

sac were stitched to the abdominal wall and the operation was abandoned for the time. On Feb. 6th she was again anaesthetised but on attempting to remove the plugging hæmorrhage was so great that it had to be replaced at once. Nothing further was done for a fortnight, during which time the plugs kept sweet and the temperature normal. On Feb. 17th (three weeks after the first incision and insertion of gauze packing) an opening was made horizontally in Douglas's pouch and a five months foetus was delivered. The cord was tied and the placenta was left *in situ*. The after-course was perfectly normal and the temperature was satisfactory. The discharge from the sac was foetid till March 10th (21 days after delivery) when the placenta came away. The cavity then healed up very quickly. The hæmorrhage at the first operation was due to the incision being in or near the placental site. Had immediate plugging not been resorted to the patient would have died on the table. As it was her condition was for a time serious though she improved rapidly under saline infusions. This patient was again admitted to my wards a fortnight ago with a distinct history and symptoms of extra-uterine foetation of two months' duration. She is there at present under observation.

CASE 8. *Left intra-ligamentary pregnancy of three months; operation; recovery.*—The patient, aged 35 years, was seen with Dr. J. Brander. She had been married 12 years and had a child a year later but since then she had not been pregnant. She was examined in December, 1904, for severe pain and abdominal swelling. She had not menstruated for three months. Bimanual examination revealed a swelling on the left side of a slightly enlarged uterus. A diagnosis of extra-uterine pregnancy was made and operation was advised. She was operated on on Dec. 12th and a left intra-ligamentary pregnancy was found. The sac was opened, the foetus was removed, and the edges of the opened broad ligament were stitched to the lower part of the abdominal wound. The cavity was packed with aseptic gauze with a reinforcing vaginal pad. There were moderate bleeding at the operation and once or twice oozing from the cavity during the separation of the placenta but the case ran an aseptic and uneventful course.

The other two cases were private ones but I think they present some features of interest.

CASE 9. *Chondroma of pelvis narrowing outlet; Cesarean section; recovery.*—(Dr. McKerron has kindly furnished the early notes of this case.) The patient, a primipara, aged 33 years, was in good health throughout pregnancy. In the beginning of the eighth month foetal movements began to be less active and by the middle of the month entirely ceased. Examined towards the end of the eighth month the abdomen was small for the period of pregnancy and no foetal heart sounds could be heard. At term, as there were no signs of commencing labour, a vaginal examination was made which disclosed a tumour projecting from the left side of the sacrum. The tumour, which was of cartilaginous consistence, was of about the size of a Tangerine orange and so narrowed the outlet of the pelvis, which was of the generally contracted type, that the distance from the symphysis to the nearest part of the tumour was only one and a half inches. The tumour was extremely tender, pressure on it causing severe cramp-like pains down the left leg. Labour was artificially induced. The breech presented and extraction was easily effected, the child being small and soft but not macerated. At the end of June, 1904, I saw her with a view to Cesarean section which had been determined on by Dr. McKerron and Professor Stephenson as the proper treatment, her second pregnancy being then near term. Sænger-Cæsaean section was performed on July 5th in the Richmond-hill Nursing Home. The child was a healthy female of average size. Both mother and child did well and left the home about a month later.

CASE 10. *Cæliotomy for painful adhesions during pregnancy; recovery.*—The patient, a multipara, aged 35 years, was seen with Dr. J. J. Y. Dalgarno. She was four months pregnant and had considerable pain over the lower left side of the abdomen, in which a painful thickening close to the fundus of the uterus could be felt. Operation was advised and she went to a nursing home for that purpose. The cause of the pain and thickening was found to be adhesions of the omentum. These were easily separated. The pain entirely disappeared after operation and the pregnancy went to full term.

Aberdeen.

## BLOOD CHANGES IN MENINGITIS IN CHILDREN.<sup>1</sup>

BY SYDNEY W. CURL, M.A., M.D. CANTAB.,  
M.R.C.P. LOND.,

PHYSICIAN TO THE ESSEX AND COLCHESTER HOSPITAL; LATE PATHOLOGIST AND BACTERIOLOGIST TO THE NORTH-EASTERN CHILDREN'S HOSPITAL, HACKNEY-ROAD, LONDON.

OUR knowledge of the changes occurring in the blood in diseases other than those of the blood itself has until quite recently been in a more or less undeveloped state; latterly, however, thanks to the labours of workers in this special branch of pathology, we have come to recognise that in some diseases of a general or constitutional nature the blood, as well as other tissues, frequently exhibits changes which are almost characteristic of the disease in question. This advance in our knowledge of the pathology of the blood has been assisted by the increased simplicity of technique in blood examination and by the fact that nowadays the blood of patients is investigated as a matter of routine and almost irrespective of the disease from which they may be suffering. From the vast amount of literature on the subject, which has already accumulated, and from inferences drawn from such, special blood changes are now known to occur in many diseases; an additional means of physical examination has thus come into prominence and the assistance given to the diagnosis and consequently to the treatment of disease has been very great. Further, the presence of many complications occurring during the course of a disease may frequently be either confirmed or refuted by a careful blood examination and a logical interpretation of the results; in illustration of this latter statement, one may quote the case of a patient suffering from an attack of typhoid fever; in this disease there is usually a diminution in the number of leucocytes in the circulating blood; towards the end of the second week or the beginning of the third the patient exhibits a sudden change, as indicated by severe abdominal pain, rapid pulse, and a rapid fall in the body temperature; one naturally discusses the possibility of the occurrence of a perforation of the intestine but owing to the dulling of the patient's sensibility by the continued fever and the toxæmia, the symptoms and physical signs are too indefinite to allow of a diagnosis being made with any degree of certainty. A leucocyte count may here be of the greatest value, for should the white cells show a definite increase above the normal number it is extremely probable that perforation has occurred, radical measures of treatment can then be carried out with the least delay and the life of the patient possibly saved, whereas, if the case be treated in a palliative manner till definite signs of peritonitis have appeared the patient will almost certainly die, whatever the treatment. Further, the presence of pus in any part of the body can often be confirmed by a leucocyte count and it is now quite common for a count of the white cells to be made in cases of appendicitis with a view of determining whether the attack be merely catarrhal or suppurative in nature, the presence of a marked, and especially an increasing, leucocytosis greatly favouring the diagnosis of suppuration.

From these few remarks it is evident that a knowledge of the changes which the blood undergoes in various diseased states is of the greatest value, since it may insure the rational treatment of disease which, after all, is the main aim of medicine. In order to interpret correctly any pathological changes found in the blood an intimate knowledge of its chief characters in health is essential; I shall therefore briefly describe the blood of a healthy adult, and since my paper deals with children I shall indicate the chief points of difference between the adult's blood and that of the child.

### SECTION A.

The blood consists essentially of the red and white corpuscles floating in a clear fluid, the liquor sanguinis. The red corpuscles are small bi-concave discs, from 7 to 8  $\mu$  in diameter and mostly rounded in form; they are thicker at the rim than in the centre and are composed of a fine network of protoplasm,<sup>2</sup> containing within the meshes a colouring matter, the hæmoglobin. In the male they number 5,000,000 to the cubic millimetre of blood and in the female somewhat less, about 4,500,000. The white corpuscles,

<sup>1</sup> Thesis for degree of M.D. Cambridge.

<sup>2</sup> Bruecke and Rollett: Quain's Anatomy, vol. i., part 2, p. 210.

of which there are 7500 to the cubic millimetre, consist of the following varieties: 1. Polynuclear cells. These are large cells, from 9 to 10  $\mu$  in diameter, the protoplasm of which contains fine granules, which stain faintly with both acid and basic dyes. The nucleus of the cell is very inconstant in shape, sometimes appearing in the form of a horseshoe, at other times as if divided into several distinct parts. These cells form from 60 to 70 per cent. of the leucocytes. 2. Small lymphocytes, cells of the size of, or a little larger than, the red corpuscles. Their nuclei are round, indented, or even horseshoe-shaped, stain deeply with basic dyes, and are surrounded by a small amount of clear protoplasm. They form from 20 to 30 per cent. of the leucocytes. 3. Large lymphocytes. These are as large as, or even larger than, the polynuclear cells, the protoplasm is "hyaline," and the nucleus, which is rounded or oval, stains faintly with the basic dyes. 4. Transitional forms. These are similar to the last with the exception that their nuclei are indented or horseshoe-shaped. The large lymphocytes and transitional cells form from 4 to 8 per cent. of the white cells. 5. Eosinophile cells. These are similar to the polynuclear in size and shape but the protoplasmic granules are larger and stain deeply with acid dyes; they form from  $\frac{1}{2}$  to 4 per cent. of the leucocytes. 6. Basophile cells. These are rarely seen in normal blood and although similar in form and size to the polynuclear cells their cell granules stain only with basic dyes.

The blood of a child differs from that of an adult in the following particulars: 1. Shortly after birth it contains a greater number of red corpuscles; thus Sorensen<sup>3</sup> states that in male infants from five to eight days old the number is 5,769,500 and in the female child from one to 14 days old 5,560,800 per cubic millimetre of blood. According also to Cabot the number in the newly born is very high. 2. The white corpuscles are very numerous; this excess is most marked immediately after birth and then gradually falls; thus Da Costa says that at birth the count varies between 15,000 and 20,000 or even higher; this decreases after the first 48 hours and at the end of the first week has fallen to 10,000 or 15,000, which number is normal for children under one year of age. From the sixth year and upwards the number of leucocytes is 7500.<sup>4</sup> Of the different varieties of leucocytes in childhood the small lymphocytes are relatively more numerous—from 40 to 60 per cent.—and the polynuclears are less numerous—from 16 to 40 per cent.

In considering the effect which a given disease may have in altering the composition of the blood one ought if possible to eliminate all those factors, which, whilst not forming an integral part of the disease in question, yet may profoundly affect the blood. Of these influences the following are those which most concern us here: 1. Digestion. The effect of digestion on the composition of the blood has been very fully studied by numerous observers, all of whom agree that the number of leucocytes in the circulatory blood increases soon after the taking of food, rises to a maximum four or five hours later, and then gradually subsides; this increase averages 33 per cent. of the whole number of leucocytes.<sup>5</sup> In children this digestion leucocytosis is especially marked and in infants under two years of age it frequently rises to as high a figure as 30,000. What influence digestion has on the differential count is doubtful, some observers having found an increase in the polynuclear cells<sup>6</sup> whilst others have found a lymphocytosis.<sup>7</sup> After a meal, especially when considerable quantities of liquid have been taken, the red cells fall in number owing to a temporary dilution of the blood. 2. Loss of fluids from the tissues of the body, such as occurs in severe vomiting or diarrhoea. The blood being by these means temporarily concentrated, there will be an apparent increase of both red and white corpuscles in number.

With this brief description of the blood in health and its chief physiological variations I shall now pass on to consider in detail the cases of which this thesis is composed.

#### SECTION B.

In the examination of the blood of my cases I adopted the following methods. The percentage of hæmoglobin was estimated by means of Gowers's hæmoglobinometer and

von Fleischl's hæmometer. For the enumeration of the red corpuscles the Thoma-Zeiss apparatus was used, the diluting agent being Toison's fluid. For counting the leucocytes the same pipette as that used for the red cells was made use of and, except where otherwise stated, at least 500 white cells were counted in making the differential count. The stains used for the blood films were Jenner's, Leishman's, and Ehrlich's triacid stains. Of the ten cases of meningitis which I examined eight were tuberculous in nature—namely, Nos. 3, 4, 5, 6, 7, 8, 9, and 10 (*vide* Table). No. 2 was also probably of the same nature, as although an examination of the brain could not be made, yet there was miliary tubercle of the intestines and peritoneum and the symptoms during life were fully in accordance with what one would expect in tuberculous meningitis; I have therefore considered it as belonging to this group. The remaining case, No. 1, was one of serous meningitis or hydrocephalus. Since the hæmoglobin findings exhibit no constant or characteristic features they may be dismissed as being of little value as an aid to diagnosis. The count of red cells was in most of the cases relatively high. Very little has been written on the behaviour of the red cells in cases of tuberculous meningitis and the only statement which I can find is that "the red cells show, as a rule, but slight changes, as is so often found in other forms of tuberculosis."<sup>8</sup> In six of the nine cases of tuberculous meningitis—namely, Nos. 3, 5, 7, 8, 9, and 10—there was no leucocytosis; of the remaining three, in two, Nos. 4 and 6, there was decided leucocytosis—namely, 42,000 and 21,000 respectively—and in No. 2 the count varied, on one occasion being 12,092 and a week later 27,000. From these results it is seen that in over 60 per cent. of cases of tuberculous meningitis there is no increase in the number of white cells. These results differ somewhat from those obtained by other observers; thus Cabot has recorded 27 cases, in 18 of which a leucocytosis was present; his results, however, are indecisive, since apparently the diagnosis was confirmed by necropsy in three only of the cases. In one case reported by Kneas a leucocytosis was present.<sup>9</sup> Rieder<sup>10</sup> has reported two cases, one showing a leucocytosis, the other a normal or subnormal count of white cells. Türk studied three cases, all showing a leucocytosis. Osler<sup>11</sup> says "a leucocytosis is not infrequently present throughout the disease." On the other hand, von Limbeck, in four counts made shortly before death, found no leucocytosis.<sup>12</sup>

As regards the percentages of the different varieties of leucocytes two points are to be especially noted—namely, a diminution of the number of eosinophiles and an increase in the large lymphocytes and transitional cells: thus in eight of the nine cases of tuberculous meningitis, Nos. 3, 4, 5, 6, 7, 8, 9, and 10, the relative proportion of eosinophile cells was below normal, in no case reaching  $\frac{1}{2}$  per cent. and in four cases they were entirely absent—namely, Nos. 4, 7, 8, and 10. These results are in marked contrast to the condition found in the case of serous meningitis, in which the eosinophile cells were normal or increased in number. In six of the tuberculous cases the large lymphocytes and transitionals were increased in number—namely, in Nos. 2, 3, 5, 6, 9, and 10, and in one of these they formed 23 per cent. of the total leucocyte count (No. 2). Very few differential leucocyte counts have been reported in tuberculous meningitis, but in those which I have found recorded a diminution or absence of the eosinophile cells was the rule.<sup>13</sup>

Granted, then, that in tuberculous meningitis there is usually no leucocytosis, have we any satisfactory explanation as to why such should be the case? There are, I think, four possible explanations. Firstly, the tubercle bacillus may enter the body and exert its characteristic effects without influencing in any way the number of leucocytes in the circulating blood. We know that the number of leucocytes in the blood is not the same for every individual but varies according to the robustness or delicacy of the person's constitution; thus in the robust it is relatively high, whilst in the feeble it is low, sometimes reaching as low a number as 3000 per cubic millimetre.<sup>14</sup> Other things being equal, one may therefore assume that persons with a low leucocyte count are more

<sup>3</sup> Cited by von Limbeck: *Gründriss einer klinischen Pathologie des Blutes*, Jena, 1896.

<sup>4</sup> Da Costa: *Clinical Hematology*.

<sup>5</sup> Cabot: *Clinical Examination of the Blood*.

<sup>6</sup> Burian and Schür: *Wiener Klinische Wochenschrift*, February, 1897.

<sup>7</sup> Cabot: *Loc. cit.*

<sup>8</sup> *Ibid.*, pp. 283-84.

<sup>9</sup> *Journal of Nervous and Mental Diseases*, New York, 1903, vol. xxx., p. 431.

<sup>10</sup> Quoted by Cabot.

<sup>11</sup> *The Principles and Practice of Medicine*, fourth edition, p. 279.

<sup>12</sup> Quoted by Cabot.

<sup>13</sup> Türk: Quoted by Cabot.

<sup>14</sup> Cabot: *Clinical Examination of the Blood*, pp. 105 and 95.

APPENDIX OF CASES.\*

No.	Age.	Tem- perature.	Per- centage of hæmo- globin.	Number of red cells per cubic milli- metre.	Number of white cells per cubic milli- metre.	Poly- nuclear cells (per cent.).	Small lympho- cytes (per cent.).	Large lympho- cytes and transi- tional forms (per cent.).	Eosino- phile cells (per cent.).	Remarks.
1	One year and three months.	98·0° F. 98·0° F.	92 100 (?)	5,066,660 4,552,000	18,430 12,684	43·8 47·2	39·4 42·3	11·7 5·6	4·4 4·1	Necropsy. Serous meningitis with hydrocephalus. A week elapsed between the two blood examinations.
2	Seven months.	98·2° F. 100° F.	55 60	5,200,000 5,096,000	12,092 27,390	36·1 44·18	39·2 42·9	23·42 12·5	1·1 0·36	Necropsy. The brain could not be examined but there was miliary tubercle of the intestines and peritoneum and during life symptoms of meningitis. A week elapsed between the two blood examinations.
3	One year.	98° F.	64	4,952,000	11,455	78·9	11·0	9·5	0·47	Necropsy. General tuberculosis with tuberculous meningitis. Only 200 white cells were counted in the differential count.
4	11 months.	99·8° F.	82	5,480,000	42,984	83·3	11·5	5·1	—	Necropsy. General tuberculosis, excess of subarachnoid fluid, but no definite tubercles were seen.
5	Two years and eight months.	101·4° F.	94	4,760,000	10,476	65·7	24·5	9·5	0·18	Necropsy. Tuberculous meningitis.
6	Three years.	100·2° F.	74	4,704,000	21,410	70·6	20·07	9·5	0·19	Necropsy. Miliary tuberculosis with tuberculous meningitis.
7	One year and one month.	98·0° F.	55	5,904,000	9,461	73·0	20·7	5·8	—	Necropsy. General tuberculosis with tuberculous meningitis. Only 275 white cells were counted in the differential count.
8	Four years and five months.	99·0° F.	70	6,720,000	5,343	88·8	6·7	5·0	—	Necropsy. General tuberculosis with tuberculous meningitis. Only 280 white cells were counted in the differential count.
9	Seven months.	101·8° F. 97·0° F.	70	5,352,000	15,826 12,632	64·5 69·3	24·8 21·4	10·4 9·1	0·16 0·14	Necropsy. General tuberculosis with tuberculous meningitis. A week elapsed between the two blood counts.
10	Five years.	100·6° F. 100·4° F.	77	6,614,400	12,631 12,317	81·4 83·0	7·8 7·1	10·5 9·8	— —	No necropsy. Symptoms of meningitis during life. Three days elapsed between the two blood examinations.

\* In the above list of cases the blood was taken from the patients shortly before or after a meal, so as to reduce to a minimum errors due to the effect of digestion on the composition of the blood. In no case was there any excessive drain on the blood, such as severe vomiting or diarrhœa.

liable to infectious diseases of all kinds than their more robust companions. On this supposition there is no direct connexion between the occurrence of tuberculous disease and the leucocyte count, both being the result of a common cause—namely, depressed vitality. Secondly, there may be no leucocytosis because the irritation of the tissues by the tubercle bacillus and its toxins is either too mild in nature or too severe. In support of this we have experimental evidence; thus, if in an animal a dose of bacterial products of sufficient strength or large enough in quantity to cause death be injected, no leucocytosis occurs; to obtain a leucocytosis the dose must be of moderate size or strength and the resistance of the animal good. Now, there is no evidence that the toxic products of the tubercle bacillus are of a very mild nature; on the contrary, the exact opposite is the case, as evidenced by the effects produced by injection of tuberculin. For reasons, however, which will be discussed later, there is evidence to show that the most active constituent of the tubercle toxin can be taken up by the body tissues in very small quantities only and, such being the case, the irritation is insufficient to cause a leucocytosis (one of the signs of reaction of the body to an irritant). Thirdly, the tissues being constantly exposed to the toxins of the tubercle bacillus may in time come to be endowed with a relative immunity to them and therefore will no longer exhibit any reaction on further exposure to the irritant.

Since every case of tuberculous meningitis is secondary to a deposit of tubercle in some other part of the body one may justly assume that during the time that elapses between the dates of primary infection and the onset of secondary disease in the cerebral meninges a continual absorption of tubercle toxin has been going on and although at the time of primary infection the irritant effect of the toxin may manifest itself in the usual manner, yet by a constant exposure to the poisons the body has so accommodated itself that a reaction in the form of a leucocytosis now no longer occurs, even when a general dissemination of tubercle takes place. Fourthly, the most active and powerful toxin produced by the tubercle bacillus may arise in the body of the bacillus itself but owing to the nature of the bacterial cell is unable to escape in sufficient quantities to exert its irritant action on the body tissues. All pathogenic organisms produce as a result of their growth extracellular toxins; some, however, such as the diphtheria, tetanus, and tubercle bacilli, also form intracellular toxins. Now the composition of the cell bodies of the diphtheria and tetanus bacilli is such that no great opposition is offered to the escape of any intracellular toxin which may be formed; this on being absorbed gives rise to a leucocytosis, the usual condition found in cases of diphtheria and tetanus. On the other hand, the tubercle bacillus being composed of various insoluble fatty acids

any intracellular toxin formed can either not escape at all or can escape in very small quantities only unless the bacilli die in large numbers, when the intracellular toxins are diffused into the surrounding tissues and can then be readily absorbed. If tubercle cultures be filtered germ free and the filtrate be injected into an animal a less marked reaction occurs than if the bacilli as well as the filtrate be injected (some say this is due to the filtrate containing a toxalbumin having a temperature-lowering effect); this seems to favour the view that the intracellular toxins of the bacillus are more active than the extracellular. A patient, then, having become infected with the tubercle bacillus, there are produced at the seat of invasion extracellular toxin which has very little irritant effect on the tissues and intracellular toxin; the latter, however, is taken up by the body in such small quantities that the irritation produced is negligible and a reaction in the form of a leucocytosis does not appear.

Regarding the second finding in my cases—namely, a diminution in the number of eosinophile cells—we have no adequate explanation, the function of these cells being at present but imperfectly understood. Some observers consider that they are concerned with the defence of the body against invading micro-organisms, the granules of the protoplasm being secreted by the cells themselves and consisting of a substance which is injurious to bacteria. This view is supported by the fact that either finely granular oxyphil cells or coarsely granular (eosinophiles) are generally the first to arrive at the seat of invasion by a micro-organism.<sup>15</sup> Kanthack and Hardy,<sup>16</sup> however, state that in the case of the rat, the frog, or the guinea-pig, the coarsely granular oxyphil cells are never phagocytic. From these conflicting statements it is therefore evident that we are still far from knowing what the function of these cells is and any attempt to explain an increase or a diminution of them would be unwarrantable. The large lymphocytes and transitional forms being, as is well known, intensely phagocytic in nature their increase is most probably due to an attempt on the part of the body to rid itself of the offending agent—namely, the tubercle bacillus.

The conclusions to be drawn from the results of my investigations are that in tuberculous meningitis in children, (1) the red corpuscles are usually present in normal or increased numbers; (2) the majority of cases show no leucocytosis; (3) the eosinophile cells are reduced in number; and (4) the large lymphocytes and transitional cells are generally present in increased proportions.

For permission to make use of the cases in the wards of the North-Eastern Children's Hospital I am indebted to Dr. W. A. Wills, Dr. J. Taylor, Dr. J. Porter Parkinson, and Dr. G. Carpenter, and for access to the clinical notes of the cases my thanks are due to the house physicians, Mr. S. L. Harke, Mr. R. M. Im Thurn, and Dr. R. G. Nothwanger. Colchester.

## TWO CASES OF TRICHROMIC VISION.<sup>1</sup>

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

**CASE 1.**—One case (Professor J. J. Thomson) sees only three colours in the bright spectrum—red, green, and violet. He can distinguish nothing of the nature of pure yellow, like the sensation given him by the sodium flame in the spectrum. There is no definite colour to him at the portion of the spectrum where the normal sighted see pure blue. Reddish-green would describe the orange and yellow regions and greenish-violet the blue.  $\lambda$  5950 (orange-yellow) is the point which differs most from red and green. There was no shortening of either end of the spectrum. The point of junction of the red and green differed somewhat in repeating the observations because of his great sensitiveness to simultaneous contrast. It was, however, always in the orange or orange-yellow, never in the yellow of the normal sighted.

**Difference of hue perception.**—I then tested him with my apparatus for ascertaining the size of different parts of the spectrum which appear monochromatic and found that he was defective in distinguishing differences of hue. A portion

of the spectrum corresponding to the D lines and isolated by two shutters in the eye-piece of the spectroscope was first shown. The shutter on the red side was gradually opened until a difference of hue was seen. The monochromatic patch extended from  $\lambda$  5889 to  $\lambda$  6052, being exactly half as large again as that of the normal sighted, which occupies the space from  $\lambda$  5889 to  $\lambda$  5998. The monochromatic patch he called greenish-yellow. His monochromatic patch in the centre of the green bore exactly the same proportion to mine as in the case of the orange-yellow, being just half as large again.

**Colour mixtures.**—He was then tested with Rayleigh's apparatus for matching spectral yellow by a mixture of red and green. 0 of the scale corresponded to pure red, and 25 or 90° to pure green. He made the following 10 matches:—

Match.	Difference.	Match.	Difference.
†1. 13.40 ... ..	+ 0.67	* 6. 11.75 ... ..	- 0.98
*2. 12.0 ... ..	- 0.73	* 7. 11.75 ... ..	- 0.98
†3. 12.75 ... ..	+ 0.02	† 8. 13.50 ... ..	+ 0.77
*4. 12.0 ... ..	- 0.73	† 9. 12.90 ... ..	+ 0.17
†5. 13.50 ... ..	+ 0.77	*10. 13.75 ... ..	+ 1.02
Average, 12.73.		Average difference, 0.684.	

I find that my colour vision agrees with that of the large majority of persons and may therefore be regarded as normal. I made the following ten observations for comparison with those given above:—

Match.	Difference.	Match.	Difference.
*1. 11.0 ... ..	+0.629	* 6. 10.0 ... ..	-0.371
†2. 10.25 ... ..	-0.121	† 7. 10.50 ... ..	+0.129
*3. 9.80 ... ..	-0.571	* 8. 10.50 ... ..	+0.129
†4. 10.33 ... ..	-0.041	† 9. 10.60 ... ..	+0.229
†5. 10.33 ... ..	-0.041	*10. 10.40 ... ..	+0.029
Average, 10.371.		Average difference, 0.229.	

\* Red shown first in the mixed colour. † Green shown first.

My match appeared to him bright-red and bright-green, the yellow appearing as green. The match appeared more correct through a pin-hole. The mixed colour of his match always appeared green to me. It will be noticed that his average difference is very nearly three times the amount of mine. On comparing the differences, according to the colour which was shown first, it will be found that these were all positive when the green was shown first and four out of the five were negative when he commenced with the red.

Green first	+ 0.67	Red first	- 0.73
	+ 0.02		- 0.73
	+ 0.77		- 0.98
	+ 0.77		- 0.98
	+ 0.17		+ 1.02
Average, 0.48		Average, 0.888	

Below I give my differences for comparison.

Green first	- 0.121	Red first	+ 0.629
	- 0.041		- 0.571
	- 0.041		- 0.371
	+ 0.129		+ 0.129
	+ 0.229		+ 0.029
Average, 0.1122		Average, 0.3458	

He seemed to get very easily fatigued with colours.

**Classification test.**—Called 1 (orange) "reddish-orange" and matched it with orange and dark yellows. Described 2 (violet) as mauve and put with it violets and purples. Named 3 (red) correctly and picked out various reds to go with it. Called 4 (blue-green) "green" and matched it with some greens. Only a few colours were selected in each case. On being asked to pick out all the yellows he chose those with orange in them. He regarded orange-yellow as his yellow and rejected pure yellows because he said that they had green in them. He had considerable difficulty in matching the colours. In common with the cases I have previously observed the effects of simultaneous contrast were much more marked than in the normal sighted. Two wools changed colour to him on being contrasted when no change was evident to me. This was particularly noticeable when one of the contrasted colours was red, green, or violet, and the other, one of the intermediate and adjacent colours.

**Lantern test.**—He correctly named the red, green, and violet with and without the neutral glasses and saw them at the normal distance. He had difficulty with yellow and blue. He called pure yellow "greenish yellow." It will be noticed that the examination with the spectrum gives a key to the mistakes made.

<sup>15</sup> Lazarus-Barlow: Manual of Pathology, pp. 406 and 282.

<sup>16</sup> Kanthack and Hardy: Ibid.

<sup>1</sup> A paper read before the Royal Society. The research was made with the aid of an instrument purchased with a grant from the Government Grant fund.