in which the resistance of the lining epithelium has been diminished by distension, or stagnation of secretion, or by the contact of a secretion (of the bile in this case) of a chemically irritating or toxic character.

Finally, this view of the mucous canal as opposed to the blood stream route of invasion by micro-organisms suggests possibilities of treatment which are absent in the latter supposition.

The preventive methods of cleanliness and personal hygiene in the case of the external orifices have been pointed out. The question of the arrest of the growth of the organism while still in the lower portions of the urinary tract, and in the earliest beginnings of the attack, by altering the constitution of the excretory fluid—the urine in the case of the bladder—by the administration of urotropine or other urinary sterilizing drugs, is worthy of serious consideration. Moreover, the power to control the growth of the intestinal flora would doubtless very materially aid in diminishing biliary complications also.

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AN UNUSUAL MOTOR-CAR ACCIDENT.

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ON October 26th, 1906, about 11 p.m., there was admitted to my ward in the Glasgow Western Infirmary, W. O., aged 36, suffering from a perforating wound of his left buttock caused by the shaft of a spring-van. On admission he was quite conscious, and was not suffering from any symptoms of shock. He gave the following account of the accident. While driving in a motor-car, and sitting in front on the left of the chauffeur, the car came into collision with a spring-van that had no lights, and was on the wrong side of the road. One of the shafts of the van penetrated his left thigh and buttock, and then passed through the back of the seat on which he was sitting, so that he was pinned down to the seat and could not move. To allow of his removal from the motor-car the back of the seat on which he was sitting had to be cut through, and the van shaft that penetrated his thigh and buttock had also to be sawn across.

The photograph, taken by magnesium light, shows the state of matters on patient's admission. The van-shaft with some of its harness is in the position it was in when the patient was brought in. It will be seen that it has passed through the left buttock, and that its free end, with the patient in the recumbent posture, lies along his left side as high as his left armpit, while the other sawn end reaches as far down as the foot. About 9 in. of the centre of the shaft are embedded in the soft tissues of the buttock, and these are somewhat tightly stretched over it. There was no hæmorrhage and the patient made no complaint of pain, except when the leg was moved. Around the wound of entrance of the shaft there was considerable bruising of the tissues.

The removal of the shaft was accomplished by cutting through the raised bridge of skin, fat and torn muscle.