VISUAL AGNOSIA AND ALTERNATING DOMINANCE; ANALYSIS OF A CASE.

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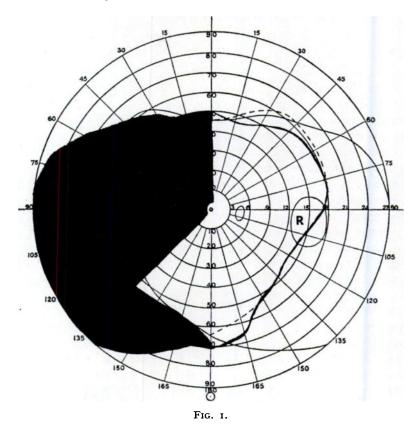
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The case here described is of interest in that it concerns a young man of good intelligence who presented a disorder limited to visual-gnostic functions; this is noteworthy as the majority of the cases described are elderly patients and there are usually other than visual gnostic functions involved. The case has special circumstances of additional significance. The patient had lost the sight of his right eye in infancy and he was a definite right-handed man; the damage involved the right hemisphere; this exceptional coincidence raises interesting problems of hemispheric dominance.

The patient, J. S. B-, 32 years of age, a civil engineer with Honours University degree, had a history of an endogenous depression going back a few months prior to his admission. He thought he had contracted tubercle, refused promotion offered to him, reproached himself that he had not made the most of his degrees and felt his career was finished. When admitted to hospital on 1.ii.52 he was in a depressed state, worrying about his condition and was somewhat agitated. He was very apprehensive about being put into a mental hospital and felt that he would have to stay in hospital for his lifetime. The morning after his admission he made in a sudden impulse a cut with his right hand into the right side of his neck. There was fairly heavy bleeding. He struggled with the nurses and doctors when attended to, and it was necessary to compress manually the right side of the neck for twenty minutes until the bleeding points were ligatured. The patient was carried to the theatre and the wound was inspected and sutured under general anaesthesia. There was no injury to the carotid artery nor to the internal jugular vein. He had two pints of blood transfused, and in the next two days he was kept under fairly heavy sedation with sodium amytal. His temperature showed a slight rise in the first few days but was normal later. The wound healed without complications.

It was possible to carry out an examination of the patient on the third day after the incident occurred. He was then still rather slow and hesitant in his answers. There were no aphasic symptoms. His memory seemed to be unimpaired; he could recollect all the details of what happened in recent weeks, and could remember his journey to hospital and his suicidal attempt. He could give a detailed history about his illness, and his information was essentially the same as that obtained from his relatives. Some stock phrases recurred in the earlier examinations.

When first examined it became apparent that the patient had some difficulties in vision. When an object was exposed he had to look for a long time before he could say what object it was, and he gave a wrong answer on various occasions. On confrontation test there seemed to be a loss of the temporal half of the visual field of the left eye. He himself stated later that he had for several days after his suicidal attempt the feeling as if he was blind, but any more detailed description of this condition could not be obtained from him.



Detailed testing was carried out in numerous sessions and the following is a summary of the results.

On neurological examination there was a slight paresis of the left lower facial muscles. His visual field was checked on a number of occasions and showed in the left eye a temporal defect which was complete in the upper quadrant, a narrow medial sector being preserved in the lower quadrant (see Fig. 1). Macular vision was intact. He was unaware of his field defect. His visual acuity tested a week after his suicidal attempt was on the left $\frac{\theta}{9}$. There was a left-sided papilloedema. There were scattered white exudates all over the right fundus, perivascular sheathing and a complete atrophy of the disc; there was no vision in the right eye (Dr. Fraser). The lumbar puncture showed c.s.f. pressure of 200 mm. and 120 mgm. per cent. protein.

There was some retardation but the flow of speech was quite good when once started. For about a week he would repeat a number of phrases referring to his condition, which recurred frequently in various interviews. He could express himself well, and he had no difficulties in finding words either in conversational speech or in naming objects when he recognized them. His comprehension was perfect. From the beginning he could read fluently without any mistakes, and there were no mistakes in spontaneous writing or upon dictation. He had no difficulties with reading or writing of numbers; he could pick out a number from a multitude distributed over a larger space. He was able to do calculations and arithmetical problems correctly though slowly. He was rather slow in his thinking, but he showed a good level in explaining differences, similarities and proverbs. His general knowledge corresponded to his educational level. His memory for past experience was good and there was no retention defect. There was no defect in body orientation. On only one occasion in the first week he mixed up right and left when asked to indicate a definite time on a watch he drew, but he never made mistakes in right-left of the body or outside space. His tactile recognition of objects was intact.

The functional loss was confined to the *visual gnostic field*, and involved at the beginning most of the functions connected with it. Some of these functions recovered in a comparatively short period whilst recovery in others was protracted.

Recognition of objects.—At the first examination the patient had difficulty in distinguishing some of the common objects around him. In subsequent days, lasting about a week, he showed some hesitation and a prolonged reaction time in recognition of objects which were not quite so familiar. He also could not identify various belongings, e.g., pen, comb, tooth-brush, bed and locker as his own.

Recognition of single pictorial objects:—He never recognized or named a pictorial object instantaneously. After a prolonged reaction time he finally recognized as a rule familiar pictorial objects; but he had great difficulty in distinguishing pictures of objects belonging to a common group; for instance, he called a cow a horse, a cat a squirrel, and a dog a small animal without being quite sure what kind of animal it was. He did not recognize a cupboard, although he could describe it in most of the details if not quite accurately. He had still some difficulty six weeks after the incident in recognizing the drawing of a cow; he described it as "a horse's body with a cow's udder, sort of half horse and half cow—may be it is not a horse. Yes, it is really a cow." The same picture was shown to him twelve days previously, and this might have influenced his reaction at the time. At the end of March, eight weeks after the incident, he had no more difficulties with single pictures.

Pictorial situations.—At the beginning the patient was quite unable to recognize even the simplest pictorial situation. This may be illustrated by the following comment he gave to the Terman picture of a man carrying an umbrella; "It is a figure and something half-moon shaped, a segment, a circle, a figure with something on his back, some sort of satchel, man with a saw upside down. I should know . . . it is a sort of sharp . . . he is holding a long shaped thing, a sort of heavy frame." To the Terman picture

of a white man fighting Indians he said: "A figure holding something in the hand like that, a bar with metal; he is lifting something with the hand, a kind of excercise; a figure there and a figure there." Later he said: "There is a man who is holding something but it doesn't convey anything to me at all. There is a bar along there. This figure is holding something, something with weights, a thing used for lifting." When further pressed he said: "A group of three figures. This figure is holding something in his hand: it could be a hammer." Finally he though it might be athletics—"sports people." Similar responses were obtained for about three to four weeks and then a gradual improvement took place. After this period his difficulties were mainly in pictorial situations of photogravure type such as pictures of Murray's Thematic Apperception Test. The following are his reactions to Picture 17 G.F. of the T.A.T. (Houses, boats on the river; a woman on the bridge). He had some difficulty turning it into the right position. "It is a bit complicated; it is a wheel; that is the harbour and these are spokes of the wheel pointing to the sun." He now turned it into the correct position but was not happy about it and again turned it round. He now saw a house with windows, then after a time he saw a figure and thought that this figure was on a sort of ladder, then he corrected; "It might not be a wheel; it is the sun, the rays of the sun." He then saw another house and he summed up: "It is a morning scene, somebody setting off to work in the fields." After six weeks he had no difficulty in interpretation of such drawings as depicted in Terman tests but recognition was still very incomplete in the pictures of the Thematic Apperception Test and he was unable to grasp the situations in an adequate way. After about three and a half months he was able to give a satisfactory and detailed interpretation even of complex pictures.

After attending cinematographic performances during the first few weeks of his illness he could only give a very poor account of the action and stated that he was unable to follow the theme properly as everything was "muddled."

Spatial orientation.—From the beginning he could estimate correctly the absolute and relative distance of objects. There was severe disorientation in place; when in the corridor leading to his room he did not know in which direction to go. He was unable to identify his own room—a small dormitory and was unable to find his bed. When taken to his bed he was uncertain whether it was in fact his. He tried to confirm this from various possessions on his locker, but because of the difficulty in identifying his belongings he could not make up his mind. This very gross defect in orientation lasted for about a fortnight. After this his orientation improved, though he had some residual difficulties. He was still unable to make an adequate sketch of his dormitory, nor could he find his way back from the consulting room to his own room after he had made this journey every day for more than a fortnight. He was taken to Glasgow four weeks after the incident. He had been quite familiar with the city before his illness, but now he could not say, when in the centre of the city, where he was nor could he find his way to outstanding places or buildings. The orientation in space improved at about the same rate as the recognition of pictorial situations. But it took him still longer than usual to orient himself in new surroundings even at the time of his discharge.

Recognition of persons.—His relatives and previous friends did not come to see him in the first few weeks of his illness, but at the beginning he had great difficulty in recognizing nurses and doctors whom he saw frequently. He was also quite unable to describe adequately either the features or build of these persons. He regarded as very similar in their appearance persons who were in fact quite different. In describing persons whom he saw every day he would say that a thick-set, rather small man was tall and thin, and he made other similar mistakes. These difficulties in remembering and identifying persons receded at the same pace as the difficulties in recognition of pictorial situations and the spatial orientation.

Memory for recent visual experience.—Immediate retention of visual impressions of objects was reduced. When four geometrical figures were exposed for three seconds he recalled two of them and these only approximately. He had not lost the ability to store visual experiences, but he recalled much less material than one would have expected from him normally. His defective memory for persons and his difficulties with spatial orientation can also partly be ascribed to defective visual memory. He could describe from memory in some details familiar objects, animate and inanimate. One has, however, to remember that such descriptions can be made without visual imagery. He was, however, at a complete loss when incomplete pictures were presented to him; they did not convey anything to him. Similar negative responses were obtained when he had to complete such drawings; he was completely sterile in such completion tests. His best effort was to close by straight lines an incomplete figure presented to him. In contrast to this he produced very good responses after his recovery. The improvement in visual imagery more or less coincided with his improvement in drawing.

Drawing.—His profession required skilful drawing and he had never had any difficulties before. The following were characteristic of his drawings at the beginning of his illness: Copying and free drawing showed an undue haste as if he wanted to complete it as quickly as possible. He was at the same time careless; he did not mind if the line was straight or not and he did not check up or make corrections. His drawings were altogether very sketchy and disorderly. No similar reactions were observed in writing or other performances.. In copying pictorial objects he did not keep to the original but drew his own pattern which was not precise and left out some details. When copying animal pictures he sometimes left out distinguishing features (e.g., udder of a cow). He could draw familiar objects from memory but the style of his drawing was very simple. Thus he would draw a man's limbs in simple straight lines, whilst later when his symptoms had disappeared he presented a skilful and elaborate drawing (see Figs. 2A and 2B). His drawing of animals was particularly revealing. There was little differentiation in his drawings of similar species; e.g., a dog looked more like a cow. He could copy correctly geometrical figures of any complexity, including Abelson figures. He could draw the map of Great Britain quite well and insert the principal towns in the right places. The drawing of a map of Africa, however was only approximate. He recovered what might be regarded as his previous skill in drawing after about three to four months.

Colour agnosia.—Before his present illness he had no difficulties with colours. In his occupation as a civil engineer he regularly made coloured drawings and never made any mistakes in choosing the correct colour.

The following tests on colour vision were carried out: the various tests on Holmgren skeins, naming the colours of coloured objects shown to him,

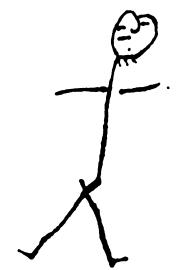


Fig. 2A. A week after the brain injury.



Fig. 2B. Four months after the brain injury.

naming the colours of objects with standardized colours from memory, naming of various objects of a particular colour, copying of coloured drawings, copying of a band of colours, picking out colours from a multicoloured object, Ishihara test, and colorimetric testing.

In the first four weeks he was unable to do any systematic sorting on the Holmgren's skeins; he found everything "in a tangle." He would sort out a few reds, and then he might add a blue or a bright brownish colour to it. He could not separate the various assortments he had made and thus became completely mixed up. He had equal difficulties with all colours. In naming of single colours or comparing two colours and in any of the other tests mentioned he was never quite certain, and wrong answers were more often than right ones. When deciding on a colour at this time he would look for a long time at the exposed skein, would often complain about the poor light; although it was bright daylight, he would often rub his eyes as if he could not see the colours properly and in the end he was never certain about the result. He admitted on various occasions that he had been guessing. He could only read the first figure in the Ishihara test at this period.

There followed a slow, gradual improvement; after eight weeks there was more order in the sorting of skeins, though there were mistakes with all the colours and particularly with blue and green. Colorimetric testing at this time (Dr. Pickford) showed a marked defect in red which classed him as red anomalous without darkening of the red, and a slight weakness in yellow. The finding was considered consistent with a moderate deuteranopia with the exception of the weakness in yellow, which could have been an independent minor defect.

In the period following this examination the colour distinction made further progress; he was able to sort out correctly, with some regularity and confidence, yellow and next red, but he was still uncertain about green and blue which were frequently confused. After three months he usually separated the primary colours in the sorting test and named them correctly when presented singly, but he made numerous mistakes with mixed colours. In particular he had difficulty in distinguishing brownish colours from red and dark colours from black. He still looked at each skein very carefully before he made his choice. He consistently made more mistakes in the copying of multicoloured drawings and of colour bands than in other tests. At this period the results still varied greatly in the various sessions, particularly in the sorting test.

There was also a gradual improvement in the Ishihara test and the number of correct results increased. The mistakes, however, were fairly equally distributed over all the plates and did not seem to indicate any specific anomaly. On 9.x.52, seven months after the damage had occurred, he was again tested by Dr. Pickford, and he showed a marked improvement on both the colorimetric test and on Ishihara as compared with his performance five months previously. There was only slight weakness in red and a very small weakness in the direction of blue colorimetrically, whilst yellow, which was weak before, was now normal. He now made eight mistakes on Ishihara in twenty-five plates; this is within normal limits. There was, therefore, in this examination not enough evidence for regarding him as a red anomalous or moderate deuteranope: his colour vision had to be regarded as within normal limits.

When last tested at the beginning of November, 1952, he still made some mistakes on the sorting of Holmgren wools; he placed the brownish colours occasionally to red and had some difficulty in distinguishing dark colours from black. Moreover, in copying coloured pictures or coloured series he had difficulties in choosing the correct shades.

The visual fields were checked regularly, and when last tested at the beginning of November they remained unchanged. At that time he still had complete loss of the temporal upper quadrant and the defect of the lower quadrant was the same as at the beginning of his illness. The degree of papilloedema of the left eye was unchanged. The visual acuity had remained at $\frac{e}{3}$. Lumbar puncture, performed a few days before the patient's discharge showed c.s.f. pressure of 120 mm. and 60 mg. per cent. protein.

The patient did not complain of any physical symptoms, and in particular he never had any headaches. He remained deeply depressed two months after his admission. It was then decided to give him a course of E.C.T.; he gradually improved from then onwards and at the time of his discharge he was quite well.

SUMMARY OF THE CASE.

An engineer of good intelligence, 32 years of age, attempted suicide by cutting the right side of his neck whilst in a depressive state. He was right-handed and had been blind in his right eye since infancy. Manual compression of the neck was necessary for about twenty minutes before the bleeding vessels could be ligatured. He developed a temporal visual field defect of the left eye after this incident. He had a slight agnosia for concrete objects, a more marked agnosia for pictorial objects and a still more severe one for pictorial situations and for persons. He had a severe disorientation in place and a colour disturbance. There was a gradual restitution of all these functions except for the field defect. The length of time it took the various functions to recover corresponded to the initial severity of the disturbance. The colour disturbance was the last to recover, and there were still signs of weakness in colour differentiation when the patient was last tested about nine months after the injury. From the beginning the patient had a slight oedema of the left active eye. He had a visual acuity of $\frac{6}{9}$ from the start of his cerebral symptomatology.

DISCUSSION.

Three main groups of agnosic disorders were present in our case, namely object agnosia, spatial agnosia and colour agnosia. As they are all closely connected with the visual system they are frequently found simultaneously, though they may occur independently of each other.

A distinction has been made between agnosia for concrete objects, pictorial objects, pictorial situations and for persons. In the great majority of cases the agnosia for concrete objects is least and that for pictorial situations most involved. This is also true for our case, where in the later stage of the restitution the agnosia for pictorial situations alone persisted. It seems, therefore, that the intensity of the disturbance increased with the complexity of the

visual object. It has, however, been suggested that the agnosia for pictorial situations is a separate entity due to a special defect, the inability to perceive a pictorial scene simultaneously (simultaneous agnosia, Wolpert, 1924). The disturbance in our case which was marked for single pictorial objects was more severe for pictorial situations drawn in contours, and still more so for the photogravure type of pictorial situation. His reaction time for recognition of pictorial objects was prolonged; he occasionally mixed up details, and his description was often incomplete. He usually recognized familiar pictorial objects that had well-defined and characteristic general shape, but he misidentified objects which, belonging to a common group, are only distinguished by details; he was accordingly particularly poor in animal pictures.

In pictorial situations drawn in contours more details were missed, and objects which he was able to recognize singly were not identified in the situational context. In photogravures a long exposure time was needed before definite shapes emerged. He was unable to obtain an immediate general impression and proceeded gradually from part to part. The parts were incompletely perceived, details were missed, and when he finally integrated the partial impressions into a whole his interpretation was inaccurate and inadequate. His difficulty in the recognition of single pictorial objects became therefore more marked when the pictorial object was less representative and when more details were needed for differentiation. In pictorial situations, more so in photogravures than in contour drawings, the single objects lose some of their representative character, as by additional features the parts are related to each other and to the whole and so more details have to be registered. Correspondingly, the difficulties of our patient increased. His specific defects were thus particularly exposed by the special conditions in pictorial situations and more so in drawings of photogravure type. There is, therefore, no reason in our case to make a separate factor responsible for the predominance of agnosia for pictorial situations.

The manner in which our patient responded to pictorial objects, particularly to photogravures, suggests a defect in visual perception which can best be illustrated in terms of Gestalt principles. A pictorial situation was for our patient first an unorganized mass from which the figure was sorted out from the background only gradually, and such figures as were formed lacked precision and completeness. But the perceptive defect alone cannot explain all the reactions of our patient. On occasions he was unable to identify a pictorial object even when able to describe all the details; his perception was thus adequate and the Gestalt well structured, but the necessary associations failed to emerge. Since Lissauer (1890) postulated theoretically a perceptive and an associative form, these two forms of agnosia have found special consideration in the literature of agnosia. Some cases were claimed to present pure examples of one or the other of these forms (Goldstein, 1923; Brain, 1941; Adler, 1944), but in most cases both the perceptive and associative factor seemed to have been involved. Copying is one of the ways to decide whether there is an associative or perceptive form present. In associative disturbance with intact perception the copying is correct, whilst in a perceptive disturbance the patients are unable to copy.

In our patient the copying as well as the free drawing was altered. He drew in a sketchy and casual way. His drawing was inaccurate and simple. A pattern once adopted reappeared in successive drawings. He never kept to the original drawing when copying. There was, therefore, in his performance in drawing not much evidence for either perceptive or associative defect. It indicated rather a vagueness of his visual imagery, the imagery being simplified and lacking in distinctiveness. The undue speed of his drawing pointed moreover to some difficulty in retaining a visual image. The poverty of his visual imagery became still more manifest in completion tests. Neither visually nor by drawing was he able to complete even the simplest patterns.

The agnosic disturbances for persons and the disorientation for places do not require a separate analysis. Factors mentioned previously are sufficient to explain his difficulties: the failure of perceiving details, his difficulty in shifting the visual attention and the defects in visual imagery may have been the main factors for these disturbances.

The most outstanding features of the object agnosia in our cases were thus perceptive and associative defects and defects in visual imagery. But there was also a reduced retention of visual perception, a perseveration of visual experiences and inability to shift the focus of visual attention and to reorganize visual impressions. Thus the defect involved numerous factors essential for visual function at a higher level; a process which before proceeded automatically had become a laborious, fragmented procedure. Perceptive and associative difficulties were thus only part of a general disintegration, and it would be of no particular value to weigh up one against the other. Nor can the disturbance in figure ground formation be regarded as the key to the disorder, although the mode of the altered perceptive procedure can best be demonstrated in terms of the Gestalt principle.

Some discussion has arisen about the significance of peripheral defects in visual agnosia. This peripheral factor has been particularly stressed by Poppelreuter (1923), Beringer and Stein (1930) and Stein and Buërger-Prinz (1932). More recently Bay and Lauenstein (1947) found in an analysis of a number of cases in the literature and from observations of their own that in all these cases peripheral defects were present which were severe enough to explain the agnosia; they come to the conclusion that a visual agnosia as described in the literature does not exist. Although this extreme view is not justified it is true that in a number of reported cases peripheral factors did not get the consideration they deserve. In our case the visual acuity was from the beginning good, and in contrast to other visual functions the perfect reading of ordinary print leaves no doubt about his satisfactory visual acuity. There is in this case, therefore, no doubt about the central origin of the defects.

Colour disturbance.—Three types of colour defects of cerebral origin can be distinguished; cortical colour blindness, colour agnosia and amnesia for colour names. In cortical colour blindness the colour sense is lost. In the cases described (Wilbrandt, 1892; Poetzl, 1928 et al.) the patients saw everything grey or could only distinguish between white and black, and in Wilbrandt's case there was a residual red-green blindness. In colour agnosia the patients are unable to identify and sort out particular colours whilst their colour vision as

tested by colorimetric methods is intact. In amnesia for colour names the colour vision is intact as well, and the predominant difficulties are not those of colour identification but of naming the right colours.

Our own case showed when tested colorimetrically defects from which he later recovered. This suggests that there was initially a cortical colour blindness present. However, at the time when he had colorimetrically only a weakness in red and a slight weakness in yellow, on sorting and similar tests all the colours were more or less equally and fairly severely involved, and at the time when he was colorimetrically considered normal he still showed difficulties in sorting of some colours. This suggests that there was superimposed upon a cortical colour blindness a colour defect of agnosic type, and this is also indicated by his defect in colour imagery.

Although there is a clear distinction between cortical colour blindness and colour agnosia by definition, various reactions of patients with colour agnosia seem to suggest that there is a defect in perception as well. Their responses are indeed in many ways similar to those found in colour blindness. They may often match colours according to brightness instead of hue, they frequently have difficulties with the differentiation of green and blue similar to trichromatic anomalies (Poetzl, 1928; Stengel, 1944 et al.). Many patients with colour agnosia state that they see the colours different from what they appeared to them before their illness. The difference in the conditions between colorimetric testing and testing on Holmgren skeins has been pointed out in order to explain why the patients fail in one and not in the other (Sittig, 1921): in the colorimetric test only the equality and difference of two colours have to be decided, whereas sorting and other related tests are more complicated; in the sorting tests the surroundings might interfere with recognition by the effects of contrast (Poetzl). However difficult it may be to explain these reactions, they are not a strong argument in favour of a perceptive theory; it has to be kept in mind that even slight deviations from the normal are being revealed colorimetrically. One can, therefore, hardly believe in a perceptive disturbance in colour agnosia when the colorimetric testing is so regularly found normal.

Colour recognition is a much more elementary process than recognition of objects, and investigation reveals little material to show that there is a disturbance at a higher level; but one factor present in all the observations is a defect in colour imagery. This applies to amnesia for colour names as well as for colour agnosia. In the material collected from the literature by Sittig there is only one case in which the colour imagery was not severely involved. It is significant that most of these patients are unable to find colours from memory of familiar objects with standard colours in spite of the strong association between colour names and names of objects (e.g., sky—blue, blood—red); the defect in colour imagery was also evident in our case, though only by an occasional slip. Colour imagery is like any other imagery based on past experience, and as such will have some part to play in colour recognition. Under some conditions more than others we have to refer to a standard colour, and have to go back to our colour imagery as a source of reference. Such reference will for obvious reasons not be necessary in the Ishihara test, nor will it be necessary

in the colorimetric testing where only equality and difference has to be decided. The same is largely true when two skeins have to be compared as to difference and equality as long as no conceptual procedure is involved. There is accordingly in colour agnosia no disturbance in the Ishihara or in colorimetric tests, and there are usually no great difficulties when skeins are compared as to difference and equality.

On the other hand, it will usually be necessary to refer to a standard colour by means of colour imagery in colour naming, selecting colours, and particularly in sorting of colours. Accordingly these are the tests in which the colour agnosias particularly fail. Finally there is the complaint of some patients of seeing the colours as different from before; since the colour imagery is absent or vague and there is no standard to refer to, the colour may appear unfamiliar and strange. Thus the main traits in colour agnosia can be explained by lack of colour imagery, and there does not seem to be any need to assume a perceptive disturbance for which there is no evidence.

A separate problem in our case is that of dominance. Our observation is an instructive and unique example of certain aspects of this problem. Handedness has been generally accepted as indicating the side of hemispheric dominance as far as higher cortical functions are concerned. It is well known that a person's laterality of dominance of eye, hand and foot may alternate. There is, however, no pathological material available which could give information about the distribution of dominance between the two hemispheres in cases where the laterality of dominance in the peripheral organs alternates—a question which is of some clinical importance. Our case demonstrates this relationship very clearly. There is no doubt about the laterality of dominance of eye and hand. He was definitely right-handed, and he had lost the sight of his right eye in infancy. The brain damage, as indicated by the visual field defect of his left eye, involved the right hemisphere and caused a clinical picture of visual agnosia disturbances. Such disturbances arise only in occipital lesions of the dominant hemisphere, and it can therefore be concluded that, in accordance with his monocularity, the right occipital lobe was in our case dominant for higher visual functions.

Acalculia and an amnesic aphasia is as a rule associated with visual agnosia; amnesic aphasia is particularly closely connected with colour agnosia. Wordblindness is almost regularly involved in visual agnosia apart from combining with colour agnosia. All these symptoms were from the beginning absent in our case. This suggests, as indicated by his right-handedness, that functions other than visual seem to have been dominated by the intact left hemisphere.

The absence of word blindness is of special interest. Reading is, as distinct from other visual functions, a function of language as well as a visual-gnostic function. Alexia is as regularly associated with aphasia as with agnosia, though different in type in the two conditions. In the period when the reading was being acquired by our patient the dominance of the right hemisphere for higher visual functions and that of the left hemisphere for speech had already been established. It was, therefore, the laterality for speech which prevailed and determined the laterality for reading. Reading is only one of many composite

faculties built up on more than one fundamental function. A similar split may arise for a number of composite functions in alternating dominance of this kind. The complexity arising from alternating hemispheric dominance and such functional interrelations might well account for some of the variations in the clinical symptomatology in cases where the presence or absence of symptoms is difficult to explain on anatomical grounds. It might also give some explanation for the shift of dominance of certain functions mentioned in the literature.

The brain damage in our case followed a cut of the right side of the neck without injury to the big arteries or veins. The complication apparently arose when the injured part was heavily compressed for a considerable period. Following this there appeared signs of intracranial pressure apart from the focal symptoms. The lumbar puncture showed increase in protein and raised pressure of the c.s.f. and there was an oedema of the disc. On subsequent controls the pressure of the c.s.f. and the protein gradually returned to normal, but the swelling of the disc still persisted during the observation period of nine months. Considering the restitution of the focal symptoms and the disappearance of the pathological signs in the c.s.f. a neoplastic process which might have coincidentally developed can be excluded. There were no signs indicating a rupture of a congenital aneurysm. It seems most likely that simultaneously with the compression of the neck the internal jugular vein was compressed. Although ligation of the jugular vein has, as a rule, no consequences, there are cases in which damage occurs and it seems that our case belongs to this group.

SUMMARY.

A right-handed, intelligent man, 32 years of age, was blind in the right eye since infancy. In a state of depression he attempted suicide by cutting the right side of his throat. It was necessary to compress the neck manually before sutures were inserted. Following this he had symptoms of increased intracranial pressure, and focal signs which consisted of a temporal field defect of his left eye and a temporary visual agnosia and cortical colour-blindness combined with colour agnosia. The brain damage was probably due to thrombosis of the venous system of the right occipital lobe. The agnosia symptoms have been studied during the restitutional phase and their character analysed and described. Noteworthy was the absence of symptoms which are usually associated with visual agnosia, particularly the absence of word-blindness. This could be explained by the presence of alternating dominance. It has been found that the visual functions in this case were dominated by the right hemisphere and other higher functions by the left hemisphere. The significance of such alternating dominance has been discussed.

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LITERATURE

ADLER, A., Arch. Neur. and Psych., 1944, 51, 243.
BAY, E., and LAUENSTEIN, O., D. Zischr. Nervhlkd., 1947, 158, 107.
BERINGER, K., and STEIN J., Zischr. Neur. u. Psych., 1930, 123, 472.
BRAIN, W. R., Brain, 1941, 64, 43.
FOERSTER, R., Arch. f. Opthm., 1890.
GELB, A., and GOLDSTEIN, K., Zischr. Neur. u. Psych., 1923, 83, 26.
LISSAUER, H., Arch. f. Psych., 1890, 21, 222.
POETZL, O., Die. Optisch-agnostischen Störungen, Leipzig, 1928.
POPPELREUTER, W., Zischr. Neur. u. Psych., 1923, 83, 26.
SITTIG, O., Mschr. Psych. u. Neur., 1921, 49, 63.
STEIN, J. and BUERGER-PRINZ H., Disch. Zischr. Nervhlkd., 1932, 124, 189.
STENGEL, E., J. Ment. Sci., 1948, 94, 46.
WILBRANDT, H., Zischr. f. Nervhlkd., 1892, 2, 361.
WOLPERT, I., Zischr. Neur. u. Psych., 1924, 93, 397.