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 0.6, after which it rose to 1.5 on December 5th. 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 2.4 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 0.6, 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.

By G. LENTHAL CHEATLE, C.B., F.R.O.S.,

Surgeon to King's College Hospital, Italian Hospital, and the Hospital for Epilepsy and Paralysis, Malaga Vale.

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

on the maximum points described by Dr. Henry Head.¹ The maximum points on which the incidences of this form of greyness are most common are the upper mid-orbital and the occipital (Nos. 1 and 9 respectively in Fig. 1; compare Fig. 2). In this form of greyness there are other points of incidence which do not correspond to the maximum points described by Dr. Head, but the sites of their occurrences are constant. One of these constant points is $1\frac{1}{2}$ in. above the pinna of the ear (see No. 4 in Fig. 1). This constant point of incidence is

Fig. 1.

too low for vertical or parietal maximum points; Captain Pinch, I.M.S., has seen a family in which the father and four sons had each a grey patch on this point. Another constant point is seen in No. 7 in Fig. 1, situated in the moustache at a point which corresponds to the position of the lateral incisor and canine teeth. It will be seen that the point is too internal for Dr. Head's maximum point for the naso-labial region (see Fig. 2).

Fig. 2.

In this class I have seen in a boy a grey patch in the lower mid-orbital maximum point, and not the upper (see No. 3 in Fig. 1; compare with Fig. 2), and a grey patch in a man on the fronto-nasal point (see No. 2, Fig. 1, and compare Fig. 2). I may mention under this class of case that I have seen bald patches on one side of the face which existed as far back as the man could remember; the sites of baldness were on Nos. 1 and 3 in Fig. 1, and the naso-labial maximum point (compare with Fig. 2).

Greyness of Age.

B and C. These types I classify together because they are popularly recognized as a natural consequence of age. I will describe the rarer condition (B) first. Here the incidence of greyness occurs in patches which are preliminary to a state of general diffuse and finally complete greyness. These precedent patches of greyness are also common on the maximum points described by Dr. Head, and the commonest points on which they are seen are the occipital and mental, and are often symmetrical. The most marked case I have seen, and which first called my attention to the matter, was pointed out to me in December, 1903, by Sir Victor Horsley. In this case the occipital and mental maximum points on each side were practically the only grey hairs visible, but now the greyness has become more general (see Nos. 5 and 8 in Fig. 1; compare with Fig. 2). I have seen cases begin to "go grey" on the point which is constantly appearing grey in Class A, namely, No. 4 in Fig. 1, which I have indicated does not coincide with any of Dr. Head's maximum points. C, the second of the two forms of greyness acquired as a consequence of age, is by far the most common of any type I can describe. Its incidence is characterized by scattered grey hairs usually

in the region of the temples, the greyness then gradually becomes diffused and ends in total greyness.

Curiously enough, I have seen a woman whose hair became very grey during middle age, except on the right upper mid-orbital maximum point, on which the hair remained the same brown colour of girlhood and early womanhood (see No. 1 in Fig. 1; compare with Fig. 2). I have not observed a case in which emotion or shock suddenly induced greyness, although the existence of such cases is recognized.

Finally, I have seen two cases in which the beard was grey in the area of distribution of the mental nerves of the two-fifths and so sharply limited to those areas that it is difficult to explain it by saying it was mere coincidence.

The points of incidence in certain forms of greyness may not be of intrinsic importance, but the facts form links in the chain of evidence connected with the obscure processes which are united under the vague term of "trophic disturbances."

Whether the neurotrophic influence, which seems to me to be inducing the hair's greyness, be due to central influences direct, or a peripheral modification of those central influences, as appears to me to be concerned in the etiology of cancer, I cannot accurately determine, for two reasons: First, according to Dr. Head, the commonly-described nerve areas of the head must be regarded as peripheral; and, secondly, observations upon the incidence of the hair's greyness on the body are extremely difficult; and, according to Dr. Head, it is the evidence shown by trunk-nerve areas upon which trust can be placed in estimating whether or no the nerve influence be central or peripheral in origin.

The difficulties of my position are obvious when I state that I have not seen any instance of hair on the body in which the hair covered three or even two maximum points of the same central or segmental nerve distribution. I can only say that I have seen men of middle age whose chests were covered with hair in which patches of greyness appeared on one or two points, but of different nerve areas.

REFERENCE.

¹ On the disturbances of sensation, with special reference to the pain of visceral disease. See *Brain*, vol. xvii.

THE PERCEPTION OF LIGHT AND COLOUR.

By F. W. EDRIDGE-GREEN, M.D., F.R.C.S.

A THEORY to be of any use as a working hypothesis must be able to explain the main facts of the subject, and in addition there must be no definite fact which is totally at variance with it. I propose in this paper to show how the theory which I have promulgated will explain the known facts of light and colour vision, and how impossible it is to adopt any form of elemental hypothesis.

I assumed that light falling upon the eye liberates the visual purple from the rods (just as heat would melt an ointment) and a photograph is formed. The decomposition of the visual purple by light chemically stimulates the ends of the cones, and a visual impulse is set up which is conveyed through the optic nerve fibres to the brain. I assume that the visual impulses differ in character according to the wavelength of the light causing them; that is to say, a very similar condition exists in the impulses which are transmitted along the optic nerve to that which is accepted for waves of light previous to their entering the eye. The fibres of the optic nerve pass to the visual centre. I have assumed that the visual centre transmits to the mind impressions of white light, and that by it objects are seen monochromatically, as in a photograph. The visual centre is, therefore, acted upon by impulses caused by all rays of light, the colour-perceiving centre being concerned with the quality of the impulse within the power of perceiving differences possessed by that centre or portions of that centre. It will be noticed that this theory consists of two distinct hypotheses—one retinal and the other cerebral. Each is supported by a separate series of facts. Each of these theories has enabled me to predict and subsequently ascertain the existence of a large number of new facts, most of which are quite inconsistent with any elemental hypothesis. In fact, the reader who is acquainted with the recent literature of this subject will see that other writers have, in addition to rediscovering many of my facts, arrived at a similar conclusion concerning them.

I will now review a certain number of facts of light and