Visual disorientation with special reference to lesions of the right cerebral hemisphere. By W. Russell Brain (From the Neurological Department, London Hospital) *Brain* 1941: 64; 244–272.

Writing during the Second World War, Russell Brain’s starting point is the clarity provided by examples of penetrating head wounds for understanding the specific effects of focal brain injury on visual disorientation, uncomplicated by diffuse pathology and uncontaminated by other perceptual disorders. He takes as his authority cases from the Great War described by George Riddoch and by Gordon Holmes describing failure to localize objects in space; inability accurately to judge relative length, distance or size and to-and-fro movement; failure of topographical orientation; and inability to bisect a line—but only in the context of biparietal lesions. From neurologists placed on the other side of the trenches had come the observations that visual field defects are usually present when the erroneous perceptions are exposed by comparison of two objects, whereas the fields are full when the visual disorientations are absolute. Each results in ‘optical ataxia’ for objects and ‘Optisch-raumliche Agnosien’ for space due to loss of visual engrams. In both situations, objects are often erroneously misplaced towards the midline because the fixation point shifts to a new central locus, thus moving the percept of objects towards the seeing half field. Against this background, Brain describes three cases in which a unilateral cerebral lesion results in visual disorientation in the opposite hemisphere, and three others having profound loss of route finding due to neglect of the left half of external space in association with right parietal lesions, allowing Dr Brain to speculate on the nature and classification of visual orientation and its relationship to awareness of the body. But, whereas Russell Brain extols the virtue of the penetrating brain lesion in dissecting these cases, his own material mainly comprises examples of tumour, abscess and stroke.

He describes H.L.L., the female victim of an air-raid, studied 4 months after a series of operative procedures for a penetrating wound of the left superior parietal lobule, who complains of walking into objects and difficulty in grasping objects on the right through inability to judge their distance. Unable to tell left from right or put on clothes, and with a tendency to invert letters when writing, she has no field defect but misplaces objects seen in the right half field to the left, and—conversely—indicates to the right of an object in the right half field when asked to point directly at it. One month after removal of a convexity meningioma, S.B. finds it impossible to judge the distance of objects seen in his intact left half-field but he can accurately divide a line, indicate the centre of a circle and show the relative length of three lines. In the 18 months during which symptoms and signs attributable to a glioblastoma that eventually proved fatal evolved, J.K.P. found that he localized and placed objects within arm’s length inaccurately in the left half field. For Brain, these examples of visual disorientation—H.L.L. and S.B. with lesions in the upper parietal and J.K.P. with one in the posterior temporal lobe—show that ‘although external space is finally presented to consciousness as one, estimation of ‘walking distance’ depends on neural links between the visual cortex and the leg area of the post-central convolution, while estimation of ‘grasping distance’ depends in a similar way upon association paths between the visual cortex and the hand and arm area’.

Now, he considers the concept of space in the congenitally blind leaning on the opinion of Pierre Villey who, himself blind from birth, distinguished manual, brachial and crural space—the cortex ‘counting’ the number and orientation of steps needed to reach a ‘seen’ object—as the domains that can be explored and reached by these body parts. It follows that visual disorientation is more likely to affect the sagittal (to-and-fro) than coronal (up–down and left–right) planes, and for the separate components of visual perception to become dissociated in disease—as the three cases described here reveal. Put another way, the ability to differentiate two points on the retina that define distance in the coronal plane, akin to sensory two-point discrimination, is dependent on activities of the primary visual cortex, much different from the higher order processing of images from the two eyes, proprioceptive information from the ocular muscles and motion awareness—in short, a whole raft of perceptual and ideational data—that must take place, in order to judge relative distance in the sagittal plane. But there is a further subtlety arising from the fact that two objects apparently close may in reality be widely separate if far away: therefore, information available in both the sagittal and coronal planes has to be integrated, in order to derive a composite perception of where objects are in space; and this central processing is lateralized to each hemisphere for the opposite half-field.

C.R.C.S.—also suffering from a right hemisphere glioblastoma resulting, eventually, in a left homonymous hemianopia—turns erroneously to the right when trying...
to negotiate familiar spaces: after a right hemisphere cerebral haemorrhage, K.W.N. also invariably turns to the right when walking around her home despite recognizing and describing the correct landmarks; E.Y., brewing a cerebral abscess that was subsequently removed successfully by Mr Douglas Northfield, always takes the right side of two options—doors in his home, beds in his bedroom—even though left is correct for the task in hand. Although these three cases each have left-sided field defects, their visual disorientation occurs with intact topographical memory, and no loss of right–left orientation or visual object agnosia: ‘evidently we have to do with a disturbance of spatial orientation and not with a mere inability to see normally…patients who arrived at the wrong door recognized that they had made a mistake’.

Brain opens his discussion with a previous interpretation of this rarely described symptom. As with the aphasic who loses the automatic construct of a sentence in the realm of speech and inserts erroneous words, in these patients a centrally represented route normally taken in familiar surroundings becomes disrupted and contaminated by others, equally familiar but inappropriate for the occasion. Rather, he prefers a simpler account: ‘the effect of a lesion of the posterior part of the right hemisphere is to cause the patient to neglect the left half of external space…’, and hence the errors are systematic and not merely the result of random mistakes in route finding. But Dr Brain notes that all six patients also have, to a greater or lesser extent, some defect of sensation on the left. Since each half of the body is delineated from external space both by visual and sensory frontiers, visual localization requires objects to be placed in the context of self and the external world, and to be integrated with an awareness of the body. Anatomically and physiologically, the parietal lobe links the cortical receptor zones primarily responsible for vision and sensation. Hence, vision and sensation are both likely to be affected by parietal lesions. Just as the patient with disordered perception of the left half of the body, disconnected from the security of an intact body scheme, may forget the left side of the body and either needs to be reminded of its presence or resolutely denies that it exists, so the patients here described neglect the left half of external space. Such damage ‘to the scheme for one half of the body causes events occurring on that half…to be related in consciousness to the surviving scheme representing the normal half…[and] damage...for one half of external space causes the remaining half to exert a dominant influence in the choice of routes’.

In passing, Brain notes that two of his patients show apraxia for dressing—‘a symptom which does not appear to have been specifically described hitherto…though no doubt it may occur as one manifestation of a more general ideational or motor apraxia’. H.L.L. and C.R.C.S. each have to work out from careful deductions which is the left and which the right side of their clothing, or the top and the bottom. In that sense, failure to relate the orientation of clothing to the body image is an ideational error. Whereas a lesion in either hemisphere that disorganizes the body scheme, may cause apraxia for dressing and result in a variety of visual perceptual deficits, Brain suggests that visual neglect is a newly recognized cognitive deficit that localizes to the right hemisphere. And cerebral dominance for higher order visual functions does not necessarily track that for others: thus, ‘symbolic thought and expression, visual recognition of objects, visual orientation in space, topographical memory…and awareness of the body are functions which behave independently of one another in relation to hemisphere dominance’. Therefore, it becomes necessary to be more precise in referring to the symptoms of visual disorientation.

There are defects of visual localization in space, not explained simply by field defects, in which the integrative component of visual perception that relates spatial relations of objects seen is at fault—the space-blindness of Kleist, the geometrical object-agnosia of Pötzl—resulting from upper parietal lesions for which there is no hemisphere dominance: there are defects of stereoscopic vision as described by George Riddoch and by Gordon Holmes; there is right hemisphere dependent left sided visual neglect, here emphasized but not previously recognized, corresponding to impaired sensory perception of the left half of the body and resulting from lesions of the right parietal lobe; there is visual allaesthesia in which part of an image is transposed from right to left or from the upper to lower sectors of one-half field; there is loss of topographical memory distinct from the right-turning of visual neglect; and there is visual disorientation secondary to visual object-agnosia. ‘Severe damage to the central processes of space-perception on the visual side deranges the whole basis of orientation…and patients with visual disorientation are far more severely handicapped than a blind man, and lack a blind man’s capacity to orientate himself by other senses…our awareness of [space] is not just a mere juxtaposition of sensory impressions from which some can be deduced without impairing the validity of the remainder: it is a synthesis from which springs a unity of perception corresponding to the external reality’. Thus, in his influential paper from 1941, Russell Brain explores the neurology of blindness and neglect—topics to which we return in papers published in the present issue.

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