Challenging traditions in apraxia

This scientific commentary refers to ‘Critical brain regions for tool-related and imitative actions: a componential analysis’, by Buxbaum et al., (doi:10.1093/brain/awu111).

Apraxia is a symptom on the border between cognition and motor control. It is predominantly caused by left brain damage and frequently accompanied by aphasia. Theories of apraxia have been dominated by a model of the conversion of mental images into motor commands proposed more than 100 years ago by the German psychiatrist Hugo Liepmann and revived for modern cognitive neurology by Norman Geschwind and his colleagues in Boston (Fig. 1). There are several variants of this model but they agree that deliberate control of gestures involves the conversion of a concept of the intended gesture into motor commands, that this conversion is implemented in the brain as a posterior to anterior stream of neuronal processing, and that the parietal lobe occupies a crucial position in this stream (Goldenberg, 2009, 2013). However, in this issue of Brain, Buxbaum and colleagues present data from voxel-based lesion-symptom mapping experiments that suggest that revisions to the model may be required (Buxbaum et al., 2014).

The putative sequence of conception and production of gestures was fundamental for distinguishing two forms of apraxia: ideational apraxia due to insufficient generation of the concept; and ideomotor apraxia due to its insufficient conversion into motor execution. Clinical criteria for their distinction remained controversial, but most authors agreed to classify defective manipulation of real objects as ‘ideational’ and defective performance of empty-handed gestures as ‘ideomotor’ apraxia. The rationale for this diagnostic classification was elaborated by Liepmann (1920), who reasoned that empty-handed execution of gestures deprives the hand from support and guidance by external objects and therefore taxes the integrity of the route from the mental image to motor execution. Empty-handed performance includes gestures that may be meaningless or meaningful, and they may be requested by verbal command or in imitation. Production of meaningful gestures on command can be examined for gestures with conventional meaning (such as ‘OK’ or ‘military salute’) or for pantomimng tool use. For pantomime, comprehension of the command can be supported by a picture of the tool. Both types of meaningful gestures can also be tested in imitation. Meaningless gestures are usually tested only in imitation, because their verbal description may overtax language comprehension of aphasic patients. In spite of their diversity, all of these gestures should be disturbed when parietal lobe damage interrupts the conversion of the concept of the gesture into motor execution.

The validity of this prediction has been cast into doubt by case reports of patients with parietal lobe lesions who committed severe errors on imitation of meaningless gestures but could produce meaningful gestures including pantomimes of tool use both on command and in imitation (Goldenberg and Hagmann, 1997). By contrast, other patients failed pantomimes on command but could imitate meaningless gestures (Tessari et al., 2007). This double dissociation cannot easily be reconciled with damage to a unique path of gesture production, because damage to it should disturb the production of gestures regardless of their meaning.

Recently, new challenges to the traditional model of apraxia have emerged from the application of refined techniques of voxel-based lesion–symptom mapping to large groups of patients with circumscribed brain lesions. Lesion information had been considered also in the clinical studies discussed above but was mainly restricted to the laterality of lesions, at best complemented by recording which lobes were affected. The new techniques permit fine-grained anatomical analysis of lesion locations and statistical evaluation of their influence on behavioural test results.

A handful of studies have already emerged from this new approach and others are in preparation or already submitted. Among them, the paper by Buxbaum et al. (2014) excels by virtue of its methodical astuteness, large sample size and the clarity of its results. Buxbaum et al. examined patients with left brain damage for the production of pantomimed tool use on command, the imitation of the same pantomimes, and the imitation of meaningless gestures of similar motor complexity, and used voxel-based lesion-symptom mapping to determine the crucial lesion locations underlying the observed deficits. As all of these tasks demand empty-handed execution of gestures, the traditional model would predict that they share a common location corresponding to the neural implementation of the conversion of the concept of the gesture into its execution. The postulate that the conversion is achieved by a posterior to anterior stream of neural processing with a central role of the parietal lobe would further predict that this common location includes parietal regions.

The results turned out to be more complex than the predictions. There was indeed a region in which lesions caused deficits in all three tests, but it was located quite far from the parietal lobe, in posterior temporal region 37, extending posteriorly to the occipital lobe (area 19) and anteriorly into middle and inferior temporal lobe (area 20, 21). The temporal extension of this lesion site was distinctly greater for pantomime to command than for imitation of meaningless gestures. By contrast, parietal lesions had a substantial influence on imitation of meaningless gestures but nearly none on pantomime to command. Imitation of pantomime was associated with both temporal and parietal lesions that were in the same location as, but somewhat smaller than, those of the ‘pure’ conditions, imitation of meaningless gestures and pantomime on command.

The differential importance of parietal and temporal lesions for pantomime on command and imitation of meaningless gestures gives a satisfactory explanation for their clinical double dissociation. The common lesion location at the posterior border of the middle temporal lobe satisfies the general prediction of the traditional model that there is a commonality between different manifestations of ideomotor apraxia, but not the more specific prediction that this common zone is in the parietal lobe. In their
In their recent discussion, Buxbaum et al. propose a rather intricate speculation for reconciling the paucity of parietal influence on pantomime with a central role of parietal regions for gesture production. However, to my understanding the main upshot of their study is the challenge of the traditional model of apraxia rather than its confirmation. Abandoning the traditional model opens the stage for new theories and experiments unveiling the anatomical and functional underpinning of the fascinating clinical syndrome of apraxia.

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**References**


**Figure 1** Alternative versions of the posterior to anterior stream of motor control and of the central role of parietal lesions in apraxia. (A) Hugo Liepmann (1920) proposed that mental visual images of intended actions were created in occipital regions and transferred to the precentral motor region for motor execution. Interruption of this stream leads to ‘ideo-kinetic’ (later renamed ‘ideomotor’) apraxia. Parietal lesions are likely to cause ideo-kinetic apraxia because they interrupt the fibres travelling through the parietal lobe on their way to motor regions. (B) Norman Geschwind (1975) moved the origin of the posterior to anterior stream of action control to Wernicke’s area in the posterior third of the superior temporal gyrus and inserted a premotor relay into its connection to motor cortex. Inferior parietal lesions cause apraxia when they encroach upon the arcuate fasciculus which runs beneath the parietal cortex. (C) Heilman and Rothi (1993) postulated that parietal lesions destroy motor representations stored in angular and supramarginal gyrus. PM = premotor; M = motor; SMG = supramarginal gyrus; AG = angular gyrus; W = Wernicke’s area; VVA = visual association area; VA = primary visual area. When gestures are performed on verbal command the representations are activated from Wernicke’s area, and when they are imitated, from visual areas. Adapted from Goldenberg (2009) with permission by Elsevier.