Motor control: **Mechanisms of motor equivalence in handwriting**

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Handwriting is a classic example of how the details of movement can be scale and plane invariant: letter forms reflecting personal style are unchanged, whether one is writing on a piece of paper, on a blackboard or in the sand using the foot. Recent research points to a role for the parietal cortex in such motor equivalence.

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The forms and shapes used in cursive handwriting are characteristic of the individual. Through the school years, varied instruction on letter formation from a number of teachers, combined with choice reflecting the interaction between personality, social and cultural influences, result in the adoption of a wide range of stylistic variations [1]. Thus, for example, one person might favour a cross-bar join between the t and the h in "the", whereas another might link the base of the t with the h and only return to cross the t after completing the e. There are, of course, many possible letter combinations in the language, and so a large set of different handwriting features is generated through variation in linking strokes and letter forms. But a given individual tends to use the same form of any given handwriting feature, especially if the surrounding letter context is taken into account, and feature variation is generally greater within than between individuals.

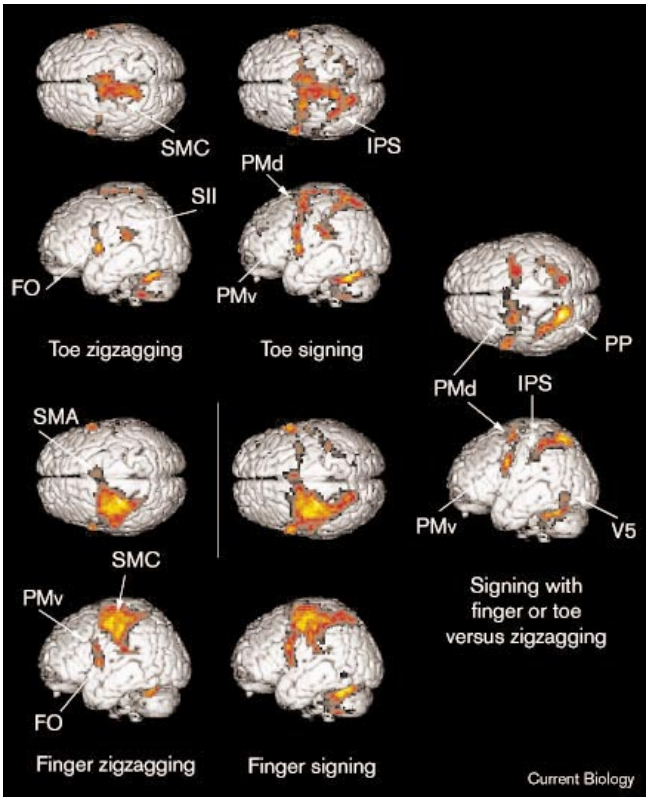
The probability of a particular set of features occurring in the handwriting of two people — other than through deliberate forgery — can thus be vanishingly small as the sample base increases in size. This provides a scientific basis for the assessment of authorship of disputed documents in legal proceedings [2–4]. But individual consistency in form of handwriting is also interesting because it has been suggested to be a paradigm example of a central issue in understanding the relation between the organisation of action and the control of movement.

Although we talk about 'hand' writing, it is evident that writing style is the same if movements are produced by the arm, rather than by movements of the thumb and fingers. For example, letters have the same form when written on a blackboard several centimeters high, too large for the range of movements produced by the digits [5]. There are a number of fundamental differences between the movements involved in writing on a blackboard and on paper: thus, when writing on a blackboard, the muscles that subserve the action are different than when writing on paper; the torques required for larger body segments are much greater (and do not simply scale with letter height); and gravity introduces an asymmetric load on up-strokes and down-strokes. But despite these differences, in terms of the written product, the outcome of action is the same. Thus there is constancy of the 'movement product' despite major changes in motor implementation.

This phenomenon, referred to as motor equivalence, applies, at least to a first approximation, across actions carried out with hand or foot [6], or with the preferred versus the non-preferred hand [7]. Motor equivalence is of theoretical importance, because it suggests that actions are encoded in the central nervous system in terms that are more abstract than commands to specific muscles. For example, handwriting may be represented in terms of 'strokes' that are encoded in terms of relative position and spatial direction, but without any specific motoric reference. Details of motor implementation, such as stroke size or speed, may be left unspecified until the effector is known. Once the effector is known, allowance can then be made for effector-specific complexities, such as gravity or joint segmental interaction torques, which distort trajectories in a way that depends non-linearly on movement speed [8].

A recent study was carried out to determine which area, or areas, of the brain underlie effector-independent representation of handwriting. Rijntjes *et al.* [9] used functional magnetic resonance imaging (fMRI) to examine patterns of brain activation associated with signing using either the hand or the big toe. Participants were asked to lie in the scanner with their eyes closed and arm and legs supported (to minimise proximal muscle activation which would tend to move the trunk and head and so affect the scan quality), and to write their names in the air with minimal horizontal translation (so letters were formed on top of each other). Over a 24 second period, six complete fMRI scans were taken of the whole brain volume, and the signing task was carried out repeatedly in this period. To determine which brain regions show activity specifically related to writing, as opposed simply to the production of a series of movements, the researchers also asked participants to produce simple, repetitive, up–down zigzag movements with approximately the same frequency as the up–down movements made in signing. Baseline measures of brain activation were taken with the subject quietly resting.

Figure 1



Patterns of brain activation in signing and zigzagging with finger or toe [9]. Sites showing statistically significant activation are projected onto superior and lateral views of the surface of an averaged anatomical image of the brain of the nine participants. The top shows the results for toe zigzagging compared to rest (left) and toe signing compared to rest (right). The bottom shows the results for finger zigzagging compared to rest (left) and finger signing compared to rest (right). On the far right are shown the areas activated in both finger and toe signing compared to the corresponding zigzagging movements. This shows that the areas involved in signing, irrespective of the performing extremity, are the secondary sensorimotor areas that are part of the anatomical finger representation. Brain areas: SMC, primary motor cortex; SMA, supplementary motor area; PMv, ventral premotor cortex; PMd, dorsal premotor cortex; IPS, intraparietal sulcus; PP, posterior parietal lobe; SII, secondary sensory cortex; FO, frontal operculum; V5, V5/MT, the visual motion centre. (See text for details.)

When they compared the patterns of brain activation during the zigzaging task and during rest, Rijntjes *et al.* [9] observed task-associated activity in most parts of the sensorimotor system, with somatically segregated active regions associated with movements of the hand or foot (Figure 1). These regions included the contralateral primary sensorimotor cortex, the adjacent superior parietal lobe (area 5), the supplementary motor area (SMA), the anterior cingulate, the thalamus, the basal ganglia, the cerebellar hemisphere and the vermis. Compared to rest, zigzagging with the finger also gave rise to activation of middle and ventral intraparietal areas (MIP and VIP), which was not the case when zigzagging with the toe.

Signing with the finger, compared to rest, activated the same areas as the zigzagging finger movement, but with the addition of activity in the posterior parietal cortex (PPC) and the occipitotemporal junction. Signing with the toe also showed these two areas of activation. The occipito-temporal junction has been reported to have a visual motion center [10] — known as V5 or MT — and its activation may be related to a comment by Rijntjes *et al.* [9] that all their subjects reported a strong visual image of their signature while signing. The PPC (area 7) has previously been shown to play a role in the visual guidance of movements, especially when the visual information is retrieved from memory [11], and lesions to this area disturb such functions [12,13]. It thus seems plausible to suggest that this area of cerebral cortex is involved in the retrieval (or generation) of an effector-independent visual representation of the movements that generate the strokes in handwriting.

Toe signing compared to toe zigzagging was not just similar to finger signing in involving additional activation of the PPC and the occipitotemporal junction. Toe signing also involved activation of all finger areas involved in finger signing, except for those in the primary sensorimotor cortex — that is, finger areas in the premotor areas PMd, PMv and SMA, in the intraparietal areas MIP and VIP, in the thalamus and in the cerebellar hemispheres. As these same areas were activated during finger zigzagging as well as signing, it suggests that the PPC activation during signing did not yield a truly effector-independent representation but, rather, reflected an area of cortex responsible for hand movements in general.

Why might this have been the case? One possibility is that it might relate to participants' reported visualisation of the handwritten signature when signing with the toe. Previous studies, using positron emission topography (PET), have shown that, when we imagine reaching and grasping movements, there is activation of brain areas, including parietal cortex, similar to those activated during real actions [14,15]. It may be that participants' visualisation in Rijntjes *et al.*'s [9] experiment included imagining the act of signing with the hand as an aid to toe signing, and that this activated motion areas specific to the hand, including parietal regions. An issue for this interpretation is that a previous PET study of brain activation while subjects imagined writing single words failed to show parietal activation, although there was involvement of other cortical regions, including prefrontal cortex and SMA [14]. Assuming the latter is not merely a reflection of the lower sensitivity of PET, it suggests that, if the parietal activation observed during toe signing reflects visualisation of the hand writing, it is contingent on the support provided by such visualisation of concurrent movement of the foot. This point, if supported by further research, would be important as it indicates the degree, or intensity, of visualisation depends on the function of that visualisation.

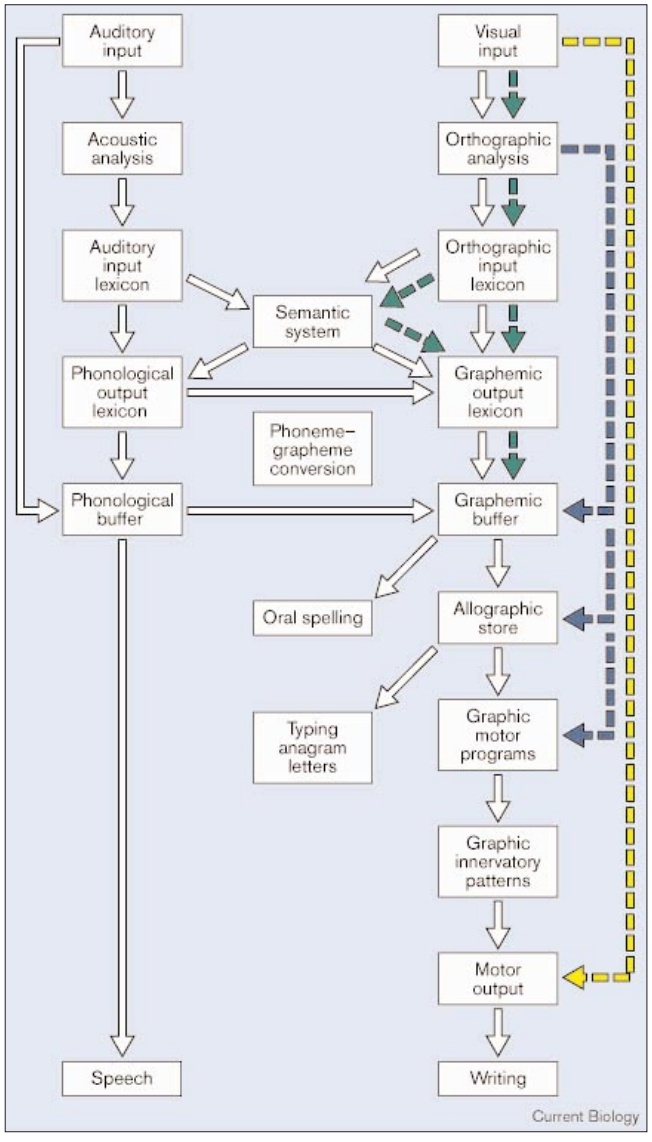
Given the brain imaging evidence that handwriting movements depend on the parietal cortex [9], it might be expected that lesions in this area of the brain — for example as a result of a stroke — would affect the generation of handwritten letter forms. Neuropsychological studies have, in fact, distinguished a number of types of acquired writing disorder, or agraphia, with a primary subdivision being between those in which spelling is affected — usually across oral, typed or written output — as a result of impaired phoneme-to-grapheme conversion (see Figure 2), and those in which letter formation is impaired [16]. The latter group can be further subdivided into cases in which letters are reasonably formed but show poor spatial arrangement (spatial agraphia), and those in which letter formation is affected (apraxic agraphia).

An example of apraxic agraphia reported by Zangwill [17] resulted from a left occipitotemporal lesion that produced severe impairment in writing despite good oral spelling ability. In writing to dictation, the patient produced malformed letters which resulted in an illegible output, and yet he could copy letters or words with normal speed and accuracy, demonstrating the impairment was not simply attributable to motor weakness. Another case, described by Baxter and Warrington [18], involved a patient with a left PPC lesion that resulted in preserved spelling abilities but writing spontaneously or to dictation that was so impaired that not a single legible word was produced. Once again, this patient was able to copy letters and words with near normal fluency. Interestingly Baxter and Warrington [18] noted that their patient's impairment of letter formation was quite specific and did not extend to pictorial material, in that the patient was still able to draw pictures from memory.

In contrast to the specificity of apraxic agraphia, cases of spatial agraphia are normally considered secondary to a more general problem of spatial analysis and perception. This results in general layout errors of spacing and orientation, as well as particular types of letter errors, such as a repetition of strokes (especially in the letters m, n and u). Such problems are commonly found with lesions in posterior right hemisphere [19].

These neuropsychological studies of agraphia are consistent with the brain imaging results of Rijntjes *et al.* [9] in suggesting the importance of the parietal cortex in general, and PPC and occipitotemporal junction in particular, in the representation of handwritten letter forms. Such parallels between research based on brain imaging and the effects of focal brain lesions are important and suggest further avenues for investigation. For example, future neuropsychological research might build on the brain imaging results and examine the extent to which dysgraphia is independent of the writing effector. If movement recordings are made of writing in the air by hand or foot, do patients with dysgraphia exhibit parallel errors in the two output modalities?

Figure 2



Processes in the production of handwriting. The flow diagram illustrates the relationship between speech and handwriting [21–24]. The written output branch (bottom right) involves a series of stages in which graphemes (letters or letter groups) are specified first (through lexical look-up or by use of phoneme-to-grapheme correspondence rules). This is followed by selection of effector-independent allographs, which specify features such as letter shape, lower versus upper case, and so on, perhaps in terms of stroke sequence. Effector-specific motor programmes – indicating muscle activation patterns to achieve desired letter size, and so on, given the specific writing context – are then generated and these, when executed, will result in the written trace. The coloured dashed arrows represent three alternative processing routes: yellow, pictorial copy based on low-level movements (for example, letters drawn as visual patterns); blue, grapheme copying based on mid-level strokes (for example, used for single letter or non-word copying); green, lexical copying based on analysis with access to meaning.

As it is unpractised, writing in the air with a foot is likely to result in more errors than writing with a hand, even in

healthy control subjects. The question would thus be whether errors made by dysgraphic patients in writing with a foot include as a subset errors equivalent to those made when writing with a hand? If so, the interpretation would be that this subset comprises those letter forms whose stored representations — or generative processes — have been affected by the lesion. Future brain imaging studies of writing might follow the lead of dysgraphia research and examine relations between lexical and phonological processes and the writing system.

Rijntjes *et al*. [9] did not find marked lateralisation of parietal areas activated in signing compared to zigzagging, even though language is lateralised to the left hemisphere. Yet left temporoparietal junction at the supramarginal gyrus — Brodman's area 40 — has been identified with phonological short-term memory [20] and might have been expected to contribute to spelling processes associated with handwriting. One possible reason for the lack of lateralisation in the results reported by Rijntjes *et al*. [9] is that repeated signing may shift the task towards the production of a complex spatial movement pattern, drawing on the right parietal cortex, and away from the normal language-based generative processes of writing — another topic for future research!

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