

CASE REPORT

Dystypia in acute stroke not attributable to aphasia or neglect

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SUMMARY

A 68-year-old right-handed man had a sudden onset of impaired typing ability due to an ischaemic stroke that recovered over 2 months. The typing impairment was grossly out of proportion to his transient handwriting disturbance. Diffusion MRI showed a recent acute left temporoparietal infarct. There was no evidence of aphasia, alexia, agraphia, visuospatial inattention, sensory loss, neglect or poor coordination that could account for his isolated typing impairment. This example of a stroke that disproportionately affected typing more than handwriting abilities has practical implications for what deficits to look for in patients with stroke when assessing their fitness for work and rehabilitation requirements.

BACKGROUND

Keyboard typing is a multifunctional task. To type a familiar word such as 'ball' requires first accessing some linguistic representation of the object, whether orthographic or phonological. Next is the conversion of this information into specific procedural motor functions. Typing differs from writing in that one must be able to read the letters on the keyboard to know which one to press. Alternatively it requires some form of visuospatial memory that maps each specific letter to a location on the keyboard, as is the case with 'touch typing'. Lastly, intact visual fields and visual-spatial attention along with coordination and control of the fingers is needed to push the correct keys.

There is likely to be a substantial overlap between the processes required to write and type and yet few studies have examined whether writing and typing can actually be impaired separately.¹⁻³ Recent work has shown that different patterns of agraphia can be reversibly induced by electrical stimulation during surgery of the left superior parietal gyrus with similar impairments in typing and writing but minimal or no effects on oral spelling.¹ The finding that a stroke can disproportionately affect typing more than handwriting has practical implications for assessing patients' fitness for work after a stroke, particularly in an increasingly computerised society. It also may help us elucidate the anatomical brain locations involved in the typing process.

CASE PRESENTATION

A 68-year-old man presented with a sudden onset, 1-day history of typing difficulty. Prior to presentation he had awoken from an afternoon nap and

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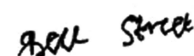


Figure 1 Handwriting sample written 1 day after his stroke showing the words 'Bell street'.

tried to use his computer. He had difficulty navigating the menu items and when he opened a word processing programme he typed jumbled words. His hand did not feel 'normal' when he wrote but he was able to write with occasional difficulty. He presented to hospital 1 day after his stroke with impaired typing ability that took 2 months to recover to baseline, while his handwriting was neat and legible (figures 1–3). The patient was an experienced typist who previously typed very well with a 'two finger' technique without looking at the keys.

The patient had a 2-year history of Parkinson's disease and associated micrographia. His writing was legible and there was little change from his prior ability when he presented to the hospital. His Parkinsonian symptoms were well controlled on cocareldopa. He had hypertension and was taking ramipril, aspirin and a fibrate.

Examination findings

On neurological examination his coordination was intact and he had no dysidiadokinesia. He was also able to follow three-stage commands involving taking a piece of paper in the right hand, folding it and placing it on the ground. He also performed the finger-to-nose test with no dysmetria or intention tremor. His resting tremor was well controlled. He could write legibly showing intact fine motor control. His cranial nerves II–XII were normal and he did not show gaze preponderance, visual field defects or spatial inattention in the double-simultaneous stimulation test. This implies that visual-spatial neglect was not the cause of his typing errors. Upper and lower limb neurological examination were normal and there were no other examination findings.

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Figure 2 Typing sample 1 day after his stroke. There are a further series of emails showing gradual improvement for a further 2 months.

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It's notjng mucjh to worry about – trensieth ischemis attack. There has been a marked change since even yesterdat, and I should think that the typing may back to my normal level of eccord I mean error by Friday; but I'll keep strying.

Figure 3 Typing sample 4 days after his stroke.

Four weeks poststroke he underwent further neurological assessment and scored 28/30 on the mini-mental state examination, 88/100 on the Addenbrooke's Cognitive Examination-revised version (attention and orientation 17/18; memory 22/26; verbal fluency 8/14; language 26/26; visual spatial 15/16) and he scored 50/50 on the National Adult Reading Test. He was only able to list seven words beginning with the letter "T" and only 10 animals in 1 min which accounted for his reduced verbal fluency. He did not show neglect or visual spatial impairment when copying pentagons, cubes and clock drawing as part of the Addenbrooke's. He could also count the number of dots and correctly identify masked letters.

INVESTIGATIONS

Two days after his stroke, an MRI scan at 1.5 T showed a recent left temporoparietal infarct consistent with his symptoms (figure 4). Diffusion, FLAIR and T2-weighted imaging showed scattered areas of increased signal in the left posterior insular cortex involving a small area of the transverse temporal gyrus. In addition there were extensive white matter hyperintensities in keeping with background small vessel changes which were beginning to coalesce. There was also an old cortical infarct in the right frontal cortex but no haemorrhage or microhaemorrhage.

A Speech and Language Therapy (SALT) assessment was carried out at 3 weeks poststroke and showed that his typing impairment was disproportionately worse than his writing. This suggests that it was not just agraphia. His handwriting was legible although micrographic with no spelling errors and the same standard as before his stroke. There was also no deterioration for low frequency, irregular spelling words or non-word-spelling. Irregular word spelling demonstrated that he could store and use 'whole-word' representations. Whereas his non-word spelling showed that his phonological processing was intact. In comparison his spelling on the keyboard was significantly worse and the errors made were typically an additional stroke of an adjacent key causing an incorrect letter insertion, or a letter order error, for example, seize becomes 'sieve'. Importantly the errors were not confined to any one part of the

keyboard as they might have been if they were the result of visual-spatial neglect. Neglect is also more commonly the result of right-sided parietal lesions. He was able to construct fully grammatical paragraphs. He demonstrated insight and was aware of the mistakes in his typing and could correct them.

The typing deficit was not attributable to alexia. The patient was able to read the newspaper and hospital forms and he reported his reading ability was unaffected by his stroke. His SALT assessment confirmed that he was able to read aloud fluently and scored 40/40 on the Western Aphasia Battery paragraph reading section. His visual scanning was slightly slowed but he performed well in the letter identification task and continued to proofread writing for other people frequently in his spare time.

The patient's speech showed slight hesitation but was otherwise normal. He spoke in full grammatical sentences with no obvious word finding difficulty or semantic or phonological paraphasic errors. He described a loss of confidence with fluency of his verbal expression but this was present before the stroke. He was only able to list 10 animals in 1 min and listed seven words beginning with the letter 'T'. It was the opinion of the speech and language therapist that this was an example of thought freezing that he experienced under pressure. His auditory comprehension was unaffected. He was able to follow complex sequential commands, retain and repeat complex sentences and showed no difficulty with right or left orientation.

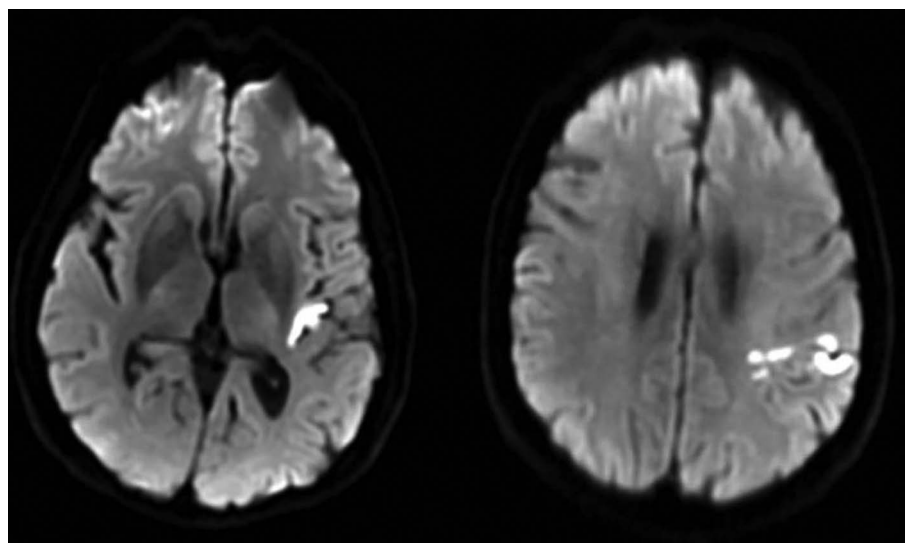
DIFFERENTIAL DIAGNOSIS

The appearance on diffusion MRI—the signal change and lesion distribution—as well as the appearance on the other sequences were pathognomonic of a recent cortical infarct. There is no differential diagnosis for this particular MR appearance.

TREATMENT

Secondary stroke prevention in line with current guidelines including early mobilisation and lifestyle advice. The patient is already on aspirin, a fibrate and an ACE-inhibitor.

Figure 4 Two images from MRI, diffusion imaging at 36 h after onset showed an acute 21 mm infarct in the left posterior insular cortex involving a small area of the transverse temporal gyrus. There was also an old cortical infarct in the right frontal cortex.



OUTCOME AND FOLLOW-UP

The patient's typing fully recovered after 2 months evidenced by a series of emails and self-report.

DISCUSSION

There are only a handful of studies that report a specific typing impairment that is not attributable to agraphia, alexia, aphasia or neglect. Only two of these studies to date have localised the lesion on MRI.^{2,3} One of these is a recent case from Korea showing a left frontal subcortical infarct on an MRI.² The authors suggest that damage to the dorsolateral prefrontal cortex, the posterior parietal cortex or the connecting networks between them may result in impairment in visual-spatial memory for the position of the individual keys. Indeed there is good evidence from cognitive neuroscience that the hippocampus and frontoparietal network play an important role in visual-spatial memory.⁴ This explanation is compatible with our patient where there is a temporoparietal infarct which could interrupt this network. The other report is of a case from Japan where the infarct is in a different area of the left frontal lobe.³ Interestingly, studies of 'agraphia' for pictograms have also shown the lesion to be in the left frontal lobes.^{5,6} Unfortunately the value of comparing these studies to our own is limited by the individuals communicating in different languages. Korean and Japanese both use pictograms rather than letters.

The first case report to describe a case of isolated typing impairment was an American touch typist who had a left parietal infarct.⁷ However, this was only demonstrated on a CT scan and she also presented with a mild aphasia and multiple lesions. They argued that the parietal infarct caused a failure of somatosensory monitoring on the grounds that when the patient used her intact visual system to monitor typing she made far fewer errors. There are other compatible explanations, for example when the individuals looked at the keys, they no longer had to rely on their visual-spatial memory that matches each separate letter to a location on the keyboard. Rhu *et al*² posit that a loss of visuospatial memory, particularly for the keyboard arrangement, is the reason for an isolated typing deficit in their Korean patient. They showed that their patient had reduced performance on the 'Rey-Osterrieth Complex Figure Test' and the 'Fill-in Keyboard Test'. These may be more relevant tests of visuospatial memory than the ones that we used because our patient scored 15/16 on the visual-spatial component of the Addenbrook's Cognitive Examination.

Recent work demonstrated that a small glioblastoma in the left parietal lobe of an Italian patient resulted in a combined agraphia for handwriting and typewriting.¹ Furthermore electrical stimulation of the left superior parietal gyrus during surgery could reversibly induce different patterns of agraphia including written spelling errors or a complete cessation of writing with minimal or no effects on oral spelling.¹ Magrassi *et al*¹ argue that handwriting and typing share a working memory grapheme buffer. However, the mechanisms associated with visuospatial memory for typing are not fully understood and there may be other processes involved. Our patient's stroke may have resulted in a further pattern of agraphia that disproportionately affects typing more than handwriting.

Our patient was impaired in semantic (participants have to say as many words from the category 'animals' in 60 s) and phonemic (as above but naming words beginning with the letter 'T')

fluency. Parkinson's disease patients are usually significantly more impaired on semantic compared to phonemic fluency.⁸ Therefore, the deficit could be related to the 'dystypia' or alternatively white matter disease present prior to his stroke. A further possibility is that the isolated typing impairment may be a selective form of apraxia that was not detected in the investigations but he could perform three stage commands, imitate gestures (eg, cough, stick out tongue) and reported no functional difficulties except for his typing.

CONCLUSION

The patient is an example of a stroke that disproportionately affected typing more than handwriting. This has practical implications for the deficits that we look for in patients with stroke to assess their fitness for work.

Learning points

- ▶ The patient is an example of a stroke that disproportionately affected typing more than handwriting. This has practical implications for the deficits that we look for in stroke patients when assessing their fitness for work.
- ▶ The increasing and near-universal dependence on computer skills and communication through typing—email, text, tweets, data entries—means that such deficits are likely to become more common.
- ▶ Typing skills should be considered as part of rehabilitation after stroke or other focal brain injury.

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Competing interests None.

Patient consent Obtained.

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