tinins, in cases of Shiga infection, co-agglutinins to the *B. dysenteriae* Flexner were encountered from time to time, but as these were present in lower titres than those given by the specific infecting organism no difficulty arose on this account. In connexion with the agglutination of the concomitant non-lactose-fermenting organisms it is possible that group agglutination might explain some of the results, but in others where a high titre was obtained such an explanation cannot hold.

The hypothesis on which this investigation was based had its origin from a consideration of the well-known pathological facts concerning bacillary dysentery, but in 1919 Besredka,¹⁵ of the Pasteur Institute, published an account of the experimental investigations he had carried out in connexion with the mechanism of infection in typhoid, paratyphoid, and dysenteric infections. He succeeded in killing several rabbits by giving them killed cultures of the Shiga bacillus by the mouth, subcutaneously, and intravenously. However infected, by the blood-stream or by the subcutaneous route with living cultures, or with killed cultures, or endotoxin, the animal reacted only by its intestinal apparatus. His experiments led him to the conclusion that the immunity is established in the intestine at the site of the lesions following the first ingestion of the bacillary bodies.

My view is that the intestinal exudate, although subject to extraneous additions modifying its titre, is the fluid "par excellence" for the examination of antibodies to the *B. dysenteriae* and its concomitant organisms, and while recognising that my investigations are incomplete these notes are published in the hope that some observer with time and material at his disposal will investigate the matter more fully, especially in view of Besredka's researches. My belief is that the examination of the antibody content of the intestinal exudate in bacillary dysentery is of great value from the point of the laboratory worker, dependent as he often is on a specimen of stool only and often sorely pressed for time and assistance when dealing with epidemic dysentery. Dudgeon¹⁶ states that under the most favourable conditions a competent bacteriologist can determine whether an infection is due to the Shiga or Flexner bacillus in 12 to 16 hours, and adds that this fact is of little value under the conditions that exist with an army in the field. By an examination of the intestinal exudate, using Garrow's technique, recognition of the infecting organism may be obtained in 12 to 16 minutes. From a therapeutic point of view this method makes it possible to employ a pure anti-Shiga, or a pure anti-Flexner serum at the earliest opportunity (a desideratum emphasised by Dudgeon), and further to follow up such treatment by employing anti-sera or vaccines prepared from such concomitants demonstrated to be operative in any given case, as elicited from an examination of the intestinal exudate.

Summary.

This paper deals with an attempt made to ascertain whether the examination of the fluid portion of dysentery stools would afford information as to the nature of the dysenteric infection. It is based on the argument that in bacillary dysentery specific antibodies to the *B. dysenteriae* would be present, whereas in amoebic dysentery, caused by a protozoal agent, such bacillary antibodies would be absent from the intestinal exudate; the intestinal tract being the seat of infection, the fluid exudate would contain a larger antibody content at an earlier date than the blood serum, and therefore an early diagnosis could be made. The antibody investigated was the agglutinin. Details of technique and the results obtained both in early and in late cases are given. Agglutinins were found to be present in low titre, and their significance is discussed. Besredka's researches showing that the immunity in bacillary dysentery is established at the site of the intestinal lesions are quoted, and in view of Besredka's findings the hope is expressed that the intestinal exudate may receive attention

from laboratory workers with the view of ascertaining whether a method may be established which has the advantages of simplicity and rapidity and of involving the minimum of laboratory equipment. It is submitted that this method affords an additional means of dealing promptly with an outbreak of dysentery from the points of view of diagnosis and treatment and is of special value in adverse surroundings.

I am deeply indebted to Captain William Rutherford, M.C. (O.C. No. 3 Military Laboratory) for his unfailing generosity and good comradeship during the difficult time of May-June, 1918. To Captain R. P. Garrow I wish again to express my thanks for his invaluable technique, and my thanks are given to Colonel H. S. Anderson, C.M.G. (O.C. Citadel Military Hospital, Cairo), and also to Major Charles Kerr. To Major A. R. Ferguson, O.B.E., and to Colonel Carmalt-Jones I am grateful for their encouragement, and also to my Director, Prof. R. Tanner Hewlett, for his help and advice in preparing this paper.

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CHEESE POISONING:

WITH SPECIAL REFERENCE TO THE DOVER OUTBREAK.

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OUTBREAKS of illness following the consumption of cheese are events of great rarity in this country. An outbreak which was clearly traced to cheese occurred in London in 1901, but the majority of recorded outbreaks have been in America. The main features of cheese poisoning from the ætiological and clinical sides resemble those of poisoning from other infected foods, but one or two minor points of difference might be indicated. In the first place the case-mortality rate is lower. From the records which are available, comprising many hundreds of cases, I have been unable to find evidence of a single fatal case in any outbreak where the circumstances incriminating cheese have been at all conclusive; whereas in other food-poisoning epidemics the case-mortality rate is in the neighbourhood of 1.5 per cent. Secondly, the incubation period tends to be rather shorter in cheese poisoning; and thirdly, the usual food-poisoning organisms are hardly ever found in epidemics due to cheese.

The Nature of Cheese Poisoning.

When we come to consider the nature of cheese as a food an explanation for these differences becomes apparent. Cheese occupies a unique position among our foods. It is the only article of food in this country which, manufactured many weeks or months before it is intended for consumption, is not subjected to any process of preservation during that period. Moreover, bacterial action, causing proteolytic and other changes, so far from being inhibited, is encouraged along definite channels in order to bring about ripening. It would seem very unlikely, therefore, that in the event of pathogenic micro-organisms being introduced into a cheese in process of manufacture, such organisms would remain alive at the time of consumption, having regard to the conditions of temperature, &c., being favourable to an enormous overgrowth of the sapro

phytic bacteria and moulds associated with the ripening process and unfavourable to the growth of pathogenic organisms.

We know, however, that organisms of the Gaertner group—the bacilli almost invariably associated with this type of food poisoning—produce an extremely stable toxin, capable of withstanding a temperature of 100° C. for five minutes, or more, without being destroyed. It would seem likely, then, that this toxin would remain unaltered during the ripening process, while the micro-organisms from which it was derived were overgrown and killed by the multiplication of saprophytes.

Cheese poisoning, therefore, viewed from this standpoint, would be a pure intoxication, as distinct from poisoning in which other articles of food are the vehicle, where there is usually a combination of intoxication and infection with Gaertner organisms. This explanation is, of course, in the regions of hypothesis, and further research is necessary to confirm it, but in intoxication as distinct from infection one would expect: (1) a shorter incubation period; (2) a milder attack, with fewer fatal cases; (3) rapid recovery following elimination of toxin; (4) recurrence of symptoms on ingestion of second dose of toxin. In the Dover outbreak which occurred in July of this year these points are very clearly brought out; furthermore, the evidence incriminating cheese is most definite and convincing.

The Dover Outbreak.

On Saturday, July 1st, 1922, the first cases of poisoning made their appearance. The symptoms, the details of which will be described later, were, in the main, abdominal pain, diarrhoea, and vomiting. Forty-three families, comprising 126 individuals, were discovered to have been attacked in this way, most of the cases occurring on the morning of Sunday, July 2nd. As far as I am aware, no fresh cases arose after July 5th. The outbreak was confined to a small district, within the Borough of Dover, entirely inhabited by the working-class. In every one of the 126 individuals who were affected the symptoms commenced a few hours after a meal at which cheese had been eaten. In every case the cheese had been purchased from the same shop, and no other article of food was a feature common to all the cases.

During the course of the investigations, the manager of the shop in question informed me that a new imported cheese was first cut on the late afternoon of Friday, June 30th. It was on sale all the following day and the last of it was sold out on Monday morning, July 3rd. Only those individuals who ate cheese purchased from this particular shop between these hours were affected. This particular cheese, moreover, possessed a rather distinctive appearance, being very dark, almost red in colour, somewhat resembling the colour of Gloucester cheese; accordingly, it was not difficult to trace its connexion with every case investigated. Other cheeses were on sale at the same shop during the same hours, notably a white imported "Cheddar" cheese, but no ill-effects could be traced to these, nor to any other article of food, cooked meats, &c., purchased from this shop.

The cheese was one of a batch of "red Canadian Cheddar" cheeses, shipped from Montreal to a wholesale house in London, whence it had been sent to Dover about a week before the outbreak occurred. The weight of these cheeses is in the neighbourhood of 80 lb., and as the peccant cheese was completely sold out in pieces averaging about 1 lb., or rather less, it follows that it must have found its way into at least 80 households, in only 43 of which it was subsequently traced. In appearance, taste, and smell the cheese seemed perfectly sound and wholesome. A few minute holes, spherical as though formed by bubbles of gas, were noted in pieces of the peccant cheese which were not seen in other cheeses of the same batch.

The shop was a general store, dealing chiefly in grocery and provisions. The members of the staff were examined to exclude the possibility of one of them being a carrier. They were all in good health

and had not been in contact with any cases of sickness. It was fairly apparent from the start, however, that a carrier hypothesis would not account for the outbreak, as the poisonous effects were confined to a certain definite cheese, and were not shared by any other food, cooked meat, brawn, &c., sold in the shop. All circumstances seemed to point to the cheese having become contaminated in process of manufacture. To trace the cheese back to the farm in Canada where it was produced has so far proved impracticable, and so, as in so many recorded outbreaks, the last link in the chain of evidence is missing. Every house in which a case of poisoning occurred was visited by me personally, and the history was elicited from the various members of the family, who were encouraged to tell the story in their own words, leading questions being avoided as far as possible. The similarity of the history in the different houses visited was most striking.

The following typical cases are quoted to indicate the convincing evidence against the "red" cheese, and to illustrate some of the clinical and ætiological features referred to in the text.

CASE 18.—Mr. Y., aged 30, Mrs. Y. (30), their daughter (7), with Mr. and Mrs. Z. (26 and 21) sat down to a supper of bread, butter, cheese, and cocoa on July 3rd at 9.30 P.M. At 6 A.M. the following morning Mrs. Y. was awakened with severe abdominal pain followed by diarrhoea. About 7 A.M. the child was seized with very bad abdominal pain, vomiting, and diarrhoea, and about an hour later Mr. and Mrs. Z. complained of the same symptoms. They felt so ill that all remained in bed for the day. Mr. Y., who had eaten no cheese for supper, remained perfectly well. The cheese (red) was bought at the X stores on July 1st.

CASE 12.—Mr. and Mrs. Q. (aged 47 and 44 respectively) had supper of cold lamb, bread, and cheese; and their two daughters bread and cheese only at 10.30 P.M. on July 1st. About 6 A.M. the next morning Mrs. Q. and one daughter were seized with severe pains in the abdomen, vomiting, and diarrhoea. Shortly afterwards Mr. Q. and the other daughter were similarly affected but did not vomit. On the 3rd the married son and daughter-in-law of Mr. and Mrs. Q. returned home from holiday, and at 9.45 P.M. had supper of bread, butter, and stout. The daughter-in-law ate in addition some of the same cheese. At 5.30 A.M. the next morning the daughter-in-law had very violent pains, diarrhoea, and vomiting. The son remained perfectly well. "Red" cheese bought at X stores, evening of June 30th.

CASE 10.—Three sisters, A. (51), B. (40), and C. (62) ate a meal of boiled mutton, potatoes, cabbage, and gooseberry pudding, followed by cheese at 12.30 P.M. on July 3rd. At 4 P.M. A. was taken ill with colic, which doubled her up, severe diarrhoea, and vomiting. She became very weak, pale, and collapsed, and "thought she was going to die." After a few hours the acute symptoms abated. At 10 P.M. B. had a similar attack but of less severity. About the same time C. complained of mild abdominal pain and a little diarrhoea. All ate very little and approximately equal amounts of cheese. On the 5th A., not suspecting the cheese, ate some more, and had a return of the pain. "Red" cheese bought at X stores on July 1st.

CASE 7.—Mr. O. (72), Mrs. O. (71), I. I. (9), and G. E. (20) had supper of bread and cheese at 9.30 P.M. on July 1st. In the early hours of the following morning the first three named were awakened with violent abdominal pains and diarrhoea, and Mrs. O. and the boy, I. I., with vomiting also. G. E., who had eaten more cheese than any of the others, remained entirely unaffected and completely free from any symptoms throughout. On the 2nd, at 9.30 P.M., F. I. (30) ate some bread and cheese and complained the following morning of a little diarrhoea and some mild pain in the abdomen. "Red" cheese bought at X stores on July 1st.

Ætiology.

Age and Sex.—All ages and both sexes were indiscriminately affected, and no relationship could be established between either of these factors and the severity of the attack or the length of incubation period.

Season.—In food poisoning epidemics, seasonal prevalence is very marked, the vast majority occurring in June, July, and August. The Dover outbreak is no exception to this, although the significance in this case is a little vague, as there is no evidence at present available to determine when the cheese was infected.

Infectivity.—Out of a total of 135 individuals, who were known to have eaten the peccant cheese, only 9

(6·7 per cent.) remained in good health and free from the symptoms of poisoning. The infectivity rate is, therefore, very high (93·3 per cent.).

Severity of Attack.—As has been found by most investigators in food-poisoning outbreaks, no relationship could be traced between the severity of the symptoms and the amount of peccant food eaten (see Cases 7 and 10). It seems very probable that symptoms were the result of preformed toxic substance in the cheese and not of ingestion by the victims of a food-poisoning organism. If this is so, the absence of relationship between severity of attack and amount of cheese eaten would argue either an uneven distribution of toxin throughout the cheese, or a varying degree of tolerance among the victims.

Incubation Period.—The incubation period was very short. The longest discovered in this outbreak was only 11 hours, the shortest 3½ hours. The average incubation period lay between 7 and 8 hours.

Recurrences.—These are of some interest, and three such cases occurred in the Dover outbreak. These three individuals, after recovery from the initial attack, and not suspecting the cheese to be the cause of their illness, ate some more, and after another short incubation period these persons suffered from a second acute attack, similar to the first (see Case 10). In two of these cases, the secondary illness was milder than the primary, while in the third it was of greater severity.

Another type of illness was observed, also where the cheese was not suspected—the continued type. Here very mild symptoms were produced, a little abdominal pain and looseness of the bowels during the days the cheese was being eaten. These symptoms persisted until the cheese was finished when they ceased. Seven such cases were discovered, one of which culminated in an acute attack of great severity.

Mortality.—No deaths occurred in this outbreak.

Clinical Condition.

Pain.—Abdominal pain was a constant symptom, being complained of in every case without exception. In the majority of instances the pain was severe, paroxysmal in type, described generally as “cutting” in character and causing the patient to double up or to roll about in an endeavour to find a position of ease. The intensity diminished after two or three hours, but it was reported in many cases that spasmodic pains tended to recur at intervals for the following day or two, especially soon after food. A small proportion of those affected were able to carry on with their work, but in most cases the severity of the pain caused them to give up and take to their beds or to lie down.

Diarrhoea.—This was the next most constant feature, being present in 89 per cent. of all cases. This likewise varied in degree and frequency, but in a few cases the loss of tissue fluid by the bowel was so profuse as to give rise to obvious “pinching” of the features and shrinkage of the limbs, which were remarked upon. Cramp in the calves was not complained of.

Vomiting.—This was present in 63 per cent. of cases, and nausea among many of the remainder. A feeling of sickness and a distaste for food, sometimes accompanied by an unpleasant taste in the mouth, generally persisted for 48 hours.

Prostration.—This was a most marked clinical feature. A few of the sufferers recounted very accurately the symptoms of collapse during the acute stage—extreme weakness, pallor, cold sweat, palpitation and rapid breathing, and fear of impending death—but every individual examined remarked on the debility which followed the acute attack and which lasted for three or four days.

Fever.—Most of the patients complained of feeling “feverish” during the attack. The only three cases seen by me in the subacute stage had temperatures of 99·2°, 99·2°, and 99·6° F. respectively. The last-mentioned had a temperature of 98·6° two days later.

Rash.—Noticed in only one case. A woman, highly neurotic, described a blotchy nettle-rash, which appeared suddenly 48 hours after the onset of symptoms, and which was distributed all over the

body. It was accompanied by intense itching and nausea, and disappeared within 12 hours.

Nervous Symptoms.—Palpitation and weakness have already been mentioned, and one man described, in a recurrent attack, flashes of light before the eyes.

Laboratory Investigations.

For the following results I am indebted to the experimental work of Dr. W. G. Savage and Mr. P. Bruce White, carried out in connexion with the research investigation on food poisoning which is being undertaken for the Medical Research Council in coöperation with the Ministry of Health.

Chemical Examination.—A thorough examination of the samples for tyrotoxin was negative. Search was made in cheese and rind for arsenic, antimony, and other poisonous metals, but no trace could be found.

Bacteriological Examination.—Examination of the cheese and of faeces, urine and vomit of sufferers, by aerobic and anaerobic methods on various media offered no clue. No organisms known to be associated with food poisoning were found. From one sample platings gave 2–3 colonies of Morgan's No. 1 bacillus. Serum from sufferers failed to agglutinate any organisms of the Gaertner group.

In some of the continental and American outbreaks the infective properties have been ascribed to toxic substances produced by special types of *B. coli*. In the cheese under consideration *B. coli* strains were present, as in many normal cheese samples, but direct examination yielded no evidence that they or their products were toxic by feeding. As further evidence of the absence of living toxin-producing bacteria, it may be mentioned that sound cheese, inoculated with an emulsion from the infected cheese, and incubated under various conditions, remained non-toxic to mice.

Feeding Experiments.

(a) *Mice.*—Mice are definitely susceptible to the “toxin”; but in the series of experiments performed the results were irregular (perhaps indicating an irregularly distributed poison). In some cases the effects were noted within 24 hours of the first meal; in others, symptoms did not appear until after several days of feeding; in yet others no symptoms were noted throughout (8–10 days). The symptoms shown by the affected mice were: Soft but not liquid stools; apparent paralysis or weakness of the hind legs, causing the animals to walk “splay-footed,” in some cases the animals became wet and ceased to care for their appearance; two mice died with spasmodic movements of the whole body, and others showed tremors. Post mortem, two of the mice showed marked hæmorrhage of the gastric and intestinal mucosa. In others, minute petechial hæmorrhages were seen. In several cases the spleen was seemingly enlarged. The mice were carefully examined for infecting organisms. Aerobic and anaerobic cultures of the intestinal contents and organs led to the conclusion that Morgan's No. 1 bacillus was the only organism that could be regarded with any suspicion. In many cases this organism was found in large numbers in the stomachs of the affected mice, and occurred (sometimes in pure culture) in the spleens of several. Knowledge of this organism, however, strongly suggests that it is a secondary invader, following on some primary alimentary irritant. Further, the same organism was isolated from the stomach of one of the control mice.

(b) *Rabbits.*—A rabbit was twice “force-fed” with large doses of the suspected cheese. It was totally unaffected. Subcutaneous injections of cheese emulsions into guinea-pigs and rabbits brought out nothing of importance.

(c) *Human.*—Mr. Bruce White ate a mixed sample of cheese, about 5 g., at 9.30 P.M., and was awakened about 4.30 A.M. with nausea and headache. Nausea and headache continued throughout that day, but had more or less disappeared the following morning. Alcoholic extracts of the cheese prepared at room temperature, and residue from alcoholic extraction were separately consumed on

several occasions without ill-effect. Aqueous extracts, prepared by extracting cheese at 52° C. for 48 hours, were taken without ill-effect. An extract was then made at room temperature with grinding and shaking for 20 minutes. A quantity of this extract, corresponding to 7 to 8 g. of cheese, was consumed, and after several hours considerable discomfort was felt which persisted for two days.

From these feeding experiments on whole cheese and extracts the pathological experts conclude: (1) That mice and humans are susceptible, rabbits not; (2) that the poison is not alcohol-soluble and is probably destroyed by alcohol; and (3) that the poison is water-soluble, but that in solution it is probably destroyed by a temperature of 52° C. acting for 48 hours.

These several points suggest that the poison is in the nature of a protein or true "toxin."

Summary.

1. The Dover outbreak was not due to inorganic

poisons, to tyrotoxin, nor to the infection of the victims with a food-poisoning micro-organism.

2. The outbreak was almost certainly due to a bacterial toxin.

3. The close resemblance of the Dover outbreak, clinically and ætiologically, to a Gaertner-group infection, suggests very strongly that the toxin of a Gaertner-group organism was the cause of the Dover outbreak.

4. Such delicate organisms as those of the Gaertner group, introduced during the process of manufacture, would be unlikely to survive maturation of the cheese, although their stable toxin would doubtless persist.

5. Considerations referred to in the text point to toxin of a Gaertner-group bacillus being the usual cause of those outbreaks of poisoning in which cheese is the vehicle.

My thanks are due to Dr. A. B. McMaster, medical officer of health, Dover, for permission to publish these observations, and for his valuable criticism.

Medical Societies.

ROYAL SOCIETY OF MEDICINE.

SECTION OF SURGERY.

THE RISE AND FALL OF SURGICAL OPERATIONS.

At a meeting of this section of the Royal Society of Medicine, held on Nov. 1st, Mr. JAMES BERRY delivered his presidential address on the Progress of Surgery and the Rise and Fall of Surgical Operations. He said that during his surgical life he had witnessed the most wonderful progress in surgery that had ever taken place in so short a time.

As a student he saw the last of the pre-antiseptic days. Since then he had watched the development of antiseptic surgery, and later still the gradual development of the aseptic régime, under which so many of the greatest advances of modern surgery had been made. In the eighties operative surgery of the skull and brain consisted of little more than the trephining and elevation of depressed fractures and the occasional (and usually fatal) opening of a cerebral abscess. Deep dissecting operations at the base of the neck were rarely performed on account of the dangers, in those days, of opening up the cellular tissues. Thoracic surgery was almost limited to the opening of empyemata, and abdominal surgery was in its infancy. The then existing fear of opening the peritoneal cavity was fully justifiable, for he had seen a man die of acute peritonitis after a trivial operation upon the abdominal wall, which had involved a small opening of the peritoneum. Hysterectomy in those days was done with a wire écraseur, and cancer of the tongue was removed by the barbarous chain or whipcord écraseur. Lithotomy was usually performed by the perineal route, and lumbar colotomy was being replaced by the inguinal operation, then not infrequently followed by such accidents as the prolapse of the bowel and the escape of small intestine into the bed owing to the insufficient closure of the abdominal wound. It was a truism that most of the progress made in surgery was due to the application first of antiseptic, and later of aseptic, principles to the treatment of operation wounds. He remembered seeing surgeons of eminence operating in old frock-coats, splashed and stained with blood and pus; the more dirty the coat, the greater seemed to be the pride of the surgeon who wore it. The surgeon of those days might not even wash his hands before undertaking an operation.

Value of Pathology to the Surgeon.

The success of many of the highly technical present-day operations depended not merely on skilful technique and strict asepsis, but also in a thorough knowledge of the pathological conditions likely to be met with. The lack of this knowledge might easily

lead to a fatal disaster. Cholecystectomy, for example, was a very successful operation in the hands of skilled surgeons, but much judgment was required to know when it should be performed, and when the simpler operation of cholecystotomy would be preferable. A cholecystectomy that would be the right operation in a particular case for a Moynihan or a Mayo, might be the wrong operation for Mr. A. or Mr. B. A slight error in the placing of the suture in a gastro-enterostomy, the careless application of a single ligature in a thyroidectomy, the incorrect placing of a clamp in a cholecystectomy might easily lead to the death of the patient. There was no such thing as the mortality of such and such an operation, for that depended upon the condition for which it was done, upon the operator who did it, and how and when it was performed. With our greatly increased accuracy in diagnosis, and better knowledge of pathology and technique, it had now become possible to penetrate with impunity to regions of the body which formerly no surgeon would have dared to approach. Extensive operations upon the bladder, the intestine, the stomach, the biliary passages, the lungs, the thyroid, and the brain were among the wonders of modern surgery.

At one period of his career Mr. Berry was surgical registrar for more than five years to one of the largest London hospitals, and at the same time full surgeon to another large hospital. It was part of his duty as surgical registrar to perform all the surgical post-mortem examinations, an experience which he found invaluable when operating on the living subject. He could not help feeling that the modern practice of entrusting surgical post-mortem examinations to a pathologist who was not interested in small matters of surgical detail, so important to the operating surgeon, was, in some respects, a retrograde step. The loss to the young surgeon of this valuable post-mortem room training was surely obvious. After a year or two in the anatomy department the young aspirant to a surgical career should soak for several years in pathological museums, in operating theatres, and in post-mortem rooms. He must learn his anatomy and surgical pathology thoroughly. He must watch the performance of innumerable operations, and see thousands of clinical cases before he was really fit to practise first-class general surgery. He must be prepared to have in his earlier days a hard time, for he should not expect to do much surgical practice of a remunerative kind until he had had 10 or 20 years of hard work at learning the elements of his profession.

Operating in the Presence of Sepsis.

While the results of aseptic operations were brilliant he (Mr. Berry) questioned whether we could congratulate ourselves as much upon the results of operations undertaken where sepsis already existed, more especially in acute suppurative conditions of the abdomen. In these cases it was far more difficult for the young