To the Editor:

The article by Hund-Georgiadis et al on crossed nonaphasia in a "dextral" with left hemispheric lesion\(^1\) deserves attention because of the light it and similar cases throw on the subject of laterality and hemispheric dominance in humans. However, this requires abandoning certain conventionalism, which is the cause of the puzzlement they expressed as to the laterality of the results they obtained, ie, ipsilateral activity of motor cortex on moving the ostensibly dominant hand (as opposed to the neurologically dominant, as I explain below). In this light, their result is a reaffirmation of the findings of high-resolution EEG experiments of Kristeva et al,\(^2,3\) Barrett et al,\(^4\) and others.\(^5\) More recently, similar observation has been made using functional MRI,\(^6,7\) positron-emission tomography,\(^8\) transcranial magnetic stimulation,\(^9,10\) and Doppler\(^11\) techniques. These observations require an anatomical substrate, depicted below.

One source of confusion of the respectable authors is their complete reliance on an inventory-based methodology in ascertaining a subject’s laterality. The other is lack of appreciation of the anatomical significance of absence of apraxia in the subject’s (ostensibly nondominant) left hand. Cognizance of these 2 critical points as well as intimate familiarity with 2 other laterality indexed clinical syndromes are essential ingredients in grasping the issues involved.

The twin entities to which I referred are syndromes of ipsilateral weakness involving the nondominant hand in certain lesions affecting the dominant hemisphere,\(^12,13\) and that of weakness involving nondominant hand after callosotomy, whether natural (such as in Marchiafava-Bignami disease or trauma\(^14\)–\(^16\)) or iatrogenic\(^17\) (and my own unpublished data). The fact that these twin syndromes are laterality indexed clearly denotes that the callosum mediates the symptoms and signs observed in both. Only electrodiagnostic methods have the temporal resolution necessary to discern which hemisphere controls the other through the callosum via an excitatory synapse (evidenced by occurrence of diaschisis symptomatology ipsilateral to the minor hemisphere on interruption of its excitatory connection from the major hemisphere, in the twin syndromes mentioned above). The older techniques of bilateral simultaneous measurement of reaction times in right- and left-handed subjects,\(^18\) the more sophisticated recent techniques for detecting movement-related cortical potentials, and other approaches\(^19,20\) support temporal priority of activation of the cortex related to the (neurologically) dominant hand by an amount equal to that of interhemispheric transfer time in comparison to the activity detected over the other hemisphere related to the nondominant hand. Thus, the earlier activation of the dominant hand in bilateral, simultaneous key pressing or other tasks\(^18\)–\(^20\) as well as bilateral activation of the motor cortex when moving the nondominant fingers indicate the...
existence of a central command for initiating all voluntary movements, located in the major hemisphere. Such findings are common to all these studies, providing a technical definition of handedness and a satisfactory answer to the authors’ enquiry in their article and to that of earlier investigators, including the 2 neurosurgeons who decades earlier commented on the "amazing [fact] that in all our cases [of ipsilateral hemiplegia] the left hemisphere was involved. Surprisingly, the same holds true in the cases of ...and Kernohan and Woltmanand... where clinical data are available".12

To recap, the validity of self-declared laterality is only statistical in nature and cannot guarantee the same on an individual basis, especially among those who consider themselves left-handers. The occurrence of crossed aphasia and crossed nonaphasia cases reflects the validity of the above statement. The lack of left-hand apraxia and absence of aphasia in their subject indicated that the patient was neurologically left-handed. The fact that he had gone through life masquerading as a right-hander is something many subjects do, the majority of them ostensibly left-handers. We now know that the role of family history in handedness is obscure21 (see below) and that the introduction of it into the subject of cerebral dominance by Foster Kennedy22 was an improvisation to rescue Paul Broca’s fledgling cerebral localization theory from collapsing under a barrage of similar cases (of crossed aphasia or crossed nonaphasia), which were threatening the survival of Broca’s claim. By declaring that all right-handers with family history of left-handedness were "real" sinistrals and left-handers with no such history were "real" dextrals, Kennedy successfully fought off the challenges—but at the cost of delaying an understanding of motor control in humans that required not an arbitrary but a technical/anatomical definition of handedness, as provided here.

To summarize, there is nothing odd about the brains of real left-handers, except with regard to which side controls the other compared with right-handers (see above), and the fact that they have been a ubiquitous minority of humanity. Here comes the dividend of the explanatory power of the above scheme: Since the callosum is the path through which the axons of the neuronal aggregate that underlie laterality traverse, the lopsidedness of laterality distribution in humans must be related to an unfavorable event related to the development of the same structure in utero (allowing a role for imitation and social factors in certain circumstances). We know that anomalies of corpus callosum (eg, holoprosencephaly)23 are the most common finding among aborted fetuses in humans. Therefore, the vast majority of those destined to become left-handers die before birth. Moreover, it is known that such life-threatening anomalies are sometimes genetic in nature.24,25

References


**Response**

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We would like to thank Dr Derakhshan for his detailed comments concerning our article. Focusing on the main point, he apparently implies that our patient was actually left-handed, at least as far as "neurological dominance" is concerned. The absence of apraxia is used to undermine this hypothesis. We, in contrast, had clearly stated\textsuperscript{1} that (1) the patient was right-handed by all means of behavioral testing and history, and moreover that (2) he was evaluated only in the chronic phase (4 years after stroke onset). Thus, initial presence of apraxia cannot be fully ruled out; the statement of Derakhshan is at best speculative.

Dr Derakhshan implies that a number of patients go through life masquerading as right-handers. Furthermore, these patients would be unaware of their actual handedness, would never have presented a left-handedness, and could not be identified by most commonly used examination techniques. Declaring a patient as "neurologically sinistral" without any hard evidence, as Dr Derakhshan suggests in our reported case, certainly requires abandoning conventionalism. We are not convinced, though, that such an approach would really help to reduce the confusion on the topic of laterality.

Moreover, it is rather questionable whether the arbitrary attribution of a masked left hand dominance—as suggested by Dr. Derakhshan—could really help to "grasp" the whole issue of hemisphere dominance in this particular case. According to previous laterality research,\textsuperscript{2,3} only a very small portion of "phenotypical" left-handers (2%) show exclusive right-hemisphere activation associated with language tasks. On the whole, left-handers are much better characterized by a left-hemisphere preponderance of language function (76%) or by a mixed laterality to different degrees. Hence, we still end up with the central question of why a phenotypical right-hander (or, more speculatively, a masked left-hander) did not exhibit aphasia following stroke in the territory of the left middle cerebral artery and exclusively employs the right hemisphere in a motor, language and alertness task. And most likely we will return to the concepts of a mirrored brain organization.

References