to the finer ducts. In the case under consideration, I think the sequence of events must have been cholecystitis, simple non-suppurative cholangitis, the formation of sand, then suppurative cholangitis; my chief reason for this being that in the early stages of the disease there was an entire absence of leucocytosis, as evidenced by Dr. Strong's examination and my own. Cholecystitis can occur without known pre-existing disease. This was proved by Maurice Richardson, who, in a paper published in the *American Journal of the Medical Sciences*, stated that in 10 out of 59 cases of operation on the gall bladder acute cholecystitis was present with no history of previous disease.

In this case we have a past history of colitic trouble which I have already noted. The occurrence of spread of infection to and suppuration in the bile ducts is marked by increased enlargement, tenderness of the liver, increased pain, often radiating through to the angle of the scapula, leucocytosis, and progressive emaciation and loss of strength. Such cases generally run a short course, and recovery is said never to take place. That spread of infection from the gall bladder to the bile ducts does not occur oftener is due to the fact that in acute infectious cholecystitis the cystic duct is usually occluded.

With reference to treatment in such a case, the only hope lies in surgical interference—namely, cholecystotomy and drainage. In this case such a procedure proved a physical impossibility owing to the great enlargement of the liver and inaccessibility of the gall bladder. Had such a procedure been possible, it is almost beyond doubt that in this case little relief would have been given owing to the diffuseness of the suppuration in the bile ducts.

With regard to prophylactic treatment, the diet should be carefully regulated, avoiding as much as possible starchy and saccharine foods. The sulphate, or phosphate of soda or Rochelle salts, may be taken in doses of 1 to 2 drachms daily; regular exercise should be indulged in.

If the diagnosis is confirmed on examination of the stools and the patient's condition is not improving, early surgical interference is necessary, and drainage of the gall bladder before adhesions have formed and before the infective process has spread from the gall bladder up the biliary ducts.

Only a limited examination was possible *post mortem*.

The following notes on the condition of the liver are by Dr. Hunter (Government bacteriologist, Hong Kong):

Pathological Appearances.—The liver was generally enlarged, increased in weight and in specific gravity, and its consistence was tough and leathery. It was jaundiced. There was no marked congestion. The general contour of the organ was maintained.

On section of the organ, the following appearance was presented: Studded throughout the entire right hepatic lobe were rounded areas of a yellow colour. These varied in size from a millet seed to a pea. Many had a deeply pigmented centre. Surrounding these areas there was a narrow zone of tissue of a deep brownish colour.

The remaining out surface of the organ was that of a jaundiced liver with commencing interlobular cirrhosis.

An examination of the yellow areas more in detail showed that they were not equally distributed throughout the right lobe. They were most abundant in the neighbourhood of the main portal, hepatic, and biliary channels.

In the intervening hepatic parenchyma they were few and scattered.

These areas were further examined as to their relation to the parenchyma of the liver, to their consistency, and their microscopic and bacterial composition.

They bore no direct relation to the actual hepatic parenchyma. These yellow areas could be picked out *en masse*, leaving a hollow or space with roughened but hard and fibrous walls. The consistence of these masses varied. Some were pultaceous; others were hard and gritty. To touch, the harder masses were like small peas, and could be thrown about without much damage to their form.

Microscopically, the softer masses were composed of fibrin, hepatic duct epithelium, pus, corpuscles, and a variable amount of bilirubin pigment.

The harder masses were made up of a hyalin-looking material, in which traces of fibrin, pus, corpuscles, etc., could be seen. In the meshes of this material appreciable quantities of bilirubin calcium were found.

Bacteriologically, nothing definite could be obtained. owing to the fact that an examination could not be made until about twenty-four hours after death. *B. coli communis* was found in large numbers. With this organism, however, numerous other extraneous bacteria were present.

The zones, of a deep brownish colour, immediately surrounding the yellow areas, presented the following micro-

scopic appearance. The areas of tissue nearest to the yellow masses were composed of young proliferating connective tissue and hepatic duct epithelium. The changes found were typical as regards a chronic inflammation of the walls of the hepatic duct and its ramifications. Immediately outside this zone of newly-formed tissue, a narrow band of degenerated hepatic parenchyma was found. Large quantities of bile pigment were deposited in this narrow band of newly-formed tissue and degenerated liver cells.

Apart from these changes, the microscopic examination of the liver substance showed marked interlobular increase of connective tissue and the presence of pronounced paracholia.

PORTAL PYAEMIA AND PYLEPHLEBITIS.

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I.—GASTRO-INTESTINAL ULCERATION AS A FACTOR IN SUPPURATIVE PYLEPHLEBITIS.

GASTRO-INTESTINAL ulceration is usually held to be the most potent cause of suppurative pylephlebitis, especially when that ulceration is in the neighbourhood of the appendix. Yet diseases in which gastro-intestinal ulceration is most extensive are not those which lead to suppurative pylephlebitis. If such ulceration were really the direct cause, surely we should expect to find this complication frequently in enteric fever and dysentery, for example. As a matter of fact it is extremely rare in these diseases. In the *post-mortem* register of St. Bartholomew's Hospital I have not been able to find a single instance of pylephlebitis in enteric fever; and Dr. J. H. Bryant has kindly informed me that a search through the records of Guy's Hospital (which reach back to 1828) was equally fruitless.

Hugue¹ found that hepatic suppuration of any kind was very rare in enteric fever, and that if it occurred there was usually colon typhoid. With multiple abscesses there was usually pylephlebitis. He adds the interesting statement that this complication is more common in those who have already suffered from some lesion of the liver. Osler² has only seen one case of suppurative pylephlebitis in enteric fever, and here there was also an empyema of the gall bladder, with a sinus running back from the cystic duct which ended close to the suppurating branches of the portal vein. Romberg³ saw 87 necropsies out of 677 cases of enteric fever at Leipzig, and in only one was there pylephlebitis. Here, besides healing ulcers in the ileum, there was a suppurating phlegmon in the mesentery. Lannois⁴ saw one instance out of 71 necropsies of enteric fever. In this there was definite ulceration of the appendix.

It should be noted that in two of the above cases suppuration other than the typical intestinal ulceration occurred, and that in the third the appendix was involved—a point at which the portal vein is specially liable to invasion.

Again, though single abscess follows dysentery, multiple abscesses with suppurative pylephlebitis is a rare sequel. Marotte⁵ records a case, and Bryant's⁶ statistics give an example. I have only found one instance of it recorded at St. Bartholomew's Hospital. I am, of course, excluding cases where septic infection followed operation on a tropical abscess. We may conclude that it is decidedly rare for pyogenic organisms to invade the portal vein from the surfaces denuded by dysenteric ulceration. It has been suggested, moreover, by Councilman and Lafleur⁷ that even the *Amoeba coli* does not usually reach the liver by the portal vein, but through the peritoneal cavity. Councilman found circumscribed masses on the walls of a tropical abscess, which proved to be the persistent remains of the portal system.

Considering the liability of the gastro-intestinal tract to ulceration, and the varied nature of the microbes inhabiting that tract, it is at first sight surprising that portal pyaemia is not a more frequent occurrence. Elsewhere⁸ I have urged that "a collection of pus outside the gastro-intestinal tract is the real exciting cause of portal pyaemia. Ulceration of the tract is of importance as leading to this accumulation." This would explain why the region of the appendix is so frequently the focus, for nowhere else are "the conditions more favourable to . . . ulceration by faecal concretions or foreign bodies and to the production of such an accumulation of pus" within the portal zone.

Since writing thus, eight cases of suppurative pylephlebitis have occurred at St. Bartholomew's Hospital; a brief summary of them appears to me to lend considerable support to my contention.

CASE I (under Sir Lauder Brunton).—A female infant, aged 3 weeks, had a collection of dark material within the folds of the umbilicus; the umbilical vein was filled with pus, as were also the large portal branches in the liver, the superior mesenteric, and the splenic veins. There were no abscesses in the liver substance, but there was an abscess in the left lung communicating with the pleural cavity which had set up an empyema. Bacteriological examination showed streptococci (probably *Pyogenes aureus*) in pure culture from the portal pus and pleural fluid.

CASE II (under Mr. Bowlby).—A youth, aged 16, who had been operated on two years previously for tuberculous peritonitis, was admitted with a small discharging sinus and signs of portal pyaemia. At the necropsy the peritoneal cavity was found to be full of purulent serum containing flakes of lymph. The cause of the peritonitis was a leaking perisplenic abscess, which was surrounded by recent peritoneal adhesions. The spleen was much enlarged and was lardaceous. The liver was very much enlarged; the left lobe contained a large abscess, and the whole viscous was channelled by purulent tracks. The portal vein, with 1 in. of the splenic and superior mesenteric veins, was thrombosed; the clot was soft and mixed with pus, which extended into the liver substance. Some of the mesenteric glands were tuberculous; others were enlarged but apparently not tuberculous.

CASE III (under the late Mr. Walsham).—A youth, aged 15, was operated on for gangrene of appendix with abscess. At the necropsy general peritonitis was found. The portal vein was blocked by a breaking-down thrombus extending down to about 1 in. from the junction of the superior mesenteric and splenic veins. On the diaphragmatic surface of the right lobe the liver was destroyed by the presence of multiple abscesses, which were in places running together to form larger cavities. On section of the liver brownish pus exuded everywhere from the portal canals. Other abdominal organs natural.

CASE IV (under the care of Mr. Lockwood).—A woman, aged 36, died after the operation of cholecystotomy for gall stones. The abdominal wound led down to a contracted gall bladder surrounded by many old adhesions. The portal vein was plugged with septic clot, and the liver was riddled with abscesses containing bile-stained pus. The spleen was normal. The peritoneal cavity contained some bile-stained fluid.

CASE V (under care of Mr. Langton).—A man, aged 52, was admitted for a fluctuating swelling midway between the ensiform cartilage and the umbilicus. This was opened and proved to be a localized peritoneal abscess. Four days later a boil appeared at the right edge of the wound; this spread, in spite of treatment, until it covered almost the entire surface of the abdomen. Cultures from it showed *Streptococcus pyogenes aureus* and staphylococci, but no actinomycosis. After death the abscess cavity was found to be surrounded by many old firm adhesions and to lead upwards towards the liver. The hepatic flexure of the colon and the duodenum were bound to the under surface of the liver by firm adhesions. No ulceration was found in any part of the intestines. The stomach was small and contracted, and bore no evidence of old or recent ulceration. The liver contained multiple abscesses, and there was suppurative pylephlebitis. The mesenteric glands were somewhat enlarged. There was no evidence as to the origin of the abdominal abscess. Cultivations from the heart's blood showed *Bacillus coli* only.

CASE VI (under care of Mr. Bowlby).—A man, aged 29, was operated on for gangrenous perforative appendicitis, with an abscess on the outer side of the caecum shut off from the general peritoneal cavity. At the necropsy general purulent peritonitis was found with suppurative pylephlebitis.

CASE VII (under care of Mr. Lockwood).—An infant, 3 weeks old, was admitted with septic inflammation of the umbilicus and died within twenty-four hours. The umbilical vein was found to be much thickened and contained pus. There was suppurative pylephlebitis and suppurative peritonitis. The peritoneal fluid showed streptococci on cultivation.

CASE VIII (under care of Mr. Bailey).—A woman, aged 56, dying from cholelithiasis showed at the post-mortem examination general peritonitis, with two abscesses in the upper part of the right lobe of the liver and suppurative pylephlebitis. The gall bladder was very much shrunken, being only capable of holding a few minims of fluid. A probe could not be passed into the common duct; the liver at this spot was puckered and the duodenum was adherent to the gall bladder. The common bile duct was enormously distended and lying in the duodenal orifice (which was much dilated also) was a large gall stone. The pancreas was hard and not evidently inflamed. The spleen was large and soft. Cultivations from the peritoneal fluid grew *Bacillus coli*, streptococci, and staphylococci. From the heart's blood *Bacillus coli* alone grew.

I am indebted to the kindness of the physician and surgeons under whose care these cases were for permission to refer to them.

Of these 8 cases, then, 6 showed localized septic foci outside the alimentary canal, and in the other two the cause (gall stones) set up inflammation in the immediate neighbourhood of the liver. In both the cases, where a part of the alimentary canal (the appendix) was involved, there was also an abscess outside the appendix.

Adding these cases to those I have already collected and published from our records, we obtain the following table:

TABLE I.—*Etiology of Twenty Cases of Suppurative Pylephlebitis.*

a. Vermiform appendix and caecum:		
Perforation of appendix with abscess	7	
Thickening around appendix	3	10
b. Stomach:		
Gastric ulcer with localized abscess		
and old abscess in spleen...	1	
Malignant ulcer of stomach...	1	2
c. Localized peritoneal abscess not obviously dependent on gastro-intestinal ulceration		
...	2	
d. Spleen: small pyaemic abscesses	1	
e. Gall stones	2	
f. Infection from umbilicus	2	
g. Focus not found (mesenteric glands calcareous)	1	
		20

II.—THE RELATIONSHIP BETWEEN PYLEPHLEBITIS AND PORTAL PYAEMIA.

It is desirable that the terminology of disease should accord with pathological conditions so far as we can determine them during life, and the division of affections of the portal vein into adhesive and suppurative pylephlebitis is unsatisfactory for the following reasons:

1. In suppurative pylephlebitis the essential feature is portal pyaemia; and I shall attempt to show that we really have no means of distinguishing between cases of such pyaemia in which the vein is involved and cases in which it is free.

2. In "adhesive" pylephlebitis, so-called, the essential feature is the occlusion of the vein, and this may be produced by a variety of causes, among which sepsis is by no means an unimportant one.

(1) From the St. Bartholomew's Hospital records I have collected the cases of multiple hepatic abscesses in which the vein was not involved, and a comparison of their causation with those of the cases of suppurative pylephlebitis (Table I) is instructive. Out of the total 18 the infection was part of a general arterial pyaemia in 8. The focus in the remainder was as follows:

TABLE II.—*Portal Pyaemia without Phlebitis.*

a. Vermiform appendix and caecum:		
Perforation and ulceration of appendix	3	
Scars of old ulcer in caecum	1	4
b. Old tropical abscess which had been drained		
c. Perinephric abscess	1	
d. Abscesses about spleen	1	
e. Focus not found (in one of these the glands near the liver were calcareous or caseous)	3	
		10

As might be expected, the causes of portal pyaemia are closely similar whether the portal vein be or be not itself affected. Indeed, "phlebitis may exist without being discovered, and pyaemia may exist without phlebitis."

The symptoms and progress are the same. Similar lesions are established in the liver. Thus Payne¹⁰ recorded in the same paper two cases of suppuration in the liver consequent on irritation in the appendix. In one the portal vein (with its tributaries and branches) was quite natural; in the other, the superior mesenteric and portal veins were involved. Yet the cases were very similar in all other respects. At the Royal Free Hospital,¹¹ within a few months of each other, two cases of multiple hepatic abscesses following appendicitis occurred in men just over 30. In the first the portal vein, its right intrahepatic branches, the superior mesenteric and splenic veins, were all involved; in the second, all the portal system appeared healthy. Yet the cases ran very similar courses. The first was jaundiced and the second was not, apparently because in the former suppurative cholangitis had occurred by direct extension. It is interesting to note that the second case had rigors and marked enlargement of the spleen while the first had not. For both these points have been urged as diagnostic features in suppurative pylephlebitis as opposed to multiple hepatic abscesses without phlebitis.¹² These two cases show how little such signs are to be relied on in the diagnosis.

Indeed we may ask what reliable sign we have of the existence of phlebitis in a case of portal pyaemia? In 53 cases of suppurative pylephlebitis I have collected signs of portal obstruction were the exception; ascites occurred in only 6 instances, in 2 of them only to a slight extent. The super-

ficial veins were enlarged in 3 cases. Enlargement of the spleen was detected in 11 cases, but in one of these lardaceous disease was the cause of the enlargement, and toxæmia alone will enlarge the spleen.

The clotting in the portal vein and its tributaries is sometimes discontinuous in suppurative pylephlebitis. Thus, out of the cases I have collected, in 2 some of the veins near the caecum were involved, but the trunk of the superior mesenteric vein was not. In 2 other cases the pancreatic vein was involved, together with the duodenal vein in 1 of them; the portal vein and its branches contained pus, but no other tributaries were affected. Emboli become detached from the peripheral thrombosis and are carried up towards the liver, here starting another septic focus. But there is no reason why the infective particle should always find even one intermediate resting-place; it may be swept up in the blood stream from the primary focus, and not settle down till the capillaries of the liver are reached, thus producing portal pyaemia without phlebitis.

It would appear that the degree of intensity of the pyrogenetic affection determines whether pylethrombosis or suppurative pylephlebitis, as ordinarily understood, results. With rapid invasion and speedy softening of the clot the septic infection will reach the liver and set up typical portal pyaemia; with a slower or less virulently septic process the resulting thrombosis blocks the way for further inroads, and symptoms of obstruction rather than of portal pyaemia follow. Sometimes, by the development of an adherent clot in the portal vein after the infection has reached the liver, signs of portal obstruction become grafted on a case of portal pyaemia; rigors and a "typhoid" state are combined with ascites, and marked enlargement of the spleen, as in the first case that came under my observation.¹³ Thus the position of the adherent clot plays a very important part in determining the nature of the symptoms, and it will be just in the most "pyaemic" cases that the signs of obstruction will be best marked.

The commonest place for the adherent clot to form is at the junction of the superior mesenteric and splenic veins; of this I have been able to collect five examples.

To summarize—in some cases of portal pyaemia obstructive symptoms occur, but in the majority they do not; their absence cannot be regarded as excluding pylephlebitis; their presence is usually the result of a firmly adherent clot in the portal vein or its annexes, rather than of the soft disintegrating purulent thrombus.

(2) In the second place the objection to the term "adhesive pylephlebitis" is that the condition recognizable during life is one of portal occlusion, which may be the result of various morbid processes, to some of which a name implying inflammatory adhesion cannot fairly be applied.

In Table III I have grouped the causes of portal occlusion apart from portal pyaemia that have occurred at St. Bartholomew's Hospital since 1867. From this table it will be seen that invasion by new growth is not infrequently the cause of obliteration of the vein. In one such case where I had the opportunity of cutting sections of the portal vein I found the intima of the vein at the point of invasion destroyed, and the coats separated by the invading growth. Some of the vasa vasorum were choked with growth. A small vein running into the portal vein was quite blocked peripherally by growth, centrally by clot. Further away from the main vein the clot in this small vein showed growth right in its centre. In such cases the mechanism of the occlusion is clear.

TABLE III.—Causes of Portal Occlusion.

Cirrhosis of liver	11
Malignant disease of liver:	
Compression	1
Invasion	1
Malignant disease of pancreas:	
Compression	4
Invasion	1
Malignant disease of stomach:	
Compression	1
Invasion	2
Septic causes:	
Retrocaecal abscess	1
Sepsis following laparotomy	1
Cause doubtful:	
? Matting by adhesions	1
? Kinking by volvulus	1

But even in malignant disease we must not overlook the possibility of a terminal infection. In one case carcinoma

of the pancreas and liver was complicated by infective endocarditis, which may well have helped to produce the infarction of the intestines, from which the patient died.

Sepsis may lead to portal thrombosis, which raises an additional objection to the existing nomenclature. Thus in one case (previously reported by Dr. West¹⁴) there was a large abscess cavity about 6 in. long behind the caecum. The mesenteric vein and its radicles—the portal vein and its branches—were all thrombosed. In another case in my series there was cirrhosis of the liver, but also tuberculous ulceration of the intestines. In the portal vein was an adherent clot; the fact that this clot was breaking down into thick dirty fluid in its centre suggests a septic origin. In the following instance the septic nature of the clot was proved by bacteriological examination.

A kitchen porter, aged 43, was admitted under care of the late Mr. Walsham, with symptoms of cirrhosis of the liver. Laparotomy was performed and the great omentum stitched to the abdominal wall with the view of providing more efficient collateral circulation. He had severe attacks of abdominal pain four, seven, and ten days respectively after the operation. On the eleventh day he died. One and a half feet of the small intestine were deeply congested and almost gangrenous. The portal vein, its branches and the inferior mesenteric vein, were thrombosed. A large round-celled infiltration had taken place into the thrombus, and clusters of micrococci, staphylococci, and streptococci were seen in the surrounding vein wall.

Here was a case of pylethrombosis associated with invasion by pyrogenetic organisms, yet it was not suppurative pylephlebitis that followed.

In the literature of pylethrombosis I have found cases recorded where a septic origin was, to say the least, probable.

Bradford¹⁵ records pylethrombosis resulting from a suppurating mesenteric gland communicating with a vein; in a case of Rolleston's¹⁶ there was old dysenteric ulceration and recent inflammation of the rectum; while a concretion was ulcerating through the appendix. In a case of Treves's¹⁷ which recovered but which had symptoms of thrombosis of superior mesenteric vein, the patient was suffering from dysentery when these symptoms appeared. In another case¹⁸ the patient had been recently confined and that she was sapraemic was shown by the existence of phlegmasia alba dolens.

Out of 48 cases I have collected from the hospital records and from the literature a septic origin for the occlusion was therefore present in 7, or approximately 14.6 per cent.

Whether the cause of the obstruction to the vein be external compression (by cirrhosis, new growth, or chronic peritonitis), invasion of the lumen by new growth, atheroma of the walls, or septic thrombosis, the symptoms are very similar. Ascites and enlargement of the spleen occur in three-quarters of the cases, haemorrhages into the gastro-intestinal tract and diarrhoea occur in half the cases, and enlargement of the superficial abdominal veins in one-third of the cases. Gastro-intestinal haemorrhages or the signs of intestinal obstruction are the leading symptoms of cases with acute onset, ascites that of cases with gradual onset.

As it is the physical condition of the vein, independent of the pathological cause, which leads to these characteristic symptoms, I suggest that the name "portal occlusion" would be more satisfactory than "adhesive pylephlebitis," because it connotes less.

CONCLUSIONS.

1. A collection of pus within the portal zone is the principal cause of portal pyaemia. Ulceration of the alimentary tract is only important as being a frequent precursor of such a collection.

2. Signs of portal obstruction are the exception in suppurative pylephlebitis.

3. There are no signs by which we can distinguish with certainty cases of portal pyaemia with suppurative pylephlebitis from those in which the vein is not involved.

4. In "adhesive pylephlebitis" the essential feature is the occlusion of the vein, and this may be produced by a variety of causes, among which sepsis is not an infrequent one.

5. Therefore, if our terminology is to accord with pathological conditions so far as they can be determined during life, we ought to speak of two conditions, portal pyaemia and portal occlusion, remembering that we may meet with cases of portal pyaemia with occlusion.

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OBSERVATIONS ON THE OPSONIC INDEX OF PATIENTS UNDERGOING SANATORIUM TREATMENT FOR PHTHISIS,

WITH SPECIAL REFERENCE TO THE EFFECT OF EXERCISE.

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IN the summer of 1904 Professor Wright was kind enough to examine the blood of several of the patients in this sanatorium, and to estimate their opsonic power against tubercle. It was at once seen that certain differences in opsonic index were associated with certain differences in clinical character, and some cases, which at first appeared to be exceptions to this view, showed by their subsequent course that they were not. The opsonic index in these cases proved a more correct indication of their true character than their clinical appearance and previous history.

These blood examinations were made with the view to the use of "new" tuberculin, and since then a series of cases has been treated with this remedy with encouraging results, which we hope shortly to publish.

The work of estimating the opsonic index is now done at the sanatorium, thanks to Dr. Wright and Captain Douglas, I.M.S., who very kindly took much trouble to teach us the technique, and it is therefore possible to make a large number of observations every week.

Since the word "opsonin" is still a new one, we may, perhaps, be excused if we explain briefly what is meant by "opsonic index," and how it is estimated. It has been shown by Wright and Douglas¹ that the rate of ingestion of bacteria by polynuclear white blood corpuscles depends upon the extent to which the bacteria have been previously acted on by some substance existing in serum to which the name "opsonin" has been given. There appears to be a separate opsonin for each

variety of bacteria. The presence of opsonin is determined, and its quantity in a given serum is estimated, by the rapidity with which polynuclear white corpuscles devour bacteria which have been exposed to the action of the serum in question.

The experiment is performed by mixing together equal quantities of (1) serum-free corpuscles obtained by washing blood in 1 per cent. citrate of soda in normal salt solution and afterwards in normal salt solution only, (2) an emulsion of tubercle bacilli, and (3) the serum to be tested. The mixture is incubated at a temperature of 37°C. for twenty minutes, and then a film is prepared and stained in the usual way with carbol fuchsin and methylene blue. The average number of bacilli ingested by each polynuclear white corpuscle is ascertained, and, taking the average number ingested when the serum of a healthy man is employed as unity, the opsonic value of any other serum is expressed in terms of this. Thus, if the average number ingested by each polynuclear white corpuscle of healthy serum is 5, and with the patient's serum is 4, then the healthy man's opsonic value being called 1, the opsonic value of the patient will be 0.8; on the other hand, if the experiment with patient's serum gives an average ingestion of 7 then his opsonic value will be 1.4.

It was noticed at the outset that our results, while showing the same range of variation after tuberculin injections, were, taken on the whole, much lower than those obtained by Dr. Wright, and we were quite unable to endorse the view

expressed by Dr. Urwick in the *BRITISH MEDICAL JOURNAL* (July 22nd, 1905) that "the opsonic power of the majority of patients suffering from pulmonary tuberculosis is higher than normal." It seemed that either our controls were higher than those of Dr. Wright and Dr. Urwick, or our class of patients differed from theirs. A comparison of our results with results obtained in Dr. Wright's laboratory by a simultaneous examination of the same blood showed that our control only differed from Dr. Wright's by 0.05. Clearly, therefore, the discrepancy could not be due to a difference in controls. While still seeking for an explanation, it occurred to us that the samples of blood sent to Dr. Wright had always been collected at 2.30 p.m., in time to catch the afternoon post, while the samples examined by us were taken at 9 a.m. A series of observations was then made to ascertain what variations, if any, took place during the day, and the times chosen were those which fitted in with the routine practised in this sanatorium. All patients are visited before they rise in the morning, and are given directions as to how they are to occupy their time until noon—that is, either by walking