Conflict and integration of spatial attention between disconnected hemispheres

The article by Dr Ishiai and colleagues' contains a critical piece of anatomical data (that is, the “sparing of the rostrum and the inferior half of the genu” (fig 1)) which has been ignored by the respected authors as to its probable role in underpinning the results they reported. This may have occurred because of their conventional theoretical perspective on motor control in the human, in the light of which such matters might be afforded. However, the doctrine of contralaterality of movement control (CMC) in humans has been recently revised1 to account for observations such as theirs in the sensory realm, as well as others in the motor realm that underpin the classical lateralised index of contralateral hemiplegia/ipsilateral apraxia (with and without speech deficits); ipsilateral (non-dominant) weakness in lesions affecting the major hemisphere; and non-dominant weakness in lesions affecting the callosal. The one-way callosal traffic theory (underpinning lateralities of executive functions) states that all voluntary actions involving the non-dominant hand entail a sequential activation (that is, major followed by minor hemisphere, mediated through the callosal) of a devoted neuronal aggregate distributed between two hemispheres. The commands for the effectors on the dominant side reach them directly whence they arise. Those for the non-dominant effectors traverse the callosal.

Evidence favouring this scheme is overwhelming, as detailed elsewhere.1 Thus the improvement of performance seen upon the voluntary movement of the non-dominant side reflected the activating effect on both hemispheres when the left hand moved voluntarily, temporarily “lightening up” the dormant right hemisphere through the remaining functioning callosal connections depicted on MRI. The new scheme, therefore, bypasses the controversies mentioned by the authors.

It is to be noted that this scheme relates to all movements, whether cranial (such as eye movements) or appendicular. Anyone in doubt of the veracity of this claim can find solecism when they hear a double click upon snapping their fingers simultaneously, instead of one click mandated by CMC doctrine, with the second click arising from the neural (as opposed to ostensible) non-dominant hand (Derakhshan I, unpublished data). The callosal delay imposed on the non-dominant hand has been known for 160 years, under the name Melody Lead of the right hand of piano players, and is thought to represent artistic expression.1 Its range, however, (10–40 ms) makes that interpretation highly unlikely. Callosal delay when turning the eyes to the non-dominant side has long been documented.9 However, for the reasons alluded to earlier, more modern workers have ignored it, some calling it “idiosynchratic”: an interpretation inconsistent with the consistency it shows itself when sufficient data are given.1

References
4 Pirzio B, Hansch EC, Oculomotor reaction time in dementia reflects degree of cerebral dysfunction. J Neurol Neurosurg Psychiatry 1981;44:349–51 [see fig 1].

Authors’ reply

As Dr Derakhshan points out, MRI showed that the callosal lesion of our patient spared the rostrum and the inferior half of the genu. In our paper, we did not comment upon the role of partially spared callosal fibres in the dissociated line bisection performance between the right and left hands. At least in the sphere of praxis, the dominant left hemisphere hardly seemed to contribute to the control of the left hand, as our patient showed severe apraxia of this hand. However, the use of the non-dominant left hand to bisect a line would possibly have activated the ipsilateral left hemisphere as well as the contralateral right hemisphere, because of command transmission through the spared callosal fibres. If so, why would the use of the right hand not activate the ipsilateral right hemisphere (with and without speech deficit); ipsilateral apraxia of the right hand would activate not only the ipsilateral hemisphere (with and without speech deficit); ipsilateral apraxia of the right hand but also the right “spatially dominant” hemisphere. In our patient, rightward errors with the left hand were achieved almost always after placement of the subject midpoint. The latter finding was considered to represent an interhemispheric conflict of attention. By contrast, accurate rightward searches contralateral to the direction of the task was followed by one or two rapid rightward searches contralateral to the display. This rapid improvement might be explained by transmission of attentional information through the spared callosal fibres. Therefore, we prefer to reject that explanation, as interhemispheric callosal transmission should be bidirectional.

While the right hand and the left hemisphere bisection a line, the left visual field and the right hemisphere perceived the longer extent to the left of the fixation at which the mark was placed. We do not consider that such perception of the right hemisphere was transmitted to the left hemisphere through the subcortical neural connection. In patients with callosal disconnection, the gaze direction is unitary, and the direction and distribution of attention may be integrated under most ordinary conditions.3 When responding with the left hand, the right hemisphere may integrate attention to the extent perceived in the left and right visual fields. The capacity of the “intact” left hemisphere to integrate spatial attention remained, together with other callosal lesion in the subcortical white matter of the cingulate gyrus.

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References
result it is a little disjointed but this has been addressed by grouping the papers under the headings above. It is neither a comprehensive review of vasospasm nor is it meant to be. It is nevertheless a very interesting and informative book and does give a very useful insight into the direction in which research is heading. It is therefore very helpful for researchers in the field and would make a useful addition to a neurosurgical or neuroscience library.

**Kevin O'Neill**

**Contemporary approaches to the study of hysteria, clinical and theoretical perspectives**


This book ranges more widely than its title implies, including sections on somatisation disorder, factitious disorder, and malingering. Indeed, much the same territory as covered by *somatisation*, also edited by Christopher Bass and published in 1992. A comparison of the two multi-authored compilations provides a fair indication of progress over the past decade.

Perhaps the most positive development is that the Slaterian heresy (that to diagnose hysterical conversion is to miss an organic disorder) seems finally to have been laid to rest. Hysterical conversion, conversion, dissociation—whatever you care to call it—exists, the problem is: what is it? The paradox is laid bare in a chapter by Wessely: Conversion: The d’horizon of hysteria and allied disorders as presented it in a very well analysed and detailed work. The title of Heilman’s enjoyable book, “a neurologist’s view of brain behaviour relationships”, suggests a philosophical, reflective work. It is, in fact, an informal but sober review of the main areas of behavioural neurology to which Heilman has contributed during his long and productive career.

It has several strengths: personal cases illustrate the topics, personal anecdotes enliven them, and Heilman’s logical mind guides the reader judiciously through the twists and turns of neuropsychology. I particularly enjoyed his chapters on attention, self awareness, and praxis, all subjects on which he has worked.

Trained by Norman Geschwind, well versed in the old European tradition of cortical localisation, Heilman’s emphasis is strongly clinical. Experimental work in animals and functional imaging are largely off stage. In keeping with its informal approach the book is only lightly referenced: a pity, for some readers.

The book introduces a number of challenging generalisations. Heilman takes up Denny Brown’s suggestion that the parietal lobes mediate “approach”, while the frontal lobes inject the necessary measure of “avoidance”, enabling us to judge when approach is wise. He sketches a series of contrasts between the hemispheres: he links the left hemisphere with propositional aspects of language, “focal” perceptual processes, object recognition, restricted (right-sided) spatial awareness, positive emotions, and knowledge of “how” to get things done; he associates the right hemisphere with emotional aspects of language, “global” perceptual processes, face recognition, bilateral spatial awareness, negative emotions, and knowledge of “when” to act; he draws attention to its closer links (than those found on the left) to the limbic and reticular activating system. All this is fascinating—but some of the claims are more controversial than one would gather here, and there is no final synthesis.

This book makes a good introduction to classical clinical neuropsychology. If it should be taken with a pinch of salt the same is true of a good deal of more fashionable work.

**AZJ Zeman**

**Cerebral vasospasm**


As many readers will appreciate, cerebral vasospasm is well recognised as a major cause of morbidity and mortality of patients following subarachnoid haemorrhage. Despite the huge amount of work done both in the laboratory and clinical setting the pathogenesis and pathophysiology of this disorder is only partially understood. As a result effective treatment options remain elusive. Macdonald and Weir are pre-eminent in this field, having devoted much of their lifetime research efforts to it. They therefore compile this book from the position of much authority.

Having researched into vasospasm myself, I realised that to make progress in understanding the pathogenesis of vasospasm required a multifaceted approach. Most of the work that had been done and was being done was very focused and therefore the knowledge base fragmented. In essence we were not seeing the whole picture. This book addresses that problem and is the most comprehensive and detailed work on the subject.

It covers information on the history of the condition to current understanding of pathogenesis and pharmacology. It covers the medical aspects of vasospasm as well as the surgical aspects and touches on future research directions by looking at molecular biology and the genetics of vasospasm.

It has brought together a vast amount of information from a number of sources and presented it in a very well analysed and systematic fashion. I would agree with Neil Kassell’s foreword that this is indeed a magnum opus and would play and important factor in finding a remedy for vasospasm.

I would add that it is a must read for any neuroscientist currently undertaking research into the subject and indeed would be a valuable background text for any neurosurgeon in training.