munity, then comes the return in increased knowledge and reputation. The good a man receives from a hospital appointment depends on himself; it must be looked on as a special opportunity, and the result is in proportion to the holder's ability to use it. But the point I wish to emphasize is this—that, so far as the hospital is concerned, it must not be used as a direct means of gaining practice or fees. To do so under existing conditions would be against the true principles of charity and of fairness to other men. The hospital physician or surgeon must assume that every one whom he sees there is a fit object for gratuitous help, and he must accept all that follows from this. In the large hospitals there is no temptation to transgress, but with small hospitals and in small towns there is often the opportunity of directly increasing private practice among those who use the hospital. The attitude of the holders of honorary appointments to this should be clear and definite. They must have nothing to do with hospital cases as their private patients afterwards. This does not apply, of course, to such as they may have attended before going to hospital, for the fact of being a hospital patient in no way alters a previous relation.

The hospital physician or surgeon does public work for the community at large. He uses buildings, food, medicine, appliances, and resident staff provided by the public. His work, therefore, should be in the public eye. Owing to the conditions of medical work, he should treat his professional brethren as that eye. His practice should be fully open to them, every qualified practitioner should be a welcome visitor with him in his wards and a spectator at his operations, so far as this can be consistently with the safety of his patients. If they come to his hospital for instruction he should do his best to afford it. And he should betimes let others share in the results of his experience.

If a practitioner sends in a case for operation, his recommendation should be taken as proof of the fitness of the patient for hospital treatment. Notice should also be sent to him of the date and time of operation so as to let him attend if he wishes. Such, I take it, is the present position and responsibility of the hospital physician and surgeon. I am inclined to think, however, that a time is approaching when there may be a demand for a readjustment of the relations of the hospital surgeon to the public. Possibly the physician may calmly await, in his position of dignity and honour, a euthanasia due to his gradual supersession by the ever-invading surgeon. But with the latter it is different. Year by year larger demands are made on him. Take our own hospital: Twenty-five years ago the operations numbered 140; last year they amounted to 476. The great advances in surgery in that period have brought within possibilities of remedy by operation whole classes of disease which were formerly considered to be outside the field of surgery. And the end is not yet. Many ailments thought at present to be purely medical will at no distant date be remediable by the surgeon. It seemed at one time that increased safety in operating due to improved technique might result in surgery becoming less specialized, but on the contrary the tendency has been in the opposite direction. To do modern surgery well a man must have an intimate knowledge of the position, consistence, and appearance of all internal organs in health and disease. His knowledge must be such as to avail him for immediate decision. He must be so drilled that he carries out an elaborate technique as a matter of routine and yet with intelligence. Consequently the present tendency seems to be more and more to leave this work in the hands of those who devote themselves to it. Not only so, but to have it done in places (hospitals and homes) organized for the purpose, and less and less in private houses. I have had a rough estimate made of what the hospital surgeon gives annually to the public. I have had the operation tables of five provincial hospitals analysed. They were taken at random, but they represent North, South, East, West, and Midland districts. A value was assigned to each operation—what I take to be the irreducible minimum. This calculation makes each surgeon of these hospitals give to the public operation work of an average value of £630 annually, and each assistant surgeon £270. This is in addition to and independent of what he gives in ward work and in the out-patient departments. In return a hospital surgeon receives a position in which much of the surgery of his district may come to him; he has the opportunity of making a large and valuable experience and he has work of the most intense human interest. But the questions may arise, Is not the price too heavy? Is he not bearing, and in the future likely to

bear in an increased degree, what should be one of the burdens of the community at large? The whole subject is an involved one, and mixed up with the other question: What shall be the future of our charitable hospitals? I do not propose to offer you any suggestions or schemes for changes. The time has not yet come to do more than discuss the subject in its various bearings and relations to broad principles. But I venture to commend it to your earnest consideration.

OBSERVATIONS ON THE OPSONIC POWER OF PEOPLE SUFFERING FROM TUBERCULOSIS.

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In order to kill bacteria within an organism it is necessary to employ some substance which enters into destructive combination with the bacteria. There are two possible methods only; the one which has been chiefly used is that of introducing antiseptics into the organism; the other method is to rely upon the action of protective substances produced by the organism, and to attempt to increase the production of such substances by the injection of bacterial vaccines. The former method has been tried without success in the case of tuberculosis; the latter method remains to be tried.

Protective substances are substances which enter into destructive chemical combination with bacteria, and are found in all human blood. As we are considering tuberculosis, we are only concerned with those which enter into combination with the tubercle bacilli, and these, according to Ehrlich's nomenclature, are called tuberculotropic substances. Of such tuberculotropic substances we know two—(1) agglutinins, (2) opsonins.

(1) Agglutinins are substances which enter into chemical combination with tubercle bacilli in such a way as to immobilize and conglomerate them.

(2) Opsonins (*Opsono*, I cater for, I prepare victuals for) are substances which modify tubercle bacilli in a manner which renders them an easy prey for the phagocytes.

The term "opsonin" was first used by Dr. Wright, and the following observations have been made by him and Captain Douglas:

(1) Opsonins are contained in the blood fluids—serum or plasma.

(2) They lose their power when heated to 60 to 65° C. for 10 to 15 minutes.

(3) They exercise their influence by effecting a modification in the bacilli, and not by exerting a direct stimulating effect upon the phagocytes.

The method employed in estimating the opsonins or opsonic power in the blood is that described by Dr. Wright, of which I will give a brief outline.

Equal quantities of the patient's serum, an emulsion of tubercle bacilli, and blood corpuscles (washed in a solution of $\frac{1}{2}$ per cent. sodium citrate in normal salt), are taken in a capillary pipette and incubated together for 20 minutes, after which films are made of the mixture and stained in the ordinary way for tubercle bacilli; the average number of tubercle bacilli ingested by each polynuclear white corpuscle is calculated, and the figure thus obtained is called the phagocytic index.

It has been found that the source of the corpuscles used makes no difference to the result; they can be taken from the patient or from some healthy person, as is shown by the following results, which have been proved several times:

Serum of a healthy person + an emulsion of tubercle bacilli + the corpuscles of a healthy person give a phagocytic index = x.

Serum of a healthy person + an emulsion of tubercle bacilli + the corpuscles of a diseased person give a phagocytic index = x.

Serum of a diseased person + an emulsion of tubercle bacilli + the corpuscles of a healthy person give a phagocytic index = y.

Serum of a diseased person + an emulsion of tubercle bacilli + the corpuscles of a diseased person give a phagocytic index = y.

So we see that a very inferior position is assigned to the polynuclear white cells in the work of phagocytosis; in fact, their use may be compared to the use of culture media in bactericidal experiments to estimate the number of bacteria which have been destroyed.

Having obtained this phagocytic index, we need some standard for comparison, and at present the phagocytic index of a healthy person is taken and comparison made to that as

The Opsonic Power of the Blood of People Suffering from Various Tuberculous Affections.

Case No.	Initial.	Age.	Clinical Description.	Opsonic Power.
1	A. C.	40	Lupus for 10 years, nearly cured after x-ray treatment	1.90
2	R.	5	Tuberculous disease of skin of face since birth	0.70
3	L. G.	21	Lupus of nose for 9 years, improving slightly	0.70
4	W. P.	21	Lupus of nose for 14 years, improving slightly	0.65
5	H.	36	Tuberculous disease of skin of face for 18 years	0.75
6	E. C.	18	Tuberculous glands in neck for 8 months	0.40
7	M. C.	5	Acute tuberculous disease of hip-joint, duration 1 month	1.20
8	N. M.	7	Tuberculous glands in neck and axilla; tuberculous abscesses in various parts of the body	2.40
9	J. P.	13	General tuberculosis	1.20
10	H. C.	39	Tuberculosis of lungs, testicle, and several bones	1.30
11	G. S.	8	Acute tuberculous disease of knee-joint	1.30
12	F. S.	10	Acute tuberculous disease of kidney and bladder	1.50
13	C. B.	18	Tuberculous caries of spine with abscesses for many years	0.80
14	E. E.	13	Chronic tuberculous disease of hip-joint, duration 13 months	0.65
15	J. W.	25	Chronic tuberculous disease of hip-joint, duration 8 months	0.30
16	K. D.	8	Psoas abscess, duration 18 months	0.40
17	W. H.	18	Psoas abscess, duration 2 years	0.80
18	A. N.	8	Chronic tuberculous disease of hip-joint, duration 4 years	0.80
19	E. E.	68	Tuberculous disease of elbow-joint, duration 6 months	0.50
20	E. L.	26	Tuberculous disease of skin of arm and leg, tuberculous disease of bones of extremities, duration 24 years	0.70
21	E. J.	4	Acute tuberculous disease of ankle-joint	0.80
22	W. C.	18	Phthisis, duration 3 months. Family history, 1 sister died of phthisis	1.50
23	H. T.	21	Phthisis, duration 6 months. No family history of phthisis	1.70
24	E. C.	42	Phthisis, duration 4 months. No family history of phthisis	1.50
25	G. S.	28	Phthisis, duration 9 months. No family history of phthisis	1.60
26	J. Mc.	22	Phthisis, duration 2 years. Family history, father and 2 sisters died of phthisis	1.50
27	J. W.	27	Phthisis, duration 10 weeks. No family history of phthisis	1.40
28	E. W.	21	Phthisis, duration 6 months. No family history of phthisis	1.70
29	M. M.	26	Phthisis, duration 7 weeks. No family history of phthisis	1.20
30	J. S.	29	Phthisis, duration 3 years. Family history, father and 1 sister died of phthisis	0.80
31	E. W.	39	Very advanced phthisis, duration 3 years. Family history, 1 brother and 1 sister died of phthisis	0.90
32	H. K.	25	Phthisis, duration 6 weeks. No family history of phthisis	1.00
33	B. C.	18	Phthisis, duration 10 weeks. No family history of phthisis	2.10
34	H. B.	23	Acute tuberculous consolidation of lung, duration 1 month	1.50
35	J. F.	16	Phthisis, duration 11 months; acute pleurisy, 10 days	1.90
36	F. N.	13	Phthisis, duration 3 months. No family history of phthisis	0.70
37	J. S.	33	Phthisis, duration 6 months. Family history, 1 sister died of phthisis	1.10
38	J. M.	33	Phthisis and tuberculous laryngitis, duration 6 months. No family history of phthisis	1.60
39	F. S.	38	Phthisis, duration 3 months. Family history, 1 brother died of phthisis	1.90
40	A. M.	35	Very advanced phthisis, duration 5 years. Family history, father and 1 sister died of phthisis	0.90
41	J. C.	45	Very advanced phthisis, with nephritis, duration 1 month. Family history, 1 brother died of phthisis	0.30
42	W. C.	54	Phthisis, duration 3 months. No family history of phthisis	0.80
43	G. C.	30	Phthisis, duration 18 months. No family history of phthisis	1.90
44	R. W.	18	Phthisis, duration 2 years. Family history, 1 brother died of phthisis	1.60
45	J. B.	50	Phthisis, duration 2 years. No family history of phthisis	1.90
46	J. J.	41	Phthisis, duration 1 year. Family history, father died of phthisis	1.20
47	T. W.	36	Phthisis, duration 16 months. Family history, mother died of phthisis	2.10
48	H. P.	17	Phthisis, duration 18 months. Family history, 1 sister died of phthisis	2.10
49	A. S.	17	Phthisis, duration 2 months. No family history of phthisis	2.40
50	W. W.	36	Phthisis, duration 2 years. No family history of phthisis	1.60
51	R. H.	21	Phthisis, duration 1 year. No family history of phthisis	1.40
52	M. J.	34	Phthisis, duration 7 months. No family history of phthisis	2.10
53	A. H.	19	Phthisis, duration 3 years. No family history of phthisis	2.60
54	W. N.	39	Very advanced phthisis, duration 1 year. Family history, 1 aunt and 2 uncles with phthisis	0.60

unity, the figure thus obtained being called the opsonic index or opsonic power of the serum tested.

The use of this standard is justified by the following facts:

1. The opsonic power is approximately the same in all healthy people.

2. The opsonic power of healthy people varies very slightly, or not at all, from day to day.

As it is found that the opsonic power decreases gradually after the serum has been withdrawn from the body, it is necessary that the standard serum and the serum to be tested should be withdrawn within a few hours of the same time; in all my experiments the blood to be examined was withdrawn within a few hours of the withdrawal of my own, which has been used in every case as the standard.

By these preliminary statements I hope that I have cleared the way for the subject-matter proper of my paper.

Before examining the results given by these observations, I will state the general conclusions arrived at by Dr. Wright, that an increased opsonic power is to be expected in those cases where there has been an active response, on the part of the machinery of immunization, to the stimulus of infection; and a decreased opsonic power is to be expected in individuals in whom there is an inherent deficiency in opsonic power or in whom this machinery is becoming exhausted.

If now we look at the list of cases we see that 1 to 5 are patients suffering from tuberculosis of the skin; of these, four show decreased and one an increased opsonic power. The decrease in such cases I attribute to an antecedent deficiency, not to exhausted machinery of immunization, and for the following reasons:

1. The constitutional symptoms are very slight.

2. There can be little absorption of toxins from the lesions, and little strain therefore thrown upon the machinery of immunization.

3. Some antecedent lack of resistance may be supposed when the skin, usually such a resistant structure, is invaded by tubercle bacilli.

Case 1, a patient who has been cured by the x-ray treatment, shows an increased opsonic power which may be explained by the fact that the x rays determine a greater flow of blood to the part, and so more toxin is absorbed, which stimulates the protective mechanism.

Cases 6 to 21 are examples of surgical tuberculosis. Applying the same rule to them, it is difficult to see why Case 6—a girl suffering from tuberculous glands—should have such a low opsonic power; there should be absorption of toxins from the glands, and, the patient's general condition being good, the machinery of immunization should be able to respond to the stimulus received. However, it is always found that the opsonic power of people suffering from tuberculous glands is low. Cases 7 to 12 show an opsonic power above 1, and these were in an acute stage of the disease at the time of observation; in such cases we expect to get a greater absorption of toxins, and a greater stimulus to the production of protective substances, than in the chronic cases of tuberculosis exemplified by Cases 13 to 30. The low power in these cases may be due to either exhaustion of the organism by previous acute attacks or antecedent deficiency. Case 13, a girl who has had spinal caries for many years, with repeated abscesses, I consider to be an example of the former cause; Case 19, a man of 68, who has had tuberculosis of his elbow following a slight injury, I consider to be an example of the latter cause.

Case 21 a small child with acute disease of her ankle, is a case in which the machinery of immunization is overtaxed and exhausted, and so the opsonic power is low.

Cases 22 to 54 are cases of pulmonary tuberculosis; out of these thirty-three cases, twenty-five have increased, seven decreased, and one normal opsonic power. These cases have been taken at random in several hospitals, and include patients in all stages of the disease, so that we can say that the opsonic power of the majority of patients suffering from pulmonary tuberculosis is higher than normal; and the explanation can be found in the great absorption of toxins from the lungs which are so plentifully supplied with blood. In those patients who have a low opsonic power we find as a rule very advanced disease and exhausted resistance; for example, Cases 31, 40, 41, and 54, who had low opsonic power, have all died since observation; Case 30 is a woman to whom this does not apply, as she has not very advanced disease, and yet her opsonic power is low, the only point noticeable in her case being a strong family history of phthisis. This raises the question of the influence of a family strain on the opsonic power; and while it may be true

that the hereditary taint is shown by a low opsonic power which predisposes to infection, I have been unable to get any evidence of this.

Looking at these figures, it would appear that as a general rule a low opsonic power in people suffering from pulmonary tuberculosis indicates a grave prognosis, but there are exceptions to this which the following observations assist to explain.

The Variations in the Opsonic Power of People Suffering from Tuberculosis.

I have stated that the opsonic power of healthy people varies very slightly or not at all from day to day; but when we come to examine the opsonic power of people suffering from tuberculosis, we find large variations during the course of the disease. A convenient method of expressing these variations is in the form of a curve drawn on an ordinary temperature chart, the base line or unity being taken at the line of normal temperature and each degree being made to represent two decimal places in measuring the opsonic power. This is an arbitrary arrangement, but it gives a clear view of the changes in the opsonic power. If we examine the curves thus obtained we see that they obey no obvious rule. It might be thought that as a patient improved his opsonic power would steadily rise; but this is not the case. At first I could trace no method in the curves; and the theory which I shall now put forward to try and explain them is open to criticism.

Chart 1.—P. S., a boy, aged 10, suffering from tuberculosis of left kidney, bladder, and left epididymis, was inoculated with $\frac{1}{100}$ of a milligram of tuberculin on November 22nd; the opsonic power fell from 1.5 to 0.2, and the negative phase lasted for ten days. After the second inoculation, on December 2nd, the negative phase was not so marked, and the positive phase was fully developed on December 5th; on December 9th he was inoculated again, and the opsonic power fell, until on December 15th it was only 0.5. This prolonged and marked negative phase I take as evidence that this inoculation followed too quickly after the previous one, although the same small dose was given; however, the opsonic power rose again, until on the 20th it was 1.8. A fourth inoculation was given on December 22nd, when the opsonic power was 1.3; subsequent to this inoculation the opsonic power rose to 2.5; on December 23rd, and then fell to 0.8 on December 28th. I do not look upon this rise to 2.5 as a positive phase following a very short negative phase, but I regard it as a phenomenon which is sometimes seen, and which I cannot explain—a rise immediately following the inoculation and preceding the negative phase.

It is necessary first to understand what happens after the inoculation of a bacterial vaccine, such as tuberculin. The following points have been demonstrated by Dr. Wright and others:

1. Subsequent to the inoculation there is a negative phase during which the opsonic power is decreased.

Chart 2.—J. C., a man, aged 45, suffering from phthisis, with pleural effusion, nephritis, and ulceration of the intestine. The first inoculation of $\frac{1}{100}$ of a milligram of tuberculin was given on November 28th; the negative phase was presumably too short to note, and the opsonic power rose from 0.3 before inoculation to 1.5 on December 1st. The second inoculation was given on December 2nd, and the subsequent negative phase, though slight, lasted long enough to be noted, and was followed by a rise to 1.7 on the 8th. The third inoculation was given on December 9th, and was succeeded by a much more marked negative phase, lasting three days, the opsonic power falling to 0.9, which again I take as evidence that the inoculation followed too soon after the previous one. On December 13th and 14th the opsonic power was 1.9 and 1.8, but on December 15th the patient had several severe haemorrhages from the bowel, and became very collapsed, the opsonic power at the same time falling to 0.4. Although on December 17th the opsonic power was 1.7, on the 18th it was only 0.8, and the patient never rallied, but died on December 19th.

2. This negative phase is succeeded by a positive phase during which the opsonic power is increased.
3. In the case of an inoculation which is successful the opsonic power is permanently raised, and the curve thus obtained does not fall to its original base line, but is maintained at a higher level.

Chart 3.—T. H., aged 45, suffering from tuberculous disease of the left epididymis. On December 13th he was inoculated with $\frac{1}{100}$ of a milligram of tuberculin, subsequent to which his opsonic power rose from 1 to 1.4 before the negative phase was established, which I take to be a similar phenomenon to the one already noted. After the second inoculation on December 19th the negative and positive phases followed in the ordinary way.

4. The same phases occur after every inoculation; but the ultimate result depends upon the correct interspacing of appropriate doses of the vaccine. The effect of a second inoculation, given when the positive phase following the first inoculation is at its height, will be to raise the opsonic power to a still higher level, but the effect of a second inoculation, given during the negative phase, will be to reduce still further the opsonic power already reduced by the first inoculation. In both cases the effect of the successive inoculations is cumulative; in the former case in the direction of increased resistance, in the latter case in the direction of decreased resistance to bacterial invasion.

These points are illustrated by inoculation into a healthy organism; in the case of diseased organisms, where the immunizing machinery is hampered by having to deal with cells already degenerate, the effect of inoculation varies very

much. Some of these variations are illustrated by the accompanying cases and charts:

These few examples will help to show how the usual succession of events after inoculation may be modified in people suffering from tuberculosis.

If now we turn from these patients who have been artificially inoculated to those patients who are inoculating themselves during the ordinary course of the disease, we see evidence in a large number of cases that the curves obtained by charting the opsonic power are made up of a succession of negative and positive phases following auto-inoculation.

Chart 4.—F. S., aged 38. History of phthisis for three months; advanced disease at the apex of the right lung. The patient developed signs of pneumothorax on November 24th, and became very collapsed; while the opsonic power fell from 1.9 to 0.6, showing either a sudden absorption of toxins or a failure of the immunizing machinery; from this time the patient gradually improved, and the opsonic power rose to 1.5 on December 5th, after which it fell again. Here we have evidence of auto-inoculation followed by a short negative phase and gradual positive phase; the final fall we may look upon as another negative phase, the disease at the time being still active.

The theory thus illustrated becomes explicable if we remember that these variations express the cumulative effects of auto-inoculation, and these, where the immunizing machinery is strong, will tend to improve the condition of the patient, but, where the machinery is weak and easily overtaxed, will tend to make the condition worse.

From these charts it is seen that the variations in the opsonic power of people suffering from tuberculosis do not follow a simple rule or admit of an easy explanation. The explanation which I have attempted to give of some of the charts is imperfect, but the factors which produce these variations appear so complicated that it is almost

Chart 5.—F. M., a girl aged 13, admitted to hospital on November 24th, suffering from phthisis. On November 26th her opsonic power was 0.7, and from that day it gradually rose, until on December 10th it was 1.5; this we may take as a positive phase following a previous inoculation. On December 14th the disease flared up again, as was shown by the raised temperature and more cough, and on the 17th the opsonic power had fallen to 0.8; this was clearly a negative phase following auto-inoculation, and was succeeded by a positive phase, the opsonic power rising to 2.0, accompanied by marked improvement in the condition of the patient.

Chart 6.—J. F., aged 35. History of phthisis for one year, acute pleurisy for ten days. In this case the opsonic power gradually fell from 1.9 on November 26th to 0.9 on December 12th, the patient meanwhile steadily improving; the improvement continued, and the opsonic power rose to 1.6 on December 17th and 1.7 on December 21st, a fall and rise which correspond to negative and positive phases following auto-inoculation during the acute attack of pleurisy.

T.	99.6	Opsonic power	1.2
T.	101.0	"	1.3
T.	97.4	"	1.2
T.	100.4	"	1.1

With regard to the main variations I have not attempted to found any elaborate theory on them, seeing that with our present knowledge observations on the opsonic power are mainly of use as guides to the treatment of tuberculosis by inoculations of tuberculin.

In this connexion I wish to bring forward two points which I consider especially important.

First, I wish to lay great stress on the importance of frequent examinations of the opsonic power in all cases which are under treatment by inoculation. It is often stated

Chart 7.—J. S., aged 33. History of phthisis for six months. On looking at the chart we see first a negative phase becoming most marked on November 30th, when the opsonic power was only 0.4, then a positive phase up to December 17th, and this again followed by a less-marked negative phase and finally another positive phase. During the time of observation the patient remained in much the same condition.

during the negative phase, evil and not good will result. In every case, therefore, the blood should, if possible, be properly examined, the dose of tuberculin and the frequency of the inoculation being based upon the results obtained from these examinations; in those cases where examinations cannot be made, a very small dose must be given and the inoculation repeated in ten days, the future course to be pursued then depending upon the patient's clinical condition

Chart 8.—M. C., aged 22. History of phthisis for two years. From November 16th to December 8th the patient improved very little, but after this time he began to progress rapidly, and the chart shows that this rapid improvement was coincident with the beginning of greater variations in the opsonic power.

impossible for me to arrive at more definite conclusions.

To one other point I will allude, namely, the question whether there is any daily variation in opsonic power corresponding to the daily variation in temperature. I give below readings of temperature and opsonic power taken simultaneously twice on the same day, and it will be seen that the difference is very slight:

T.	98.6	Opsonic power	1.5
T.	101.0	"	1.6
T.	97.4	"	1.5
T.	99.6	"	1.5

that a small dose of tuberculin given at regular intervals of seven or ten days is sufficient to give satisfactory results without any examination of the opsonic power; in some cases this is so, and I admit it is often impossible to get the requisite examinations made; but I would point out that the negative phase after a small dose of tuberculin may last more than ten days, and from clinical symptoms and signs we get no evidence of this. If in such a case a second inoculation is given

Chart 9.—J., aged 27. History of phthisis for ten weeks. The patient's condition remained stationary until December 5th, after which time it began to improve rapidly, and again we see this improvement coincident with greater variations in the opsonic power.

Secondly, as to the choice of patients for the treatment: the most favourable cases are those of surgical tuberculosis with a low opsonic power which can be raised by inoculation; such patients often show well-marked clinical improvement. At present it is doubtful to what extent patients with a high opsonic power benefit from the treatment. They may be supposed to derive benefit in the following manner: in chronic tuberculosis with a high opsonic power and no clinical improvement, if the patient be inoculated the machinery of immunization is set in motion by the extra stimulus and we get an increased variation in the opsonic power, which, as we have seen in some of the cases quoted, is often coincident with clinical improvement. Unfortunately, however, we have also seen that it may be coincident with the beginning of a worse condition, so that in undertaking the inoculation of a patient with a high opsonic power there is the risk for us to face. Finally, in my opinion the use of the inoculation of tuberculin is to raise the opsonic power, and the effect produced by this raised opsonic power may perhaps be compared to the effect of fresh air, which cannot be called a specific for tuberculosis, but exerts its influence by placing the patient in the best position for combating the disease.

SUMMARY.

I will now recapitulate the chief points which I have discussed:

1. Opsonic power of healthy people is nearly the same.
2. Opsonic power of healthy people does not vary from day to day.
3. Opsonic power of people suffering from tuberculosis may be either high or low.

A low power is due to (a) an inherent deficiency, or (b) exhaustion of the machinery of immunization.

A high power is due to an active response by the machinery of immunization to the stimulus of infection.

4. Opsonic power of people suffering from tuberculosis varies from day to day, and the curve thus produced shows in many cases negative and positive phases following auto-inoculation.

I feel that these results are in themselves small, but my excuse for thus putting them together must be my firm conviction that in this direction, and by some such methods as I have described, a very important advance is to be made in modern therapeutics.

Charts 10 and 11.—H. B., aged 23, suffering from tuberculous consolidation of the upper lobe of the right lung; history of illness for only one month. From November 8th to 22nd the opsonic power did not vary much, although the patient's condition became gradually worse. On November 28th the opsonic power had dropped to 0.2, after which it rose to 0.4 on December 1st. In this rise and fall we see negative and positive phases following absorption of toxins, the effect of which was accumulating during the period from November 8th to November 22nd. Immediately succeeding this we see another negative phase and another positive phase, the opsonic power rising to 0.4 and the patient's condition remaining stationary. On December 28th the patient's condition was worse, and, coinciding with this decline, there was a fall in the opsonic power to 0.2, pointing to a greater absorption of toxins. This patient's chart illustrates a point which appears in many cases. For a long time the opsonic power may remain nearly stationary, and then begin to show the wide variations which I call negative and positive phases, the beginning of variations nearly always being associated with some change in the progress of the patient either for better or for worse. The two previous cases illustrate this point.

Chart 12.—B. C., aged 18. History of phthisis for ten weeks. In this case the patient was getting rapidly worse up to November 20th, after which time his condition remained almost stationary or perhaps slightly improved; here we see sharp variations up to November 20th, followed by a gradual fall.

In conclusion I wish to say that I am indebted to Dr. Wright for much invaluable advice in the prosecution of this research.

THE INCIDENCE OF THE HAIR'S GREYNESS.

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In three previous communications on cancer I have been most particular to divide the subject into its genesis, incidence, and spread. In them I also stated that genesis and points of incidence must be closely allied in the etiology of the disease; and, further, the nature of that alliance seems to be neurotrophic. The following observations make one believe the same alliance exists between the genesis and incidence of the hair's greyness. The incidence of the hair's greyness is to be observed in two types of cases.

Injury or Disease of Nerves.

In this type are included the incidence of greyness due to injury of nerves, neuralgia, neuritis, and also the greyness which occurs in those diseases at present presumed to be of neurotrophic origin, such as the incidence of grey hair seen in cases of leucoderma, and in the patches of grey hair which occur on parts which have been temporarily bald from some cause or other. In this class of case I have seen a small bald patch result from injury to the great occipital nerve, and the hair which subsequently grew on the patch was grey; whether the greyness was permanent or not I am not in a position to say.

With regard to the neurotrophic diseases in which greyness of the hair occurs, I have seen in separate cases of leucoderma a patch occur in the right parietal maximum point described by Dr. Head (see No. 9 in Fig. 1, and the right occipital maximum point described by Dr. Head, see No. 5 in Fig. 1; compare Fig. 2).

Infantile or Congenital Greyness.

The second type of case is that in which the incidence of greyness is presumably the natural greyness seen in young people and the middle-aged. Although I place this class separate from the first, I venture to believe it is connected with neurotrophic disturbance.

On the scalp and face patches of greyness are seen which have existed as far back into early life as the individual and his relations can remember; and therefore cannot be considered as the result of increasing years. In this class of greyness there may be only one patch or many patches. Sometimes these patches are unilateral, sometimes bilateral. I have never seen general diffuse greyness in this class of case. The patches are, in my experience, most obvious in boys and men, but they occur in girls and women. The causal factor to this form of greyness may be congenital, but the facts that many babies are either born bald or if born with a few hairs, they soon shed them, make it difficult to be sure of its congenital nature, hence it would be less open to criticism were its causal factor described as infantile.

Curiously enough the situations of these patches are usually