importance to this country of observing its behaviour as notified by conditions in Europe is such that study of it in its epidemiological, prophylactic, and curative aspects should be undertaken by Government. To this end it would appear to me advisable that a small Commission of experts—of physicians, epidemiologists, and bacteriologists—should be sent to the Peninsula for that purpose. It is a matter of Imperial concern and admits of no delay. No expense should be spared, for if the matter is considered from no higher standpoint than a financial one experience has shown that plague wherever it has acquired a firm hold disorganises and paralyses trade to such an extent that the money losses are immense. The quarter of a million lives lost in India show the destructiveness of the disease and the importance of making every effort to learn more about plague with the object of its prevention.

Hitherto I have said nothing as regards general sanitation. That attention to this is necessary goes without saying. While preparing to immunise individuals with Haffkine's prophylactic every endeavour should be made to immunise the locality by sanitary measures. But in this connexion it should be borne in mind that excellent water-supplies are of little protection against plague; and as it has been perhaps to the acquisition of pure water-supplies that the attention of sanitarians has of late been chiefly directed while measures for the dryness and purity of soil have taken but a secondary position, it is therefore to the latter branch of sanitation that I would commend that special attention be paid. Localities in which overcrowding, poverty, and filth exist have always been the favourite haunts of plague, and it is on these localities in every country and in every large town that protective efforts should be concentrated by the authorities concerned, in order that if the first line of defence is broken through the plague may not find conditions favourable to its growth and rapid spread. Poverty we have always with us; overcrowding may be lessened, or at least regulated or watched, and filth may be easily removed and the dirty spots cleansed.

MEDITERRANEAN OR MALTA FEVER,

WITH SPECIAL REFERENCE TO THE SPECIFIC AGGLUTINATING SUBSTANCES WHICH MAKE THEIR APPEARANCE IN THE BLOOD IN THE COURSE OF THAT DISEASE.

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MEDITERRANEAN fever is a disease too seldom recognised. It is often disguised under such names as simple continued fever, sweating typhoid,¹ &c., accounts of which still unfortunately find a place in current medical literature. Clinically, the main features of Malta fever are briefly stated as follows: pyrexia of a more or less chronic type, accompanied by constipation and copious perspiration; relapses are frequent, and convalescence is often retarded by neuralgias, joint-pains, and orchitis. This fever is the cause of much sickness among the British troops stationed at Malta and Gibraltar. Many men in the convalescent stage are invalided to England and come under treatment in the Royal Victoria Hospital, Netley. Among these men relapses are not unfrequent after their arrival and thus a wide field for study of the disease is afforded.

History of the bacteriology of Malta fever.—In 1887 Bruce² isolated a micro-organism in pure culture from the spleens of nine fatal cases and from blood drawn from the spleen during life in two instances. This organism he named the micrococcus melitensis. He succeeded in infecting monkeys by subcutaneous injection of small quantities of the pure culture of this coccus. In these animals it gave rise to a continuous fever, not unlike that in man, and usually caused

death after a variable time. In the monkeys which died from this infection he found in pure culture in the spleen the same bacterium as he had inoculated. Hughes³ has corroborated these observations of Bruce. He has isolated the micrococcus melitensis from the spleens of 14 men who succumbed to this disease. Further, he has infected monkeys with the cultures of the organism and, like Bruce, afterwards recovered the micro-organism from the spleens of the animals which died. Gipps⁴ has also obtained the same micrococcus in two fatal cases. In this country we have grown the microbe from the blood and spleens of the only two fatal cases which have come under our notice.⁵ The observations of the disease in infected monkeys, as described by Bruce and Hughes, have been amply confirmed in this laboratory by a long series of experiments undertaken some two years ago by Professor Wright and Major D. Semple with an organism sent from Malta by Hughes. We also have infected monkeys with the cultures obtained from the two fatal cases mentioned above. As the details of these experiments coincide in almost every particular with the descriptions previously given by Bruce it is unnecessary to enter further into them. Durham⁶ has shown that the infection can be conveyed to rabbits and guinea-pigs (animals regarded by Bruce as immune to the disease) by means of intra-cerebral and intra-peritoneal inoculation. These observations have also been confirmed in this laboratory by Professor Wright and Major D. Semple. Various attempts have been made by us to isolate the organism from the urine during the course of the fever in man. These have in all cases been unsuccessful. The plates either remained sterile or were overgrown by more quickly growing bacteria. No bacteriologist has described the micrococcus melitensis as having been isolated in any other disease.

Biological characteristics of the micrococcus melitensis.—These have been fully described by Bruce,⁷ Hughes,⁸ and Durham.⁹ A brief résumé of their and our own observations will therefore suffice. The micrococcus melitensis is a small coccus or cocco-bacillus about 0.33 μ in diameter, the bacillary form being more pronounced when grown on gelatin. We have not been able to satisfy ourselves as to the presence of any motility other than Brownian movement. Gordon,¹⁰ however, figures it as possessing usually a single and sometimes three or four short flagella. Neither Durham¹¹ nor the workers in this laboratory have been able to confirm this observation. It stains readily with all the basic aniline dyes, while it does not retain its stain by the Gram-Weigert method. The growth of this microbe on all media is characterised by its slowness. On agar (at 37° C.) no colonies are visible to the naked eye for at least two days. In cultures made directly from the spleen or blood in fatal cases the agar appears sterile to the unaided eye for from four to six days. The colonies, when developed, are small, transparent, and dew-like, and are often limited to the lower end of the tube. Broth (at 37° C.) becomes turbid, the turbidity appearing on the second or third day. After a time there is a deposit of flocculi, the broth still, however, remaining turbid. Short chains are also found in this medium. If the specific serum is added to the broth the fluid remains clear and a compact, felt-like growth takes place at the bottom of the tube in the course of weeks. On gelatin the growth is extremely scanty and slow; there is no liquefaction. It may be further noted that on media which are highly alkaline there is little or no growth. Its slow growth, the appearance on agar, and the microscopical characters will generally lead to its identification. The behaviour of the culture in question in the presence of the specific serum will confirm with certainty the diagnosis. In bacteriological literature no description of another microbe possessing the same biological characters can be found.

Causal association of the micrococcus melitensis with Malta fever.—The evidence collated above shows (a) that the micrococcus melitensis has been recovered from almost every case of Mediterranean fever which has been examined

³ Mediterranean, Malta, or Undulant Fever, 1897.

⁴ Transactions of the Epidemiological Society of London, vol. ix., p. 76.

⁵ The biological characters of these cultures were the same as those supplied to this laboratory by Hughes.

⁶ Journal of Pathology and Bacteriology, December, 1898.

⁷ Loc. cit.

⁸ Loc. cit.

⁹ Loc. cit.

¹⁰ THE LANCET, March 11th, 1899, p. 688.

¹¹ Loc. cit.

¹ Jaccored: Journal de Médecine, March 10th, 1899.

² Practitioner, September, 1887, and April, 1888; Annales de l'Institut Pasteur, 1893, vol. vii., p. 289.

bacteriologically after death and has not been found elsewhere; (b) that the micrococcus melitensis is capable of infecting monkeys, giving rise in them to a continuous fever not unlike that in man, and usually causing death, and that by special methods rabbits and guinea-pigs have also been infected; and (c) that from all these animals after death the same organism has been recovered in pure culture from the blood and organs, and after cultivation this has again

CASE 2.—On March 1st, 1898, a man, in connexion with some experiments regarding the elaboration of a method of vaccination against Malta fever, injected into his arm $\frac{1}{100}$ th of an agar tube of a seven-day growth of micrococcus melitensis.¹² On March 17th, 16 days later, febrile symptoms set in and pursued a course characteristic of this fever (vide Chart 2). It is worthy of note that, as in most bacterial infections, there was a marked increase in the number of polynuclear white cells in the blood just before the onset of fever.

CASE 3.—In February, 1899, the head attendant of the laboratory, who was constantly employed in bacteriological operations with this microbe, contracted an illness which soon assumed the typical characters of Malta fever and which was complicated by severe double orchitis. The method of infection could not be definitely traced in this case.

We shall have occasion to refer in more detail to these three cases in connexion with the question of the estimation of the agglutinating substances. We may here add that the diagnosis was confirmed in all three cases by the serum sedimentation reaction. The reaction took place in high dilutions of the serum and during the course of the fever there were considerable variations in the agglutinating power of the serum. In the first two cases the agglutinating reaction still persists a year and a half and a year respectively after convalescence.

The effect of specific serum on micrococcus melitensis.—If the above observations left any room for any doubt as to the causal relation of this organism with Malta fever, that doubt would be completely removed by the investigation through which Professor Wright established that the serum of

occasioned the disease in other animals. The final link in the chain—i.e., the proof that the micrococcus melitensis can produce Malta fever in man—has not heretofore been forthcoming. This is now furnished by two cases which have occurred as a result of inoculation with the micrococcus melitensis and by a third case the method of infection in which is not quite so definite; all these cases happened among the staff of this laboratory. They are given here in chronological order.

CASE 1.—On Sept. 17th, 1897, a man accidentally scratched himself with the needle of a syringe with which he had just

patients who are suffering from, or who have recovered from, Malta fever produces a specific agglutinating reaction on the micrococcus melitensis. This observer¹³ has published 14 observations in which this coccus was sedimented after agglutination by high dilutions of the sera of patients who were suffering from, or had recently recovered from, Mediterranean fever, and he showed that the agglutination and sedimentation which were obtained in these cases with the coccus was more marked and definite than the corresponding reaction which is obtained with the bacillus typhosus in typhoid fever. In a more recent publication,

CHART 1.

CHART 2.

injected into a horse a living growth of micrococcus melitensis.¹² He immediately sucked the minute wound, plunged the hand into a 5 per cent. solution of phenol which he had by his side, and almost at once cauterised the puncture with pure phenol. But all to no purpose, for on Oct. 2nd, 15 days later, his temperature rose and he went through a typical attack of Malta fever (vide Chart 1).

along with Semple,¹⁴ he has further shown that dead cultures of the bacteria can be employed with the same result as living cultures. During the course of our investigations we have arrived at a like conclusion. These observations have been confirmed by other observers. Aldridge¹⁵ has reported 14 cases in Malta in which this reaction was obtained, and

¹² This culture was derived from a culture which had been isolated two years previously by Hughes from the spleen of a fatal case of the disease in Malta.

¹³ THE LANCET, March 6th, 1897, p. 656.

¹⁴ Brit. Med. Jour., May 15th, 1897.

¹⁵ THE LANCET, May 21st, 1898, p. 1394: Report of the Health Department of Malta, 1898.

Elkington¹⁶ has detected agglutination of this microbe 75 times in 158 samples of blood of various febrile patients in Gibraltar. Durham¹⁷ has likewise obtained this phenomenon with the blood of rabbits and guinea-pigs which he had infected by means of intra-cerebral and intra-peritoneal inoculation. Further, the test is being daily employed in the laboratory of the Public Health Department of Malta.

Since the date of Professor Wright's original publications we have carefully investigated this reaction. It is the result of this investigation which we have now to present and which we shall preface by giving a short account of the methods employed. All observations were made microscopically by means of the sedimentation tubes (rather less than one millimetre in diameter) by Professor Wright.¹⁸ Equal quantities of the serum, diluted with normal salt solutions, and of sterile emulsions of the micrococcus were employed in all cases. The emulsions were prepared from fresh agar cultures with normal saline solution. It is obvious that the sedimentation reaction of any serum when one and the same culture is used will vary with the number of bacteria which the emulsion contains. In order to make the quantitative estimations which are here reported comparable amongst themselves the following procedure was adopted. 1. The emulsions were all prepared in exactly the same way. A uniform growth in agar of from five to seven days was obtained. This was emulsified with sterile normal saline solution, a fixed quantity of which—viz., 0.25 cubic centimetres—was used for every square centimetre of agar culture. The bacteria were then killed by heating at 60° C. for from 10 to 15 minutes and finally 0.5 per cent. carbolic acid was added. In this way emulsions containing approximately the same number of bacteria were obtained. 2. When the estimation of the agglutinins was made during the whole course of a case the same emulsion, if possible, was used throughout. If a fresh emulsion had to be substituted the sedimentation reaction of the same specimen of serum was compared in the two emulsions and any difference taken into account in recording the results. Following this method we first examined as controls more than 50 samples of blood of healthy people, among whom were 11 negroes, and 101 samples taken from patients suffering from the following diseases: enteric fever, 38 cases; malarial fevers, 20 cases; liver abscess, four cases; local suppurations, 10 cases; acute streptococcal infections, five cases; rheumatic fever, five cases; tuberculosis, five cases; secondary syphilis, six cases; cancer, two cases; dysentery, four cases; acute tonsillitis, one case; and diabetes, one case. In the course of these estimations we found that nearly all the specimens of blood examined gave a well-marked, sometimes complete, sedimentation in dilutions of 1 in 2. Many gave a faint trace in dilution of 1 in 10, but the majority did not give any reaction in that dilution. In none of the above instances did we ever observe a complete sedimentation to occur in a 10-fold dilution nor did we ever see a trace of sedimentation in a 20-fold dilution. Having thus fixed the limit of the agglutinating power of normal sera and of sera derived from patients suffering from other diseases we may proceed to consider the cases in which sedimentation above these normal limits was observed. We have notes of over 120 individuals in whom this reaction was found, and as many cases were examined on several occasions we have made several hundred observations in all. A list of these cases with dilutions in which the serum reacted is given in Table I. and Table II.

On referring to the above list it will be seen that 53 cases came under observation while still suffering from Malta fever, the remainder at varying periods after convalescence had been established. In 44 febrile cases in which the sedimentation value was quantitatively determined the average dilution for a complete reaction¹⁹ was between 600 and 700, though the limits were very wide; one serum was tested in which complete sedimentation took place in a 6000-fold dilution. Traces of agglutination occurred in these cases on the average in 1000-fold dilution. When we contrast these figures with those obtained by us in cases of typhoid fever we find that Malta fever serum sediments the micrococcus *melitensis* in considerably higher dilutions than does typhoid serum Eberth's bacillus, and, moreover, the clumps of agglutinated micrococci which are deposited are much more

compact and defined than those of the latter. Indeed, the serum reaction of Malta fever is one of the most delicate bacteriological tests with which we are acquainted and ought undoubtedly to supersede the ordinary clinical method of diagnosis, especially as it occurs comparatively early in the course of the disease, as we shall show later. By its means we have recognised cases invalided home as gonorrhoeal rheumatism, simple continued fever, debility, rheumatic fever, enteric fever, malaria, &c.

Data bearing on the early appearance of the agglutinins in the blood.—In experiments on monkeys performed by us and in those previously referred to as done by Wright and Semple it is definitely shown that the agglutinating substances may appear in the blood as early as the fifth day after inoculation of the living virus and that they are well developed by the ninth or eleventh day. The curves of the sedimentary values of the sera of a series of guinea-pigs which Durham²⁰ has figured show no rise before the fifth day after inoculation. This observer further states that the rate of development of the agglutinins varies inversely with the amount of the dose. Some experiments of Wright and Semple bear this out; for instance, in a monkey which had been inoculated with half an agar tube of the micrococcus the serum showed no reaction until the ninth day after inoculation, whereas the serum of a monkey infected with but one loopful of a culture gave a reaction on the fifth day. In the case of man, Aldridge,²¹ in the paper to which previous reference has been made, notes his failure to observe the reaction before the fifth day after the onset of the fever. In two cases which we have investigated from the onset of the disease and in which this could be determined with accuracy a complete reaction in 400-fold dilution was observed in one and in 1500-fold dilution in the other, both on the fourth day of the pyrexia. In Case 1, mentioned above, the first observation, owing to the fact that the man was absent on duty, was not made till the ninth day after the onset of fever and the twenty-third after accidental inoculation. This observation showed complete sedimentation in 800-fold dilution.

In Case 2 it will be seen from the chart that the sedimentation value of his serum had been raised before inoculation to 100 by previous treatment with sterilised cultures. This declined to 20 on the third day after inoculation, while on the eighth day after the onset of the fever there was noted a large increase in the amount of agglutinins present in the blood, the sedimentation value being 800. From these data it will be seen that in Malta fever the agglutinating substances appear in the blood comparatively early, much sooner than in the average case of typhoid fever. They therefore possess a greater diagnostic value.

The persistence of the agglutinins in the blood after recovery.—With a view of estimating the duration of the reaction after recovery we have investigated 68 cases at different periods after convalescence with the following results. In 27 individuals examined within six months after convalescence was established the average serum dilution in which sedimentation was manifest was found to be about 350-fold. In 18 cases examined between six months and a year after recovery the mean sedimentation value was approximately 250. In seven cases examined between one and two years after recovery an average value of about 100-fold dilution was determined. After two years the reaction has been obtained, in many instances, in no higher dilutions than in normal sera; eight out of 14 samples of blood taken from persons who had suffered from the illness from two to eight years previously gave no more marked reaction than normal sera, while the remaining six gave a complete or well-marked sedimentation in 10-fold dilutions or over. We have only met with one example in which the characteristic effects of the serum were apparent at a longer interval than seven years—this case, examined seven and a half years after recovery, gave a complete reaction in 20-fold dilution.

Incubation period for man.—Case 1 and Case 2 mark with accuracy the incubation period. In the former there was an interval of 15 days between the date of infection and the onset of fever, while in the latter this interval was 16 days. Bruce²² states that the disease has developed in England in persons who left Malta from 14 to 17 days previously, while Hughes²³ has observed instances of the onset of fever eight, 10, and 14 days after arrival in Malta. Two cases have come under our notice in which the

¹⁶ Reports of the Sanitary Officer of Gibraltar, 1897 and 1898.

¹⁷ Loc. cit.

¹⁸ Brit. Med. Jour., Feb. 5th, 1898.

¹⁹ In these cases traces of sedimentation occurred on the average up to 1000 fold dilution.

²⁰ Loc. cit.

²¹ Loc. cit.

²² Brit. Med. Jour., May 13th, 1889.

²³ Loc. cit.

TABLE I.—CASES OF MALTA FEVER EXAMINED DURING THE COURSE OF DISEASE.

No. of case.	—	Dilutions of serum.																						Remarks.
		10	20	30	50	70	80	100	150	200	300	400	500	600	800	1000	1200	1500	2000	4000	6000	9000	10,000	
1	Now ill.	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	+	...	X	...
2	"	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	+	...	X	...	...	...
3	"	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	+	X	...	...	...	...
4	"	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	+	X	...	...	...	...	...
5	"	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	+	X	...	...	...	...	...
6	"	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	+	...	X	...	...	...	...	...
7	"	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	+	...	X	...	...	...	...	...
8	"	...	...	...	...	...	...	...	...	...	...	...	...	...	...	+	X	...	...	...	...	...	...	...
9	"	...	...	...	...	...	...	...	...	...	...	...	...	...	...	+	...	...	...	...	...	...	...	...
10	"	...	...	...	...	...	...	...	...	...	...	...	...	...	...	+	...	...	...	...	...	...	...	...
11	"	...	...	...	...	...	...	...	...	...	...	...	...	...	+	...	...	...	X	...	...	...	...	...
12	"	...	...	...	...	...	...	...	...	...	...	...	...	...	+	...	X	...	...	...	...	...	...	...
13	"	...	...	...	...	...	...	...	...	...	...	...	...	...	+	...	...	...	...	...	...	...	...	No higher dilutions
14	"	...	...	...	...	...	...	...	...	...	...	...	...	+	...	X	...	...	...	...	...	...	...	...
15	"	...	...	...	...	...	...	...	...	...	...	...	+	...	...	...	...	X	...	...	...	...	...	...
16	"	...	...	...	...	...	...	...	...	...	...	...	+	...	X	...	...	...	...	...	...	...	...	...
17	"	...	...	...	...	...	...	...	...	...	...	...	+	...	X	...	...	...	...	...	...	...	...	...
18	"	...	...	...	...	...	...	...	...	...	...	...	+	...	...	...	...	...	...	...	...	...	...	No higher dilutions
19	"	...	...	...	...	...	...	...	...	...	+	...	...	...	X	...	...	...	...	...	...	...	...	...
20	"	...	...	...	...	...	...	...	...	...	+	...	...	...	X	...	...	...	...	...	...	...	...	...
21	"	...	...	...	...	...	...	...	...	...	+	...	X	...	...	...	...	...	...	...	...	...	...	...
22	"	...	...	...	...	...	...	...	...	+	...	...	...	X	...	...	...	...	...	...	...	...	...	...
23	"	...	...	...	...	...	...	...	...	+	...	...	...	...	...	X	...	...	...	...	...	...	...	...
24	"	...	...	...	...	...	...	...	...	+	...	...	X	...	...	...	...	...	...	...	...	...	...	...
25	"	...	...	...	...	...	...	...	...	+	...	...	...	...	...	...	...	...	...	...	...	...	...	...
26	"	...	...	...	...	...	...	+	X	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
27	"	...	...	...	...	...	...	+	...	X	...	...	...	...	...	...	...	...	...	...	...	...	...	...
28	"	...	...	...	...	...	...	+	X	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
29	"	...	...	...	...	...	...	+	X	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
30	"	...	...	...	...	...	...	+	X	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
31	"	...	...	...	...	...	...	+	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	No higher dilutions
32	"	...	...	...	...	...	...	+	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	Do.
33	"	...	...	...	...	...	...	+	...	...	X	...	...	...	...	...	...	...	...	...	...	...	...	...
34	"	...	...	...	...	...	...	+	...	...	X	...	...	...	...	...	...	...	...	...	...	...	...	...
35	"	...	...	...	...	...	...	+	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	No higher dilutions
36	"	...	...	...	...	...	...	+	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	Do.
37	"	...	...	...	...	...	...	+	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	Do.
38	"	...	...	...	...	...	+	...	X	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
39	"	...	...	...	...	...	+	X	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
40	"	...	...	...	...	...	+	X	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
41	"	...	...	...	...	...	+	...	...	X	...	...	...	...	...	...	...	...	...	...	...	...	...	...
42	"	...	...	...	...	...	+	...	X	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
43	"	...	...	...	+	...	...	...	...	...	X	...	...	...	...	...	...	...	...	...	...	...	...	...
44	"	...	...	...	+	...	...	X	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
45	"	...	...	...	+	...	...	X	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
46	"	...	...	...	+	...	...	X	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
47	"	...	+	...	X	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
48	"	...	+	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	No higher dilutions
49	"	...	+	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	Do.
50	"	...	+	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	Do.
51	"	+	...	...	X	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
52	"	+	...	...	X	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
53	"	...	...	...	...	...	...	...	...	...	+	...	...	...	...	...	...	...	...	...	...	...	...	...

+ denotes complete sedimentation. X denotes marked, but not complete, sedimentation.

first symptoms began at least from 18 to 20 days after leaving Malta. From these data it will be seen that the incubation period may vary between eight and 20 days, the usual period being probably about 15 days.

Geographical distribution.—Wright and Smith²⁴ by means of the serum sedimentation test confirm the fact that Bruce and others had long surmised—viz., that Malta fever is not confined to the Mediterranean basin. In the publication referred to these authors tabulated 10 cases invalidated from India in which the serum reaction gave a sedimentation value of an

average of about 300-fold dilution. Many of these cases at the time of examination presented the usual sequelæ of the disease. Since the date of that note 10 other examples have been observed by us in this hospital. These had been invalidated for such diseases as malaria, enteric fever, and rheumatism. It is worthy of remark that 14 out of the total of 20 such cases observed came from a comparatively small station in the Punjab—namely, Meean, Meer; the other places in India where infection has occurred are Calcutta, Subathu, and Nowshera. One man contracted the disease in Hong-Kong. This list may be further extended by the following cases

²⁴ Brit. Med. Jour., April 10th, 1897.

TABLE II.—RECOVERIES FROM MALTA FEVER WHICH HAVE SHOWN COMPLETE REACTION IN 10-FOLD DILUTION AND OVER.

No. of case.	Period after fever.		Dilutions of serum.															Remarks.	
	Year.	Month.	10	20	40	50	60	80	100	150	200	300	400	500	600	800	1000		1500
1	...	1	...	...	...	...	...	...	...	...	+	...	...	...	...	X	...	...	No higher dilutions.
2	...	1	...	...	...	...	...	...	...	...	+	...	X	...	...	...	...	...	
3	...	1	...	...	...	...	...	...	...	+	X	...	...	...	...	...	...	...	
4	...	1	...	...	...	...	...	+	...	...	X	...	...	...	...	...	...	...	
5	...	1	...	...	...	...	...	+	...	X	...	...	...	...	...	...	...	...	
6	...	1	...	...	+	...	X	...	...	...	...	...	...	...	...	...	...	...	
7	...	2	...	...	...	...	...	...	...	...	...	...	...	...	...	+	...	...	
8	...	2	...	...	...	...	...	...	...	...	+	...	...	...	...	X	...	...	
9	...	2	...	...	...	...	...	...	...	...	+	...	X	...	...	...	...	...	
10	...	2	...	...	...	...	...	...	...	...	+	...	X	...	...	...	...	...	
11	...	2	...	...	...	...	...	...	...	+	X	...	...	...	...	...	...	...	
12	...	2	...	...	...	...	...	+	...	...	...	...	X	...	...	...	...	...	
13	...	2	...	...	...	...	...	+	...	...	...	X	...	...	...	...	...	...	
14	...	2	...	...	...	+	...	...	...	...	...	...	...	...	...	...	...	No higher dilutions.	
15	...	2	...	...	...	+	...	...	...	...	...	...	...	...	...	...	...	Do.	
16	...	2	...	...	+	...	...	...	...	...	X	...	...	...	...	...	...	...	
17	...	3	...	...	...	...	...	...	...	...	+	...	...	...	...	...	X	...	
18	...	3	...	...	...	...	...	...	+	...	...	...	...	...	...	...	...	No higher dilutions.	
19	...	3	...	...	...	...	...	...	...	...	X	...	...	...	...	...	...	...	
20	...	3	...	...	...	...	...	...	...	X	...	...	...	...	...	...	...	...	
21	...	3	...	...	...	...	...	+	...	...	...	...	...	...	X	...	...	...	
22	...	3	...	...	...	...	...	+	...	...	...	...	X	...	...	...	...	...	
23	...	3	...	...	...	+	...	...	...	...	...	...	...	...	...	...	...	No higher dilutions.	
24	...	3	...	+	...	X	...	...	...	...	...	...	...	...	...	...	...	...	
25	...	3	...	...	...	X	...	...	...	...	...	...	...	...	...	...	...	...	
26	...	3	+	X	...	...	...	...	...	...	...	...	...	...	...	...	...	...	
27	...	4	...	...	...	...	...	...	...	...	...	...	+	...	...	...	X	...	
28	...	4	...	...	...	...	...	...	...	...	+	...	X	...	...	...	...	...	
29	...	4	...	...	...	...	...	...	+	...	X	...	...	...	...	...	...	...	
30	...	4	...	...	...	...	...	+	...	X	...	...	...	...	...	...	...	...	
31	...	5	...	...	...	...	+	...	X	...	...	...	...	...	...	...	...	...	
32	...	5	...	...	...	+	...	...	...	...	...	...	...	...	...	...	...	...	
33	...	6	...	...	...	...	...	...	...	...	+	...	...	...	X	...	...	...	
34	...	6	...	...	...	...	...	...	...	...	+	...	...	X	...	...	...	...	
35	...	6	...	...	...	...	...	...	...	...	+	...	...	X	...	...	...	...	
36	...	6	...	...	...	...	...	...	...	...	+	...	...	X	...	...	...	...	
37	...	6	...	...	...	...	...	...	...	...	+	...	...	X	...	...	...	...	
38	...	6	...	...	...	...	...	...	+	...	X	...	...	...	...	...	...	No higher dilutions.	
39	...	6	...	...	...	...	...	...	+	...	...	...	...	...	...	...	...	...	
40	...	6	...	...	...	...	...	...	+	...	...	X	...	...	...	...	...	...	
41	...	6	...	...	...	...	...	...	+	...	X	...	...	...	...	...	...	...	
42	...	6	...	...	...	...	...	...	+	X	...	...	...	...	...	...	...	No higher dilutions.	
43	...	6	...	...	...	+	...	...	...	...	...	...	...	...	...	...	...	Do.	
44	...	6	...	...	...	+	...	...	...	...	...	...	...	...	...	...	...	...	
45	...	6	...	+	...	...	...	X	...	...	...	...	...	...	...	...	...	...	
46	...	6	...	...	...	...	...	X	...	...	...	...	...	...	...	...	...	...	
47	...	6	...	+	...	X	...	...	...	...	...	...	...	...	...	...	...	...	
48	...	6	+	X	...	...	...	...	...	...	...	...	...	...	...	...	...	No higher dilutions.	
49	...	6	+	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	
50	...	7	...	...	...	...	...	+	...	...	...	X	...	...	...	...	...	...	
51	...	7	...	...	...	...	...	X	...	...	...	...	...	...	...	...	...	...	
52	...	8	...	...	...	...	...	+	...	...	...	X	...	...	...	...	...	...	
53	...	8	...	...	...	...	...	+	...	X	...	...	...	...	...	...	...	...	
54	...	9	...	...	...	...	...	...	...	...	+	...	...	...	...	...	...	...	
55	...	10	+	X	...	...	...	...	...	...	...	...	...	...	...	...	...	...	
56	1	...	...	...	...	...	...	+	...	...	X	...	...	...	...	...	...	...	
57	1	...	...	X	...	...	...	...	...	...	...	...	...	...	...	...	...	...	
58	1	2	...	...	...	...	...	+	...	...	X	...	...	...	...	...	...	...	
59	1	2	...	+	...	X	...	...	...	...	...	...	...	...	...	...	...	...	
60	1	4	...	...	...	...	...	...	...	...	X	...	...	...	...	...	...	...	
61	1	8	...	...	...	+	...	X	...	...	...	...	...	...	...	...	...	...	
62	1	9	+	...	...	X	...	...	...	...	...	...	...	...	...	...	...	...	
63	3	...	...	...	...	X	...	...	...	...	...	...	...	...	...	...	...	...	
64	4	...	+	...	...	X	...	...	...	...	...	...	...	...	...	...	...	...	
65	4	...	+	X	...	...	...	...	...	...	...	...	...	...	...	...	...	...	
66	5	...	...	...	...	+	...	...	X	...	...	...	...	...	...	...	...	...	
67	5	...	...	X	...	...	...	...	...	...	...	...	...	...	...	...	...	...	
68	7	...	...	+	...	X	...	...	...	...	...	...	...	...	...	...	...	...	

+ denotes complete sedimentation. X denotes marked, but not complete, sedimentation.

which have all been tested with cultures sent out originally from this laboratory. 1. Lieutenant W. Glen Liston, I.M.S., has privately reported to us a case of fever contracted in Secunderabad in the Deccan the serum from which gave a reaction in 80-fold dilution at least with the micrococcus melitensis. 2. Dr. Musser and Dr. Sailer²⁵ report the case of an army officer who appears to have contracted the disease in Puerto Rico during the Spanish-American war. Malarial

he was not confined to bed, but rested as much as was consistent with the fulfilment of his duties. The oedema lasted for about a month and was succeeded by pain and swelling in certain joints. The hips, knees, ankles, left wrist, and left temporo-maxillary joints were all attacked in turn. After the disappearance of these latter symptoms left orchitis set in and lasted for about a fortnight. Convalescence was established in March, 1898. Only during the period when the joints were affected was the temperature ever taken. At that time it was usually found to be about 102° F. in the evenings. The serum reaction was not tried during the course of the illness, but was tested by us one year and two months after convalescence. It was found to give complete sedimentation of micrococcus melitensis in 20 fold dilution and traces up to 50. From the facts stated above we may fairly conclude that this was a case of Malta fever contracted either in Plymouth or in London, although no definite source of infection can be traced.

Data bearing on the evolution of the agglutinins during the course of Malta fever.—We have seen above that the agglutinating substances which make their appearance in the blood in cases of Malta fever are specific and that their action on the micrococcus of Bruce, dead or alive, affords an easy and absolutely trustworthy method of diagnosis between this disease and other fevers which clinically simulate it. We have now to put on record the data which we have collected relating to the evolution of these substances during the course of the disease. We have already described the method which was used in making quantitative

estimations of agglutinins and have only to add that in drawing up the agglutination curves herewith presented the expression which has been adopted for the sedimenting value of each serum was the dilution in which that serum completely sedimented the bacteria, with which it was brought in contact, so as to leave the supernatant fluid clear. The data were obtained from observations taken during the course of Malta fever in man. In all 15 cases were investigated. Seven of these came under observation practically from the beginning of the fever, while the remaining eight were brought before our notice during relapses some months after the initial attack.

CASE 1.—This is Case 1 already referred to above. The fever ran a comparatively short course and presented no complications. On turning to the chart it will be seen that the agglutinating substances were developed in large quantity soon after the onset of fever and that, with the exception of a slight diminution just before a short relapse, this quantity was maintained till convalescence was established.

CASE 2.—This is Case 2, to which reference has already been made. The attack was a sharp one, although of comparatively short duration. It was complicated by severe muscular pains, which, however, did not retard convalescence. The evolution of the agglutinins in this case is of great interest. The salient features of the curve are these: (1) that only a small amount of agglutinins was developed as a result of the three injections of sterilised cultures,²⁷ which preceded the inoculation with the living culture; (2) that a well-marked diminution in the amount of agglutinins followed the inoculation of the living

organism; (3) that a slight and gradual recovery in the amount of agglutinins occurred during the period of incubation; (4) that a rapid increase in the amount of agglutinins began soon after the onset of the fever; and (5) finally, that, with the exception of a slight fall at the beginning of a recrudescence of the temperature, this large amount of agglutinins persisted till convalescence was well established.

CASE 3.—The patient was a man, aged 27 years. He had

²⁷ The quantity injected on these occasions was as follows: first injection, one-fiftieth of an agar tube (killed at 60° C.); second injection, one-twenty-fifth of an agar tube (killed at 60° C.); third injection, one-twenty-fifth of an agar tube (killed at 60° C.); and fourth injection, one-thousandth of an agar tube (living).

CHART 3.

plasmodia were frequently sought for in his blood but never found. A culture of micrococcus melitensis was agglutinated by high dilutions of the patient's blood. 3. Kretz²⁸ records the case of a physician who had contracted an obstinate fever in Ajaccio, Corsica. This continued six months. After recovery his blood serum agglutinated the micrococcus melitensis in a dilution of 300-fold. 4. The following case which has recently come under our notice evidently contracted the disease in this country. A man, aged 26 years, had not been abroad since the age of eight years. From March, 1897, till the end of September of the same year he was resident physician in a hospital in

CHART 4.

Plymouth. On Oct. 1st he assumed the duties of junior house physician in the out-patient department at St. Bartholomew's Hospital, London, where he was employed during the whole of that month. He first began to feel ill in the beginning of November, complaining of headache, loss of appetite, lassitude, and general malaise. This condition persisted more or less till December, when it became complicated by oedema of the feet. The oedema afterwards extended up the legs. No cardiac lesion could be detected and the urine was normal. Up to this period of the illness

²⁵ Proceedings of the Pathological Society of Philadelphia, Feb. 1st, 1899.

²⁸ Wiener Klinische Wochenschrift, No. 49, 1897.

CHART 5.

CHART 6.

CHART 7.

been stationed at Malta for six years but had never suffered from any fever during this period. Three days before leaving Malta he began to feel lassitude and general malaise. Pyrexia began on the voyage home. He came under our observation about the thirteenth day of the disease. The case was a severe one and presented all the symptoms of an acute septicæmia. The fever was practically continuous till death, which took place about the sixty-eighth day of the illness. During the short period of apyrexia (from Nov. 3rd to 7th) the patient was in a collapsed condition and was apparently moribund (vide Chart 3). The agglutination curve shows that throughout the course of the disease the agglutinins were present in the blood only in small quantity. The highest dilution which gave complete sedimentation was 100-fold. The micrococcus melitensis was obtained post mortem from the blood and spleen.

CASE 4.—The patient was a man, aged 27 years. He had served in Malta for four months. During this time he had suffered from an attack of "simple continued fever"; in other words, the nature of this fever had not been diagnosed. He was invalided to Netley at the end of August, 1898. He came under our observation in the third week in September, at which time he had been suffering from fever for about a week. With the exception of two very short periods of apyrexia fever was continuous till death, which took place about eight weeks after the beginning of the illness (vide Chart 4). The agglutination curve shows: (1) that the agglutinins were present in the blood in considerable quantity during the earlier period; (2) that their amount was markedly increased just before and during the two

which lasted for a week. This was succeeded by an orchitis which was accompanied, however, by little disturbance of temperature. About three weeks after the orchitis a second relapse began. This was complicated with swelling of the submaxillary glands. At the time of writing this relapse still persists. The agglutinins were developed gradually in the blood during the initial stage and had reached a large amount by the time the temperature fell (vide Chart 6). The quantity began to diminish at the beginning of the first relapse and continued to fall throughout its course. Since this the sedimentation curve has varied little up to the present.

CASE 7.—The patient was a man, aged 22 years. He had served in Malta for a year and a half. He had never suffered from any kind of fever during this period. A few days after his arrival at Netley he complained of malaise, lassitude, and loss of appetite; this was soon followed by fever. He came under our direct observation on the fourth day from the commencement of the fever. The case may be compared with that of Case 5, although it was more severe. In other words, there was an initial acute stage, which was followed by a subacute stage interrupted by frequent relapses. This condition has lasted up to the time of writing, two and a half months after its onset. The agglutinating substances, as the curve shows, were present in large quantity during the early period (vide Chart 7). The amount diminished at first rapidly, then more slowly in the later stage until it became comparatively small. At present it is maintained in this amount.

The remaining eight cases came under our notice at

CHART 8.

periods of comparative apyrexia; and (3) that these substances almost disappeared from the blood shortly before death. Pure cultures of the micrococcus melitensis were obtained from the blood and spleen.

CASE 5.—This is Case 3 of the series due to infection from cultures (p. 702) to which we have had occasion to refer above. Owing to his being on leave the patient did not come under observation till about the thirty-ninth day of the illness. The case was acute for the first week after admission to hospital; thereupon it became subacute and ultimately it assumed a chronic form. Neuralgia and severe double orchitis were present as complications in the earlier period and in the later stages severe articular pains accompanied by a return of the orchitis (vide Chart 5). A relapse supervened upon this. During the early period of observation there was a very large quantity of agglutinins present in the blood. This amount diminished markedly at the beginning of the subacute stage and continued to diminish till the chronic relapsing stage was reached. Since then they have varied but little. And now, four and a half months since the onset of the disease, the amount practically remains the same.

CASE 6.—The patient was a man, aged 26 years. He had served in Malta for eight months. The onset of his illness dates from about 18 days after leaving Malta and 10 days after his arrival in England. The case was acute at the outset. After about three weeks the temperature fell to normal. This defervescence was soon followed by a relapse

periods varying from two to six months after the onset of the disease. They had been invalided from Malta on account of this malady. They were cases of subacute or chronic fever, with periods of apyrexia of variable duration. Neuralgia and joint pains were common complications. The quantity of agglutinins present in the blood of these patients varied considerably, but was never great. In each individual case the agglutinating curve shows distinct rises and falls, more marked in some cases than in others. Two cases are given in detail as types of this class.

CASE 8.—The patient was a man, aged 21 years. While in hospital in Malta for some slight surgical affection he suffered from his first attack of fever. This lasted for about six weeks and left him very weak and anæmic. Four months after this primary attack and two months after his arrival at Netley the fever recurred. While the patient was under our observation he suffered from a well-marked subacute attack of Malta fever, with occasional periods of higher pyrexia. This was complicated with neuralgias, joint pains, and profuse perspirations. The agglutination curve shows that the agglutinins were never present in the blood in very large amount, on an average a dilution of about 500-fold causing complete sedimentation (vide Chart 8).

CASE 9.—The patient was a man, aged 27 years. Previously to being invalided home he had suffered in Malta from continued fever for seven weeks. He was admitted to Netley about two and a half months from the onset of the original attack and about the seventh day of a relapse,

which had begun on the voyage home. This relapse lasted for about three weeks and after a week of apyrexia was followed by a second relapse which was, however, less severe and of shorter duration than the first. The agglutination curve shows that the agglutinins during the period under review were present in the blood in variable but at no time in large quantities. The increase in the amount at the beginning of the apyrexial period is worthy of note (*vide* Chart 9).

Inferences which may be drawn from the above observations.—The easiest way of critically sifting the data which have been placed on record above will be to classify the cases in accordance with their clinical features, thereupon to take up each category of cases separately and inquire what is the agglutinating curve which corresponds to each particular category. From the brief sketch of the cases which have been collated above it appears that they may be divided into the following clinical groups:—1. Severe cases which end fatally within a comparatively short time. Case 3 may serve as an example of this category. 2. Severe cases which run a more prolonged course and succumb after one or two relapses. Case 4 may serve as an example of this class. 3. Sharp attacks of fever, which last for a comparatively short time, convalescence being rapidly established without any or after a slight relapse only. These cases have none of the usual complications of the subacute and chronic cases. Cases 1 and 2 are types of this group. 4. Cases which begin acutely but

inferences which Durham²⁸ has drawn from a series of observations carried out on rabbits and guinea-pigs infected by means of intra-cerebral inoculation of micrococcus melitensis. This observer showed that in these animals: (a) rapidly fatal infections do not lead to a development of agglutinins in any large quantity in the blood; (b) infections in which the animals survive a longer time but ultimately die may be divided into two groups—(1) those in which the amount of agglutinins, at one time comparatively large, diminishes greatly before death, and (2) those in which the quantity of the agglutinating substances remains comparatively high up to death; and (c) infections in which the animals survive may lead to either a great or small production of agglutinins. It will thus be seen that our observations on man coincide with those of Durham on animals in all points but one—namely, that we have no cases to correspond to those constituting Durham's class (b) (2); cases, that is, in which the agglutinins were present in comparatively large quantity at death.

Value of an estimation of the agglutinating substances as an aid to prognosis.—A cursory study of the cases which have been detailed above and the charts of which are appended will be sufficient to make it apparent that a single estimation of the agglutinins at any one time during the course of an attack of Malta fever would not yield much information as to the severity of the case nor would it serve as a guide as to its future progress. Further, even a series of observations without an accompanying knowledge of the main clinical

features of the case would not justify the observer in forming an opinion as to the probable issue of the case. On the other hand, a series of observations in conjunction with a knowledge of the clinical symptoms would afford much valuable help in arriving at a prognosis. From the data collated above and from the inferences we have drawn from these data it would appear to us that the following conclusions are justified:

1. An unfavourable prognosis would be suggested in those cases which are severe from the outset and have a persistently low agglutinating reaction. In such cases the prognosis would no doubt be already indicated by the clinical symptoms, but the fact that the agglutinating power of the blood was low throughout would materially strengthen the clinician in his opinion. 2. We should also be apprehensive of the result in grave attacks in which the agglutination reaction value rapidly falls from a high figure to almost zero. 3. On the other hand, a persistently high and rising agglutination curve sustained well into convalescence would augur an auspicious and speedy ending to the case notwithstanding the acuteness of the symptoms. 4. Finally, a

guarded prognosis would seem requisite in those cases in which the amount of agglutinins, at first large, decreases considerably, although the clinical features of the case might not give rise to any anxiety. In such instances a long illness and one complicated with relapses would be anticipated. It is evident that the estimation of the amount of agglutinins present in the blood is the only sure method of deciding whether a case belongs to one or other of the last two categories.

It is interesting and important to note that Courmont of Lyons²⁹ has arrived at similar conclusions in regard to typhoid fever. This observer investigated the evolution of the agglutinins in 52 cases of typhoid fever and also critically analysed the observations of Widal and Sicard³⁰ bearing on the same problem. His conclusions are as follows. 1. A large quantity of agglutinins present in the blood is always favourable (the larger it is the better), especially if this coincides with a remission of the temperature. 2. A small quantity of agglutinins with a rise of temperature is always unfavourable. 3. A strong agglutinating power is good at all times in the course of the disease; a low agglutinating power is usually bad, as the latter condition is found either in slight forms with a tendency to relapse or in very severe cases.

In conclusion, it is our pleasant task to have to thank Lieutenant-Colonel Webb, R.A.M.C., in charge of the medical division of this hospital, for the many opportunities

CHART 9.

which become subacute or chronic. Frequent relapses are usual. They last often for many months and are complicated with orchitis, severe neuralgias, joint pains, and profuse perspirations. They recover eventually. Cases 5, 6, 7, 8, and 9 belong to this category.

Having thus framed our clinical groups we may now inquire what is the nature of the agglutinating curve in each group. In the first group the agglutinins are present in the blood only in small quantity throughout the course of the disease, a 100-fold dilution being the highest in which complete sedimentation was observed in the only case in this category which we have had the opportunity of observing. In the second group the agglutinins may be present in large quantity in the early period of the disease but almost disappear from the blood some time before death. In the case which falls under this category at one time such a high dilution as 1300-fold gave complete sedimentation, whereas at death the highest dilution giving this reaction was 50-fold. In the third group the agglutinating substances are present in large quantity early in the disease. The amount remains about constant till convalescence is established. The average of the highest dilutions in the cases of this category was about 2000-fold. Finally, in the fourth group the agglutinins are found present in the blood in large quantity in the initial stage but fall to a much lower and variable amount during the later stages, the average which then gives complete sedimentation being about 300-fold dilution. The contrast between the evolution of these substances in Group 3 and Group 4 is especially worthy of note.

These results substantiate, as far as they go, the

²⁸ Loc. cit.

²⁹ Courmont: Sero Pronostic de la Fièvre Typhoïde, 1897, and Presse Médicale, No. 2, Jan. 5th, 1898.

³⁰ Annales de l'Institut Pasteur, May 25th, 1897, p. 403.

he has afforded us of investigating the cases under his charge.

Note.—The charts are divided into weeks. The continuous line shows the temperature, which was taken twice a day. The broken line shows the amount of agglutinins present.

A CASE OF HYDROCELE OF THE SAC OF A FEMORAL HERNIA.

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THE report of a case of this extremely rare condition under the care of Dr. F. C. H. Piggott by the house surgeon of Teignmouth Hospital, Mr. A. A. Bradburne, which was published in THE LANCET of July 15th, 1899, p. 157, forcibly reminds one of how rarities come under notice not singly but in small groups, to re-appear perhaps after a long interval. In surgical wards it is quite noticeable how groups of abdominal cases or strangulated herniæ may appear in a day and none appear again for a long time.

Three months ago I operated on a patient whose case is almost identical with that under the care of Dr. Piggott. It is within the experience of most of us that hernia in a child may be cured for a more or less prolonged period by the careful use of a well-fitting truss for two or three years, but I think that it is extremely rare to find such a condition cured in a patient of advanced years; least of all if the hernia is femoral.

My patient, a woman, aged 56 years, had been the subject of femoral hernia first about six years ago. It was then reducible, she informed me. Her medical attendant at that time ordered her to wear a truss. This she did for more than four years and it was at the end of this period that she was placed under my care. When first seen by me she had considerable pain and redness of the skin over the part, the tissues being soft and atrophied by the pressure of the truss-pad. The swelling, as usually found in femoral herniæ, along and below the line of Poupart's ligament, was quite characteristic; but the hernia was irreducible now. After a few days' rest in bed the local pain and redness passed away and she was allowed to get up, discarding the truss. Several times since then I had occasion to examine the part and found it *in statu quo antea*. About three months ago she had occasion to stand at a meeting of some kind for several hours. While standing there she felt something give way in the groin and pain almost at once set in. With some difficulty she got home and went to bed. I was sent for and on examination found that the hernial tumour was larger, distinctly painful to the touch, and irreducible. There was no vomiting, the bowels had just moved naturally, the temperature was normal, her face was not like that of a patient with intestinal obstruction, and the pain was confined to the tumour. Keeping the possibility of strangulation in mind I had the parts well fomented for several days. The pain became less but the swelling and irreducibility still persisted. I recommended operation, which was also advised by Dr. Hector Cameron, who was called in in consultation.

The operation was done on June 1st. Chloroform was administered by Dr. J. L. Carstairs. An incision was made over the tumour from Poupart's ligament downwards and outwards along its whole length. On getting down to the mass I found a large piece of omentum firmly matted together and slightly changed in colour and consistence, being yellower and firmer with less blood-supply than normal; in size, about half of a man's fist and tapering up to a neck-like process which ended in the crural canal. There was no defined sac around the mass of fat, but on its inner side where it made for the ring there was a sac also tapering towards the crural ring the wall of which was quite the commonly met with hernial sac with serous straw-coloured fluid as contents. This sac was quite separated from the abdominal cavity. Neither finger nor probe could get into the ring, the crural canal being effectively plugged by what had been the neck of the sac and the part of omentum within the canal becoming consolidated and adhering to the surrounding tissue. I removed the whole mass after ligaturing with strong catgut at the ring and anchoring this stump to Poupart's and Gimbernat's ligaments. The bleeding was stopped. The

wound was cleaned and sewn up with about 20 fishing-gut sutures. On cutting into the omentum removed there appeared a mass of recent blood-clot of the size of a large walnut. So the symptoms were explained: the truss had been worn lightly over the neck of the unreduced omental hernia or the omentum may have slipped down behind the truss. The steady pressure had obliterated the sac-neck and cured the hernia, leaving the omentum outside, which in the meantime united to the sac and the tissues in Scarpa's triangle and secured its blood-supply therefrom. The stress of prolonged standing had caused the rupture of a small vessel in the somewhat degenerated mass, forming a hæmatocele—hence the feeling of something giving way and the subsequent pain. The patient is now three months after operation taking a holiday down the Clyde.

My case seems to come under the category defined by Erichsen and quoted by Mr. Bradburne: "This disease must not be confounded with the accumulation of fluid in whatever quantity in strangulated herniæ or in hernial sacs that communicate with the peritoneal cavity. Its distinguishing feature is the accumulation of fluid in a part of the sac that has been cut off from all communication with the cavity of the peritoneum," and it is another proof of the advantage of keeping an open mind in operating on hernia, whether strangulated or not. It is best never to have any definite expectations as to what you will find, for here, more than in any surgical operation, the unexpected oftenest happens.

Glasgow.

GUAIACOL CARBONATE AND CREASOTE CARBONATE IN BRONCHITIS AND PNEUMONIA.

BY DR. R. SEIFERT.

SINCE the first articles published by Dr. Hölscher and myself¹ on the beneficial action of guaiacol carbonate (Duotal) in tuberculosis of the lungs, articles which were soon followed by those of Chaumier² on the similar action of creasote carbonate (creasotal), our experiments have been verified by many physicians of all countries. THE LANCET has also during the last few years more than once published descriptions by me of important new experiments on this subject.³ The interesting discovery has now been made that the favourable action of duotal and creasotal is not only manifest in chronic diseases of the lungs but even in a still more obvious and characteristic manner in all acute diseases of the respiratory organs. According to the published statements of the French physicians, Dr. Cassoute and Dr. Corgier, pneumonia in all its complicated forms seems to have been deprived of all danger by the new method of treatment.

At the Pediatric Congress which was held at Marseilles last year Dr. Cassoute, physician-in-chief to the Marseilles hospitals, gave a brief preliminary notice of his new method for the treatment of pulmonary affections, and it has now been described in detail by Dr. Corgier in a pamphlet entitled, "The Treatment of Acute Broncho-Pulmonary Affections by Creasotal" (Montpellier, 1899). Dr. Cassoute's new method is distinguished by the certainty and rapidity of its curative effects not less than by its simplicity. All other medication with the exception of cardiac tonics, when such are indicated, is omitted; so also are all the usual adjuvants in the treatment of these cases. At the very most packs are occasionally employed. The method consists in the continuous administration of fairly large doses of creasotal. In most cases a typical fall of temperature occurred during the first 24 hours of treatment, and if the creasotal was continued for a sufficiently long period of time the apyrexia was permanent. The temperature curve rose again, however, when the drug was discontinued before the auscultatory signs disappeared. Relapses and sequelæ, so frequently seen under other methods, were entirely absent. It is remarkable that even the most modern and exhaustive text-books, whilst they describe all possible methods of treatment of pneumonia and broncho-pneumonia, say nothing of the antiseptic

¹ Berliner Klinische Wochenschrift, 1891.

² THE LANCET, Jan. 22nd, 1898, p. 222.

³ THE LANCET, Nov. 14th, 1896, p. 1373, and April 2nd, 1898, p. 960.