

## GLOBAL SPATIAL DISORIENTATION

### Clinico-Pathologic Correlations

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### SUMMARY

Two patients presenting with the acute onset of bilateral parietal lobe damage showed initially the features of Balint's syndrome. After most of its manifestations had cleared, both patients exhibited severe disorders of spatial orientation: acoustic ataxia, inability to localize objects in space. In addition, 1 patient showed a topographical disorientation and abnormalities of whole body movements. This patient came to post-mortem examination, which revealed bilateral and fairly symmetrical old and recent infarctions of the superior parietal lobules.

In the light of these observations and previous reports, it is suggested that the reported abnormalities of whole body movements can be explained on the basis of a visuo-motor intrahemispheric disconnection due to the bilateral lesion of the dorsal parietal lobe. In addition, the global spatial disorientation is analyzed and is thought to be the manifestation of a derangement of a specific function centered in the parietal lobe. Moreover, it is pointed out that topographic disorientation does not require the concomitant failure of oculomotor mechanisms for its production.

The topography of the anatomical lesion is considered in relation to current concepts on brain damage after severe hypotension and cardiac arrest. It is concluded that this group of patients is most likely at risk of developing behavioural abnormalities akin to the ones here reported.

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### INTRODUCTION

The term "Visual Disorientation" was introduced by Holmes (1918) to refer to a defect in spatial localization of objects and their distances by sight. A number of

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additional behavioural defects have been reported among the so-called "disorders of spatial orientation" (Benton 1969) and include: (a) inability to localize objects in space and to estimate their size and distance from the observer; (b) impaired memory for the location of objects or places; (c) inability to trace a path or follow a route; (d) reading and counting disabilities; (e) incapacity to relate spatially separated objects or events to each other; (f) visuo-constructive abnormalities.

In a number of the reported cases, these abnormalities in spatial orientation have been associated with severe oculomotor disturbances (Holmes 1918; Holmes and Horrax 1919). The underlying pathological process in this last group has always been a bilateral lesion of the posterior half of the brain. On the other hand, there have been reports of "pure" forms of spatial disorientation, without concomitant oculomotor abnormalities (Meyer 1900; Paterson and Zangwill 1945). In these, the responsible brain lesion has been clinically localized as unilateral in the right temporo-parietal area or bilateral affecting the occipital lobes.

We are reporting 2 cases which showed a combination of permanent spatial disorientation with transient oculomotor disturbances. One of them came to post-mortem examination allowing us to establish the anatomical bases of the permanent behavioural abnormalities. Also, some ideas on the probable pathogenesis of such disturbances are discussed.

#### REPORT OF CASES

##### *Case 1 (M.V.V. (HSR No. 141002))*

A 63-year-old right-handed female, with 6 years of primary education, was first seen in our neurology service (Santiago, Chile) in December, 1973. In 1967, a carcinoma of the cervix had been diagnosed and treated with radiotherapy, without recurrence.

On the evening of 26 December, 1973, the patient suddenly complained of severe diffuse headache followed by profuse vomiting and several generalized seizures. On admission to the Emergency Ward, her pulse was regular (80/min), BP 180/100 mm Hg, temperature 35 °C and she had regular respirations. Her carotid pulses were full and without bruits. The general physical examination was otherwise unremarkable. Neurological examination: she was in deep coma with a weak response to noxious stimuli and with pinpoint pupils which contracted weakly to light. The eyes were conjugately deviated to the right and showed an incomplete deviation to the left on the doll's head manoeuvre. The 4 extremities were hypotonic with depressed deep tendon reflexes and bilateral flexor plantar responses. She could, however, move all 4 extremities in a grossly symmetrical way. A first lumbar puncture was traumatic (hemorrhagic CSF, clear after centrifugation, with abundant fresh red blood cells).

On examination 24 hr later, she remained in coma but showed some responses to noxious stimuli. The face was symmetrical, the ocular deviation to the right had disappeared and the extraocular movements were full on the doll's head manoeuvre. She could move both lower extremities (withdrawal) in response to painful stimuli, whereas the upper extremities moved barely with the same stimuli. The deep tendon reflexes were still diffusely depressed and the plantar responses remained flexor. A second lumbar puncture yielded clear CSF with a protein content of 37 mg/100 ml, glucose of 67 mg/100 ml and 5 mononuclear cells/mm<sup>3</sup>. By the third hospital day, she became alert and cooperative. Her speech was normal without paraphasic errors, and she could follow commands and repeat to dictation without mistakes. However, a striking abnormality could be detected in her visuo-motor behaviour. Her eyes were constantly moving in a slow and unpredictable wandering way, and when an object was put in the periphery of her visual field, she acted as if she could not see it or could not fix her eyes on it. When her attention was called to this peripherally located object, she would attempt its localization by moving her head more than her eyes in a wandering fashion until she would finally "see" the object after variable intervals, identifying it properly. She was unable to follow a slowly moving object with her eyes, but her extraocular movements were full in all directions on com-

mand. She thus behaved as if she were having the so-called "psychic paralysis of visual fixation", a disturbance which, together with "optic ataxia" and "visual inattention", forms the triad of Balint's syndrome (Balint 1909).

In addition to this abnormal behaviour regarding extraocular movements and visual pursuit functions, she also showed features which suggested visuo-motor incoordination. When the patient was asked to touch an object held in front of her with either hand, she first had to fix her vision on it, then she tried to touch the object. However, she consistently failed because her hand would stop a few cm in front, behind or to the side of the object, but never close enough to touch it. This contrasted with her perfectly normal ability to touch different parts of her body with eyes closed. In addition, her motor coordination was also normal, without evidence of cerebellar ataxia.

Another deficit she showed at this stage was an inability to perceive visually more than one object at a time, an abnormality which has been considered to be a visual attention defect in patients with visual disorientation (Godwin-Austen 1965). This abnormal behaviour contrasted with a normal visual acuity and fields, and full extraocular movements.

In addition to the abnormalities in the visually-mediated motor acts, she was also unable to localize properly or grasp an object guided by its characteristic noise with eyes closed. When a key-chain or whistle were the auditory stimuli, she would orient her head and hand in the wrong direction, even to the opposite side. These abnormalities occurred in the setting of normal auditory function under bedside testing conditions: symmetric aerial and osseous auditory perception, non-lateralized Weber's sign and no extinction on double simultaneous stimulation.

The motor strength, tone, deep tendon reflexes and plantar responses were normal and symmetrical, as were the superficial and deep sensory functions. Stereognosis was preserved, but she showed an abnormality in the perception and integration of her body-scheme in the form of a left somatoagnosia. She also had a dysgraphia and constructional apraxia. The verbal arithmetic calculation, right-left orientation and finger order sense were preserved. Her reading abilities for individual words and short sentences were spared.

When she started walking 3 weeks later, a different set of abnormalities became apparent. When walking, she would behave as a blind person. Her sight would be directed beyond the immediate obstacles, her right arm would be stretched in front of her to detect obstacles, and she would bump frequently into things such as chairs, tables, etc. When she was asked to identify these objects, she would name them correctly without hesitation, showing an absence of agnosic features in the visual sphere. She had a severe spatial disorientation that was apparent in most of her restricted activities. When placed in the corridor, she was unable to find her way back to her room, and would take several different paths in all possible directions until she would finally take the right one. Being in her room (which had 6 beds), she was unable to find her bed. When she would finally get in it, she would lie in the wrong orientation with respect to its long axis, even in a transverse position (Fig. 1). After many unsuccessful attempts to correct her position trying several different approaches, including a completely upside-down one, she would react catastrophically.

When she was placed in front of a chair (which she would immediately recognize as such) and asked to sit down, she would hesitate and then get in several abnormal sitting positions. These included sitting facing the back of the chair, sitting on the very edge of it, and sometimes "sitting" in the air by



Fig. 1. Case 1. Abnormal whole body movements on attempting to get in her bed, lying down perpendicularly to the long axis of the bed.

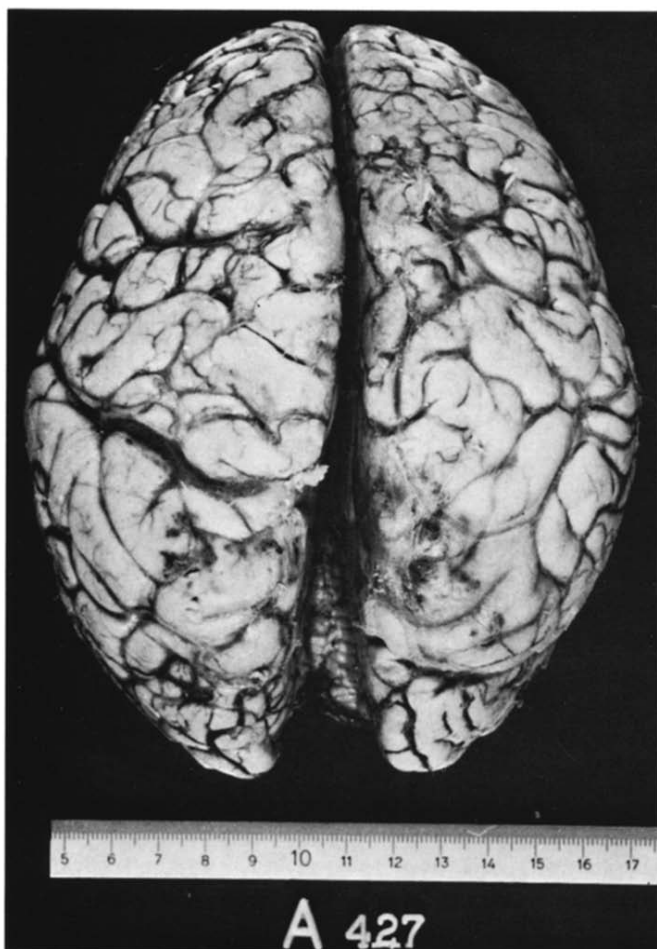
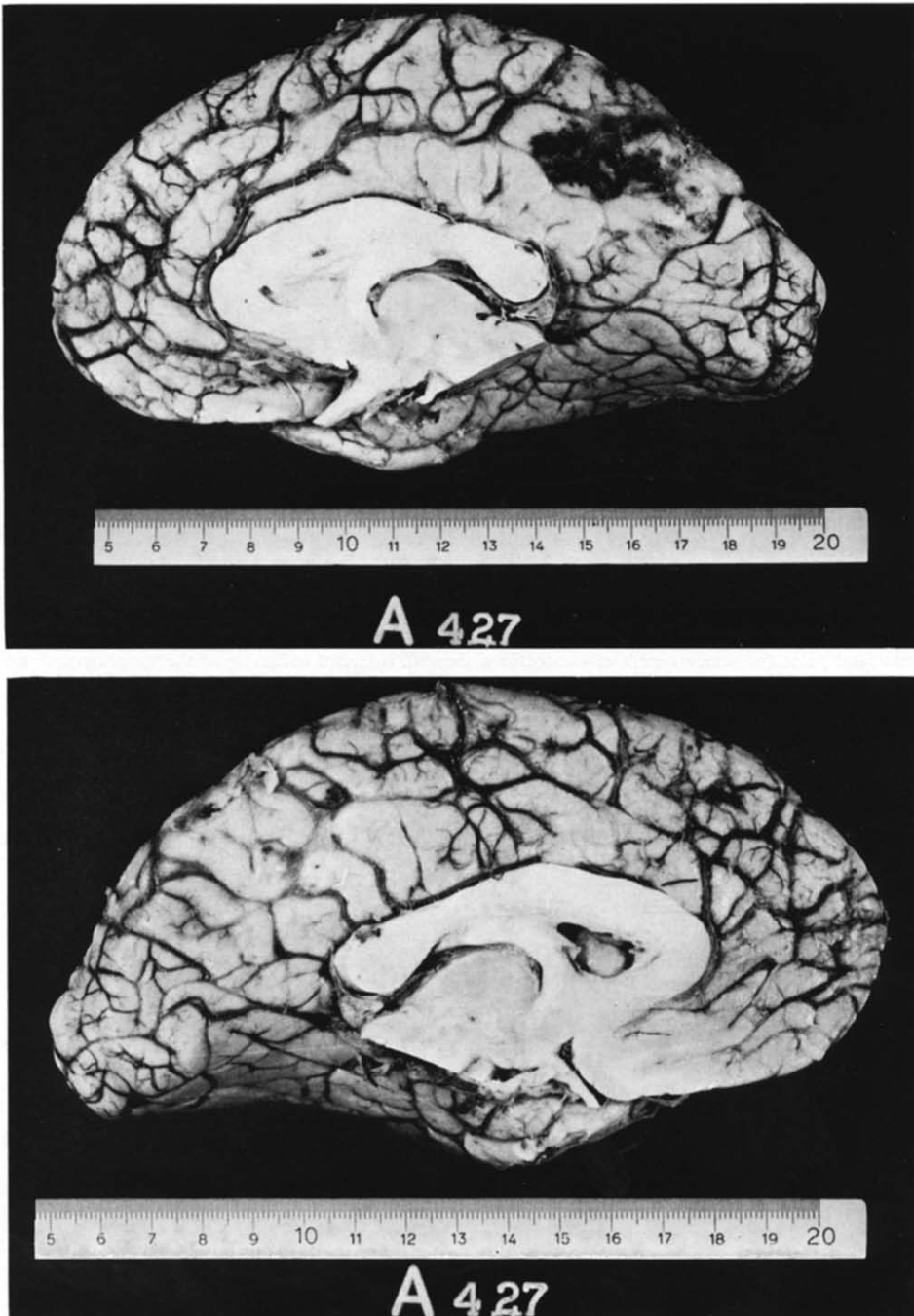


Fig. 2. View of the brain from above, showing bilateral dorso-medial parietal lobe infarction, hemorrhagic medially and pale laterally.

the side of the chair, occasionally falling down. At this stage she was able to name two or more different objects simultaneously presented to her, but she had trouble in determining their position relative to each other.

Her appreciation of lengths (a few cm long objects) or distances (several meters) was adequate. Her orientation in time, memory for recent and remote events, general judgement capacities and general behaviour (excluding the activities that essentially require spatial orientation) were within normal limits. A standard electroencephalogram (EEG) showed diffuse slow wave (theta) activity without focal abnormalities or spike discharges. Urinalysis, blood glucose and BUN were normal.

She was discharged after 1 month in the hospital and at that time the symptomatic triad of Balint's syndrome had cleared. The constructional apraxia and left somatoagnosia had also disappeared, but the abnormalities related to spatial disorientation were unchanged. This disturbance was still present 2½ months after discharge. When walking in the streets, she would frequently lose her way to familiar places or her way back home. This incapacity was disabling to the extent that it made her refuse to go out unaccompanied. On examination, she was still unable to give the relative spatial location of several different objects presented to her simultaneously. Her inability to find her way inside the hospital, inside the examining room, or to position her body on the examining stretcher was unchanged.



**Fig. 3.** Extension of the infarcts on to the medial aspect of the hemispheres. Above: right hemisphere, below: left hemisphere.

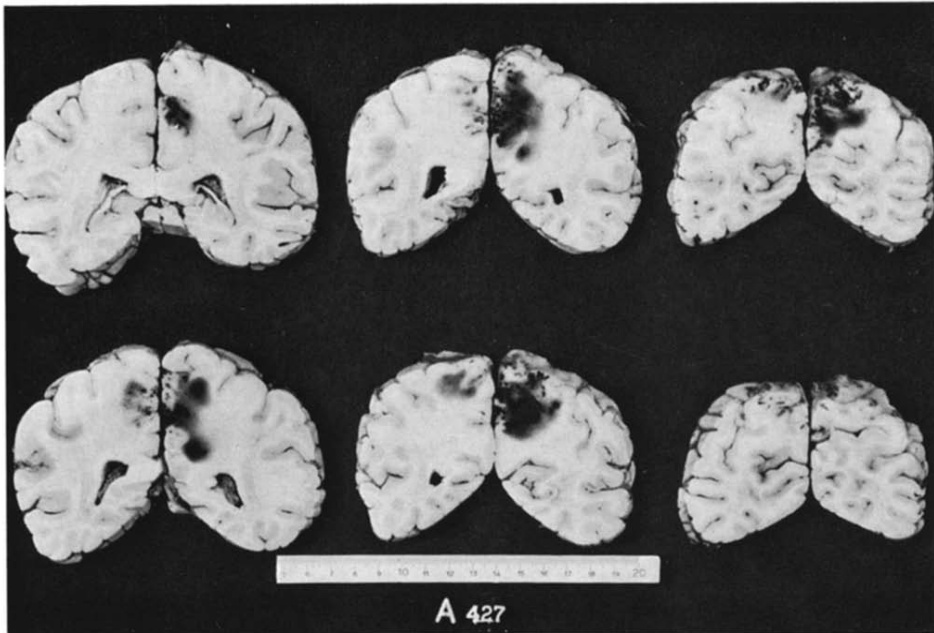


Fig. 4. Coronal sections of the hemispheres, from the splenium of the corpus callosum towards the occipital poles (posterior aspect of the sections shown). Bilateral softening of the superior parietal lobules, more extensive on the left, hemorrhagic on the right.

Being in this state, 4 months after her initial illness, one afternoon she suddenly complained of severe diffuse headache and collapsed. On admission she appeared in deep coma without reaction to painful stimuli. Pupils were 4 mm in diameter and reactive. There was no movement in the extremities and she had bilateral Babinski signs. She died 24 hr after admission.

*Post-mortem study (Autopsy A-427)*

*General autopsy*

The relevant findings were: cardiac hypertrophy, bilateral confluent bronchopneumonia, and widespread arterio- and arteriolosclerosis.

*Central nervous system*

*Gross.* Upon removal of the brain, the dural venous sinuses appeared normally patent. The fresh brain weighed 1,200 g. The arteries at the base of the brain showed moderate atherosclerosis.

On external inspection, the cerebral hemispheres showed a bilateral softening of the parietal lobes, more on the left, with a distinct hemorrhagic component (Fig. 2). On both sides, the infarcted area ended abruptly at the level of the parieto-occipital fissure, both on the medial and lateral aspects of the hemispheres, leaving the occipital lobes completely intact. The anterior margin of the infarct was at the level of the callosomarginal fissure on the right and 1 cm in front of this fissure on the left, involving medially the gyrus cingulus and all the precuneus on the right, and the posterior one-third of the paracentral lobule, the whole precuneus and the posterior one-third of the gyrus cingulus on the left (Fig. 3). On their lateral aspects, the infarcts involved the whole extension of the superior parietal lobules, sparing completely the angular and supramarginal gyri.

The coronal cuts of the cerebral hemispheres showed the large infarcts on the medial and lateral surfaces of both parietal lobes, which appeared hemorrhagic medially and pale on the periphery (laterally). The hemorrhagic component was more prominent on the right parietal lobe. The sections made from a level anterior to the splenium of the corpus callosum towards the parieto-occipital junc-



**Fig. 5.** Superior parietal lobule cortex. Extensive necrosis of the middle layers ("pseudolaminar"), infiltrated by macrophages, with capillary neoformation. Old necrosis of adjacent areas, with heavy astrocytic reaction. Hematoxylin and eosin,  $\times 80$ .

tion disclosed successive involvement of the following structures in an antero-posterior direction: gyrus cingulus bilaterally, posterior one-third of the paracentral lobule on the left, precuneus bilaterally and superior parietal lobule bilaterally (Fig. 4). The white matter softening at these levels was less prominent and was restricted to a band of infarcted subcortical white matter without signs of cavitation.

The more posterior sections which included the confluence of parietal, temporal and occipital lobes, showed the infarcts restricted only to the parietal lobes, without involvement below the level of the parieto-occipital fissure. On the other hand, the sections showing the lateral aspects of the infarcts disclosed lesions restricted only to the superior parietal lobules, never reaching beyond the intraparietal sulcus towards the angular and supramarginal gyri, which appeared spared in their complete antero-posterior extension.

**Microscopic.** Sections from multiple areas of cerebral cortex, white matter, basal ganglia, cerebellum and brain stem were stained with hematoxylin-eosin and some of them with the Verhoeff-Van Gieson technique for elastic fibres. The microscopic lesions were confined to the areas macroscopically affected, and they consisted of areas of cerebral infarction which were of two different locations and ages:

(A) One type of lesion appeared in the form of exclusively cortical infarction with a "pseudolaminar" distribution affecting layers 3 and 4. The neighbouring cortical layers above and below this necrotic band showed vacuolation of the neuropil, disappearance of the neuronal elements and abundant proliferation of reactive "gemistocytic" astrocytes (Fig. 5). This lesion represents areas of infarction of at least 2 months' evolution. This type of infarction was restricted to the affected areas described macroscopically. The sections from the inferior parietal lobules and occipital lobes disclosed a normal cortex and white matter.

(B) The second type of lesion consisted of much more recent hemorrhagic infarcts confined to the paramedian aspects of the areas already described. These appeared as foci of necrosis of the cortex and subcortical white matter, which were moderately infiltrated by polymorphonuclear leukocytes and showed abundant foci of perivascular collections of extravasated red blood cells. This type of infarct was located at the paramedian aspects of the parietal lobes bilaterally and represented a 3-4-day-old hemorrhagic infarction. In multiple sections examined we could not find evidence of any kind of embolic material occluding arteries of the affected areas or elsewhere.

#### Case 2 (M.M.P. (HSR No. 141554))

A 28-year-old right-handed female with 2 years of secondary education, who had always enjoyed good health.

On 19 September, 1973, a cholecystectomy was performed under general anaesthesia in an outside hospital. In the immediate postoperative period she hypoventilated for about 30 min and then had a cardiorespiratory arrest of unknown duration, from which she recovered with open-chest cardiac massage. She was left in deep coma, flaccid and with bilateral Babinski signs. Over the following days her state of consciousness progressively improved, and after 3 weeks she was able to cooperate for physical rehabilitation. Three months after the cardiorespiratory arrest she was sent to our neurology service in Santiago (Chile) for evaluation.

On neurological examination, she was alert and well oriented as to time, place and person. Her speech was normal in articulation, comprehension, repetition and intonation. She had a marked dysgraphia and constructional apraxia. However, she did not disclose a fixed pattern of inattention for a particular part of the visual space. Her visual acuity was hard to test, but she was able to point to the presence of a 1 mm diameter dot at 30 cm distance. The visual fields were reduced (about 15 to 20°) in a concentric way bilaterally. Verbal arithmetic calculation, right-left orientation and finger order sense were preserved.

The characteristic features of Balint's syndrome were present. When an object was placed in front of her, she showed a long delay in orienting her gaze towards it. She searched for the object with the use of head movements rather than eye movements. When she was asked to touch with either hand the object she had already seen, her performance was very inaccurate, as she would direct her finger to the sides of the object, never being able to touch it correctly. This contrasted with her performance on the finger-to-nose test for motor coordination, in which she would always touch her nose accurately but not the examiner's finger. She also had an occasional visual agnosia for objects, which were readily recognized by palpation.

Both the visual object-agnosia and the elements of Balint's syndrome cleared over the following weeks, leaving her with a distinct defect in the visual perception of spatial relationships. She was unable to establish the relative position of several objects or figures, both in vertical and horizontal arrangements. On the other hand, she also had a clear disturbance in the spatial integration of auditory stimuli, despite normal and symmetric auditory perception as tested by routine bedside procedures. When the sound of a familiar object was produced around her and she was asked to keep her eyes closed and point to the place from which the sound was coming, she would always point in the wrong direction, even opposite to the sound.

In contrast with these striking abnormalities, her topographic sense and orientation were well preserved, as were her stereoscopic vision and estimation of object sizes. Her ability to orient the long axis of her body properly in a stretcher or bed was also preserved. The rest of the neurological examination was normal, except for very mild disturbances in the tactile recognition of objects. Her gait was slightly broad-based and hesitant but did not show disturbances suggestive of visual inattention.

Laboratory tests of the blood and urine were normal. The EEG disclosed occasional and diffuse theta-wave discharge.

Her neurological examination 6 months after the cardiorespiratory arrest showed that the only permanent deficits left were the visual and acoustic disturbances in spatial orientation.



## DISCUSSION

Both our patients presented with fairly similar clinical pictures, although of different severity. They initially presented with the features of Balint's syndrome. At a second stage, as the former elements cleared, they showed severe abnormalities in spatial orientation which persisted unchanged for long periods of time.

### *Abnormalities in spatial orientation*

Different forms and degrees of spatial disorientation were prominent clinical features in our patients. They both showed a striking inability to appreciate the spatial relationships of several objects in horizontal or vertical arrangements. This was observed at a time when their extraocular movements were normal (voluntarily and on pursuit), their visual fields were full and their naming abilities for common objects were intact. Although the testing of the above functions was performed at the bedside without instrumental recording of extraocular movements, it seems reasonable to assume that the basic perceptual visual functions were essentially intact. This suggests that the reported abnormality represents an agnosic deficit with specificity for spatial relationships.

The second variety of spatial disorientation shown by both patients consisted in their inability to localize and grasp an object guided by its characteristic noise, the so-called "acoustic ataxia". It occurred despite grossly normal audition and ability to recognize the object by its sound with eyes closed. Due to technical limitations, our auditory testing conditions did not include audiograms and binaural testing. However, we believe that the localization deficit is not due to auditory imbalance. Although auditory localization defects do occur with bilateral hemispheric lesions, they are located in the vicinities of the auditory cortex in the temporal lobes (Doig 1972). In our Case 1 the only areas involved pathologically were the parietal lobes. On the other hand, it is unlikely that our second case had bilateral temporal lobe involvement, as this situation, when associated with auditory perceptive disorders, is accompanied by overt language problems (Lhermitte, Chain, Escouffle, Ducarne, Pillon and Chedru 1971). In addition, this patient did not show other signs of temporal lobe involvement, such as memory defects (Scoville and Milner 1957).

In our cases and in 1 of Holmes' (1918), "acoustic ataxia" was present together with gross disturbances of orientation in the visual sphere. We regard it as another non-specific manifestation of these patients' disturbances in the perception of spatial relationships. According to Critchley (1953), this function normally requires the interplay of multiple sensory data, mainly visuo-tactile but also auditory, vestibular and olfactory. The integration and coordination of these sensory data in the intact parietal lobes leads to a normal spatial perception. In the setting of parietal lobe damage, the loss of this multisensorial integrative function produces the spatial disorientation which we propose should more properly be called "global spatial disorientation" on account of the multiplicity of the sensory data involved.

One of the most striking of the forms of spatial disorientation here reported was the topographic disturbance shown by our Case 1. She became disoriented in both

familiar and unfamiliar surroundings, thus resembling similar cases reported by Meyer (1900) and Paterson and Zangwill (1945). Their cases (1 in Paterson and Zangwill's and 2 in Meyer's reports) were clinically diagnosed as having sustained right temporo-parietal, right occipital and left occipital lesions, respectively. The pathologically verified third case of Meyer's had bilateral occipital lesions. Brain (1941) has stated that topographic disorientation is an abnormality that arises independently from other forms of visual disorientation and from visual object-agnosia. Our case seems to prove this contention, as this abnormality was present at a time when the oculomotor disturbances and most of the visual disorientation features had already cleared.

With regard to the pathologic bases for such disturbances, our case showed a sharply demarcated area of old infarction which was restricted to the medial and superior portions of both parietal lobes, with sparing of the inferior parietal lobules and occipital lobes. At the time of her death, the remaining behavioural abnormalities were the global spatial disorientation and the topographic disorientation, in addition to abnormalities in whole body movements, which will be considered in the next section. Our pathological findings contrast with Holmes' (1918) with regard to the role played by the inferior parietal lobule and occipital lobe in these disturbances. His conclusions on the location of the bullet-produced lesions were mostly based on craniometric estimations, in relation to the entrance and exit points. On the basis of that information, he concluded that the most constantly injured areas were the inferior parietal lobule bilaterally and, to a lesser extent, the occipital lobes. From our data we can conclude that the involvement of these areas is not a pre-requisite for the existence of topographic and global spatial disorientation, which may occur on the sole basis of upper bi-parietal disease.

#### *Abnormalities in whole body movements*

Our Case 1 presented a very striking abnormality when asked to perform acts such as sitting in a chair or lying in her bed. These acts were slow and hesitant, but essentially correct in the several intermediate stages, becoming grossly abnormal at the moment of their adaptation to the visual stimulus (chair or bed). This contrasted with her ability to perform normally in other acts requiring axial body movements, such as walking or standing from the sitting or recumbent position. Similarly preserved were her abilities to grasp with either hand objects placed in front of her, and to adopt different body postures on verbal command. This kind of abnormality is rarely observed in clinical practice and we have been unable to find detailed descriptions of cases like ours in the literature.

The abnormal whole body movements in our case corresponded to poor adaptation to the visually-perceived stimulus towards which they were directed. One explanation for such a defect would be one similar to Balint's "optic ataxia", which has been interpreted as due to a disconnection between visual and frontal motor areas (Holmes 1918; Rondot and de Recondo 1974; Boller, Cole, Kim, Mack and Patawaran 1975). However, in our case the visuo-motor incoordination involved axial rather than limb movements. Its pathogenesis could also be postulated on the basis of a disconnection between visual and frontal motor areas produced by the upper bi-parietal

lesion. However, the anatomical substrate for the performance of normal axial movements is not at all clear. Geschwind (1965, 1975) postulates that the main efferent pathway for axial movements would be different from the mostly pyramidal control of independent limb movements and could be represented by the bundle of Türck. In the macaque, it originates from the posterior aspect of the superior temporal gyrus and ends in the vermis of the cerebellum, through an intermediate stage in the dorso-lateral pontine nuclei (Peele 1961). Although this is a well-known structure in primates, its existence is controversial in man. However, it could be speculated, along with the arguments given by Geschwind (1965), that our patient's remarkable axial motor abnormality was due to an interruption of the connections between visual cortex and the posterior aspects of the superior temporal gyrus bilaterally. Such an isolation of the latter structure from visual but not from auditory inputs would account for her specific deficit. However, this selective involvement of "visually-guided whole body movements" with sparing of her abilities to name common objects and read short sentences, would imply that the pathways from visual cortex to superior temporal gyri (and thence to Türck's bundle) would be different from the commonly accepted ones that subservise visual-verbal connections (visual cortex, visual association areas, angular gyrus, Wernicke's area) (Geschwind 1965). The bilateral sparing of the inferior parietal lobule in our case would thus account for her preserved naming and reading abilities. The selective involvement of both superior parietal lobules associated with her abnormal "visually-guided whole body movements" could mean that the pathways normally subserving this last function travel from visual cortex via the superior parietal lobule to the superior temporal gyrus bilaterally. This speculative proposition clearly requires confirmation with further detailed clinico-anatomical studies and experimental observations.

#### *Pathogenesis of brain damage*

The clinical and pathological data in Case 1 are suggestive of brain damage of a vascular aetiology, but the clinically suspected embolic origin of the lesions could not be pathologically verified. If the hemispheric lesions in this case are analyzed from the standpoint of their topographic distribution, they closely correspond with the lesions usually found in cases of so-called "low-flow infarcts" produced by profound systemic hypotension or cardiac arrest (Mohr 1969). In this situation, Lindenberg (1963) has proposed the existence of a topographic hierarchy of susceptibility to ischaemic damage of the brain, the first areas to be involved being the dorsal parietal regions, in relation to the borderzones between the middle and posterior cerebral arteries.

From these data we can assume that our Case 2, who sustained bilateral brain damage after cardiac arrest, most likely presented a pattern of bi-hemispheric infarction similar to the one pathologically verified in Case 1. Although they differed in the severity of the clinical abnormalities shown, they were essentially comparable regarding the type of behavioural disturbances presented. On the basis of their clinical and probable anatomical similarities, it can be concluded that "global spatial disorientation" probably represents a syndrome of upper bi-parietal damage. This calls for the

necessity of careful behavioural studies in the growing population of patients surviving profound hypotension and cardiac arrest in the present era of improved resuscitative facilities.

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