and Canadian medicine and surgery into closer touch with one another, with all the advantages that accrue from the personal factors of a more intimate knowledge of and acquaintance with the members of our profession in Canada.

And as a welcome repercussion of this event, comes the announcement that the Canadian Medical Association will hold its meeting in 1933 in London.

The British Medical Association has met outside the United Kingdom on only two previous occasions, and these both were also in Canada. The first of these overseas meetings was held in Montreal in 1897 under the Presidency of Dr. Roddick, and the second at Toronto in 1906, with Dr. R. A. Reeve as President.

The meeting this year, much further west, at Winnipeg, promises to be one of greater interest still, especially from a geographical standpoint. For it will give the members of the Association living in the British Isles unrivalled opportunities of seeing Canada from East to West, from ocean to ocean.

Thus will the educative value of the Annual Meeting of the British Medical Association be enhanced by opportunities of getting to know better both the people and the geography of the great Dominion of Canada.

REMARKS UPON THE PATHOLOGY OF ACUTE ABDOMINAL DISEASE.

By ZACHARY COPE,

M.S.

Surgeon, St. Mary's Hospital and Bolingbroke Hospital.

APART from spontaneous hæmorrhage, torsions of viscera, and injuries, most acute abdominal conditions can be most simply grouped into :—

- (1) Acute inflammations, primarily of a viscus, secondly of the peritoneum.
- (2) Obstruction of one of the various hollow tubes or ducts within the abdomen, especially the intestine.

As you are aware, these groups are not clearly divided, for the later stages of an inflammatory condition is often an obstructive ileus, whilst many an obstruction of the intestine (or even of the biliary ducts) may proceed on to local or generalized peritoneal inflammation. Now there are two outstanding problems in acute abdominal disease. The one is concerned with the beginning and the other with the ending of a great number of the cases.

The first problem is that of the factors involved in the causation of the acute visceral inflammations. The second concerns the nature of the toxæmia which ensues when the intestine has been obstructed for some time. Solve the first, and we could prevent two-thirds of acute abdominal disease. Elucidate the second, and few victims of the acute abdomen should ever die.

Of the acute visceral inflammations, salpingitis requires least discussion, for the mode of infection from the uterine cavity is usually clear. In the case of appendicitis, cholecystitis, duodenitis, and pancreatitis there are considerable differences of opinion as to how the infection gains access, and what factors conduce to its successful lodgment. I am compelled to consider appendicitis first, both on account of its frequency, and because of its possible causative relationship to some of the other conditions. Appendicitis is no new disease, though it took its name and came into prominence fifty years ago after Fitz's paper. Cases of perforation of the appendix were recorded from time to time when morbid anatomy began to be studied at the time of Hunter, and during the last century under the term "typhlitis" the same condition was no doubt treated before the newer term came into fashion. With the advent of safer operative surgery, and the recognition of the minor forms of the disease, appendicitis has become the commonest abdominal derangement. Moreover, the generally received opinion is that the incidence of the

disease is greater, both absolutely and relatively, than it was forty years ago. Factors which are generally regarded as predisposing to appendicitis are kinks or strictures of the appendix itself, concretions in its lumen, and stasis of the bowel contents. That obstruction to the lumen of the appendix increases the severity and seriousness of the appendicitis none will deny, though that does not tell us why the attack started. As to bowel stasis, it would be curious if that were a very important factor, when we consider that the disease is most frequent amongst young men between 15 and 25. Though injury has been said to be an occasional cause, and I have myself seen a case or two that could easily have been explained in that way, yet I think the connection is usually accidental, and anyway is very rare.

The usual explanation therefore is that pathogenic organisms, which are always present even in a healthy subject in the cæcum and appendix, take on an increased activity when there is any local stasis; inflammation of the mucosa ensues, and organisms pierce the membrane and lead either to local thrombosis and gangrene, or a severe inflammatory reaction in the wall of the appendix. Some assert that organisms may, in a debilitated subject, even penetrate the intact mucosa. Though it is commonly received opinion that infection of the appendix takes place from the lumen, yet there are certain indications in favour of the view that some appendices are infected via the blood-stream. I have frequently seen a red, swollen appendix in which the parenchyma of the wall was very much swollen and inflamed, but the lumen of which presented a mucosa almost healthy in appearance, and it is no uncommon appearance to find most of the appendix healthy, but a limited patch of perforated gangrene in one spot, generally near the base, in an appendix which has no concretion or obstruction. These appearances are quite in keeping with blood-infection. Poynton, many years ago, produced typical appendi-

citis in the rabbit by the injection into the blood-stream of streptococci, and similar results have been obtained by other observers, notably Rosenow. Added to this, one knows that it is not uncommon in children for a sore throat to precede the attack of appendicitis. Here then, I think, we have some facts suggestive of blood-borne infection to the appendix.

But whether the infection is blood-borne, or of local origin, we are still in doubt as to why that infection becomes established. Is it due to a lowered general resistance against infection? Is it due to a lack of vitamins? Or can it be due to an alteration in the contents of the intestine which furnish pabulum for the organisms? You are all aware that popular opinion tends to find in diet the cause of many diseases, including appendicitis, and to a certain extent medical opinion supports this. Rendle Short, in an interesting survey of the question as to what general changes of diet have occurred during the time that there has been a great increase in the incidence of the appendicitis, came to the conclusion that the main responsible change was a great diminution in consumption of the cellulose-containing foods. The suggestion is made that the increased mass of indigestible material encouraged the more regular emptying of the bowel. Rendle Short did not think that increased meat-eating had any particular effect in provoking appendicitis.

One of the most gigantic experiments in dieting ever carried out was that which followed the revolution in Russia in 1917. Owing to the total disorganization of the means of production and distribution, and the impossibility of importing food, the Russian nation was literally reduced to starvation, and the rationing which was necessitated was on a scale which scarcely supported life. Half to one pound of poor bread, a little coarse and, to the normal person, disgusting soup, and sometimes a little potato, was a daily allowance which allowed

no margin. This diet had a most remarkable effect on the variety and course of diseases in Russia. The very interesting report by Dr. Horsely Gantt showed that nearly all infective diseases were greatly increased, and the general physical condition was reduced to a dangerously low ebb. We are particularly concerned with the surgical side, and I will quote on this subject.

"The hard conditions of living affected mostly the city dwellers, and the intellectual classes, and the great increase in surgical patients came from these groups. The lack of leisure and rest, the disorganization of nutrition, the physical and nervous exhaustion, were expressed surgically by a complete loss of resistance to infection. In these severe circumstances surgical diseases assumed quite a new character. Ordinary trivial complaints became chronic and serious, and diseases formerly rare appeared suddenly as epidemics. For example: There was an epidemic of purulent infection, of hernias, abdominal ptoses, varicose, peptic ulcers, flat foot, and neuroses. On the other hand, the hospital register showed a decrease of cancer, erysipelas and acute peritonitis, and a *marked* decrease of all kinds of appendicitis. The decrease in the first three diseases mentioned was probably only apparent, as during the bad conditions these patients died off too rapidly to reach hospitals. The opinion is, however, that appendicitis underwent an absolute great decrease. The lack of abundant food may have played a part in this decrease."

The figures that Dr. Gantt quotes show that from 1914 to 1917 the incidence of appendicitis cases was nearly constant at 2.3 per cent. In 1918 it dropped to 0.8 per cent., and in 1919 to 0.01 per cent. These facts of the great Russian diet experiment lead us to several very interesting considerations. It is generally thought that mental worry and low physical condition predispose to every kind of bacterial infection, including that of appendicitis. But here we have a

great differentiation. A population which showed a very greatly increased susceptibility to most kinds of infection exhibited a greatly diminished liability to appendicitis, though, at the same time, it is expressly stated that enteritis and every variety of deficiency disease was rife. I am quite aware that it is unwise to make too broad generalizations, but I think one can safely conclude from these data that appendicitis is most unlikely to be a deficiency disease. When one considers that appendicitis is very common amongst the civilized nations, with a full and varied diet, and is rare amongst the Hindus and the Chinese, who have a diet less varied, smaller in quantity and mainly vegetable in nature, that the disease attacks quite commonly, if not usually, the young and vigorous, the well-fed and otherwise healthy persons, and when one finds that a starvation diet which encourages other infections of almost every kind leads to almost total extinction of inflammation of the appendix, one is forced to conclude that appendicitis is not a deficiency disease, but a disease of excess. I may be told that gluttony was common in the Middle Ages, when appendicitis was seldom seen, but I am sure that appendicitis must often have been concealed under the terms inflammation of the stomach, inflammation of the bowel, iliac passion, palsy of the bowels, and the like, whilst the common run of the population probably had a much more limited, and certainly a much less varied dietary than the same class to-day. It seems probable, then, that excessive diet may leave a residue favourable to the growth of organisms, and I think anyone who has studied the experiments of Wilkie will be inclined to believe that excess of meat diet is more likely to be deleterious.

The prevalence of enteritis, with the diminution of appendicitis, would not appear to support the view that injury of the lining epithelium of the intestine is an important factor in invasion of the intestinal wall by organisms, though this argument

may be vitiated by the fact that the intestinal content were hurried on by the enteritis, so that there was no stasis.

The incidence rates of cholecystitis and appendicitis run more or less parallel, and this has led many to think that inflammation of the gall-bladder may be secondary to appendicitis. I remember the astonishment I experienced when first I read the statement that appendicitis was one of the commonest causes of cholecystitis. The two diseases may certainly occur simultaneously, or one after the other in the same person, but this can be explained without regarding one as the cause of the other. Those who believe in the causative relationship suggest that anastomotic lymph paths may convey infection from the appendix to the gall-bladder when the more direct paths to the lumbar glands are obliterated by inflammation. Evarts Graham has put forward the view that infection may spread from the appendix to the liver via the portal system, and then through to the gall-bladder via the abundant anastomoses between the liver and the gall-bladder. Recent investigations, however, favour the view that cholecystitis is often caused by blood-borne infection. It has been shown that the contents of the gall-bladder may be sterile, though the wall of the viscus may show the presence of streptococci. The fact that in the Russian statistics the cases of cholecystitis did not diminish to anything like the same extent as those of appendicitis does not favour the view that one is dependent on the other.

It is quite likely that inflammation predisposes to the formation of gall-stones, though the imaginative idea that every gall-stone is a tomb-stone reared to the memory of a dead microbe which it encloses is only partly true. The presence of gall-stones no doubt favours the development of any microbes which may be present on the gall-bladder.

Though blood-infection may account for many of the cases of infection of the gall-bladder, no doubt extension frequently occurs

from an infected common duct. Whether that infection has its origin more frequently from the intestine direct or via organisms excreted through the liver, is difficult to say.

The pathology of gastric and duodenal ulcers is a subject of controversy, but most people agree that bacterial (usually streptococcal) infection plays a part, and many experiments support the view that the organism is specific for this part of the intestinal tract. Intravenous injections of the specific streptococcus into rabbits caused ulcers of the stomach and duodenum to form. Recent researches at the Mayo Clinic showed that focal streptococcal infection of the teeth, tonsil or prostate was present in nearly all cases of gastric or duodenal ulcer.

The enormous increase in the incidence of ulcer in Russia in 1918-1919 shows that bad general conditions and depreciation of health greatly predispose to ulcer—a fact which severs its pathology sharply from that of appendicitis. There are many, however, who believe that appendicitis predisposes to duodenal or gastric ulcer, though I have never been entirely convinced by their arguments.

The only practical outcome from this discussion is the recommendation to eradicate any septic focus which might form a source of origin from which streptococci might wander to the various spots of lessened resistance in the abdomen.

We have been dealing so far with the cause of lesions which, if neglected, frequently cause peritonitis. We now intend to discuss the pathology of the late stages of peritonitis and intestinal obstruction. You are all aware of the sequence of events in a fatal case of peritonitis—the increasing distension, which at first can be partially alleviated but, as time goes on, defies control, the paralysis of the intestines, the vomiting, the anxious face, but frequently to the end the acute consciousness; and finally, the typical facies described by Hippocrates, which is not so much the picture of peritonitis as that of impending death

from toxæmic shock and failure of the circulation. The same picture is presented by the late stage of intestinal obstruction. What is the cause of all this? The first fact of importance is that the final stages of obstruction, even without peritonitis, are similar to those of late peritonitis. From this we can conclude that it is most likely not so much the presence of organisms in the peritoneal cavity as the toxins within the gut which are of prime importance. I think one must allow exceptions to this statement in some cases which appear to die quickly with symptoms more suggestive of septicæmia than anything else, but it is a well-known fact that the peritoneum may be full of virulent pus without there being of necessity any such urgent symptoms as I have described. So soon, however, as the bowels begin to become atonic, paralysed and distended, serious symptoms arise. Another qualification needs to be made. The part of the intestine which matters is the small gut, for one may get a greatly distended colon tightly filling the abdomen, and yet serious symptoms similar to those I have mentioned do not arise till the small gut is involved. This fact seems to me summarily to dispose of the argument which has been put forward that the rise of intra-abdominal pressure has its chief rôle in causing the serious symptoms.

That the cause of death lies within the gut is in keeping with the fact that as soon as the bowels begin to work and empty themselves thoroughly, the condition of the patient, as a rule, at once improves, even though the peritoneal infection still persists. There has been much experimental work done as to the nature of the poisons formed within the intestines, and one must refer readers to the work of Whipple, Williams, and Brockman for further information.

The function of a clinician is to study the experimental evidence on any question, and then to try out those theories which seem to be most supported by the experiments.

Though sometimes empiricism hits upon

a correct solution yet, as a rule, sound pathology is necessary for sound treatment. It is interesting to trace the progress of treatment in the case of the toxæmia associated with paralysed and distended gut. It was early realized that emptying the bowel was beneficial. A stiff dose of castor oil or mag. sulph. sometimes saved the situation, or various enemata containing turpentine, asafoetida, or ox-gall were brought in to help. The discovery of the effect of pituitrin and eserin on the musculature of the bowel made this a method to be tried. Washing out the stomach has always been a way of getting rid of some of the poisonous material, but till recently the last resort, based on the pathological knowledge up to that time, consisted in opening the bowel and letting out the contents of the intestine. Sometimes cæcostomy sufficed, sometimes ileostomy, but most observers and operators were of opinion that jejunostomy was best, for in the jejunum the poisonous material appeared to be in the greatest abundance. Mr. Sampson Handley and others used sometimes to get rid of the contents of the small intestine by anastomosing it to the cæcum or colon. I think I have saved lives by all these methods, and if necessary should be prepared to adopt any of them. But for the last two or three years, since the new theory of the pathology of the condition has been put forth by Williams, I have based my treatment on his experiments and deductions, and I have found the best results from the administration of anti-gas-gangrene serum.

The *Post-Graduate Medical Journal* is published monthly. Annual subscription, 6s. post free. Specimen copy sent on application to the Secretary, Fellowship of Medicine, 1, Wimpole Street, London, W.1, to whom all subscriptions should be paid.