LETTER TO THE EDITOR

To the Editor

Impact of New Technologies in a Stroke Presentation: A Case of Dystextia and Dystypia

Keywords: Dystextia, dystypia, caudate, infarct, ischemic stroke

As nonverbal communication with digital devices becomes more prevalent, the onset of deficits involving the use of these new technologies will likely become more common as a presenting sign of acute or subacute neurological dysfunction. We present a case description that illustrates this situation.

A 61-year-old right-hand-dominant male presented to hospital after he and his wife noted that he was having difficulty composing text messages on his cellphone and was unable to type a password into his personal computer. He was able to recall the password, verbally state it, and write it using a pen, but he could not type it into the computer. He had also experienced difficulty with putting while playing golf over the past few days, consistently overshooting his putts. The patient was a smoker with no other vascular risk factors or co-morbidities. He was not taking any medications.

On examination, he used a slow "hunt-and-peck" one-finger style and had difficulty finding keys when typing on a keyboard. He was unable to touch type and made errors. Previously, he was an experienced computer user able to proficiently touch type. There was right-sided visual inattention, and bilateral ideo-motor and limb kinetic apraxia. His line bisection task was normal. Language assessment demonstrated normal fluency, naming, comprehension and repetition, as well as reading and writing. Motor, sensory, coordination and gait examinations were normal.

The head CT and CT angiogram (CTA) disclosed hypodensities in the head and body of the left caudate and at the left parieto-occipital junction consistent with subacute stroke (Figure 1), together with severe narrowing of the proximal left middle cerebral artery (MCA). Echocardiogram and Holter monitor were normal. The presumed aetiology was large-vessel atherosclerosis. The patient was admitted for observation and started on acetylsalicylic acid (ASA) 81 mg, Plavix 75 mg, and atorvastatin 80 mg daily.

Further characterization of cognition and, in particular, language was performed. His score on the Montreal Cognitive Assessment (MoCA) was 19/30. Occupational therapy (OT) assessment revealed deficits in auditory memory and list memorization, with normal visual memory. There was impairment in two-dimensional constructional abilities, but no evidence of motor apraxia or agnosia. He missed some stimuli in the right visual field on a star cancellation task. A speech language pathology assessment disclosed a few self-corrected verbal paraphasic errors during spontaneous speech and confrontation naming, as well as mild paragraphic errors. He showed "difficulty organizing his thoughts to write a cohesive paragraph describing a picture." Overall, the patient demonstrated mild expressive language deficits, while auditory comprehension, repetition, and reading were within functional limits.

The patient completed dual antiplatelet therapy for 90 days and continued ASA indefinitely. At one-year follow-up, his typing and texting skills had fully recovered and his MoCA score was 26/30 (with deficits in delayed recall and orientation). He continued to complain of mild, non-progressive episodic memory deficits and difficulty building three-dimensional multi-piece structures while woodworking. In the follow-up head CT/CTA, there was no significant interval progression of the left MCA stenosis or new strokes.

In our case, deficits in typing on the computer (dystypia) and texting using a cellphone (dystextia) were the presenting features of an ischemic stroke. Texting and typing involve the integration of multiple higher-order brain functions, including visuospatial processing, language skills, procedural memory, and gross and fine motor control. Nonverbal, electronic forms of communication are

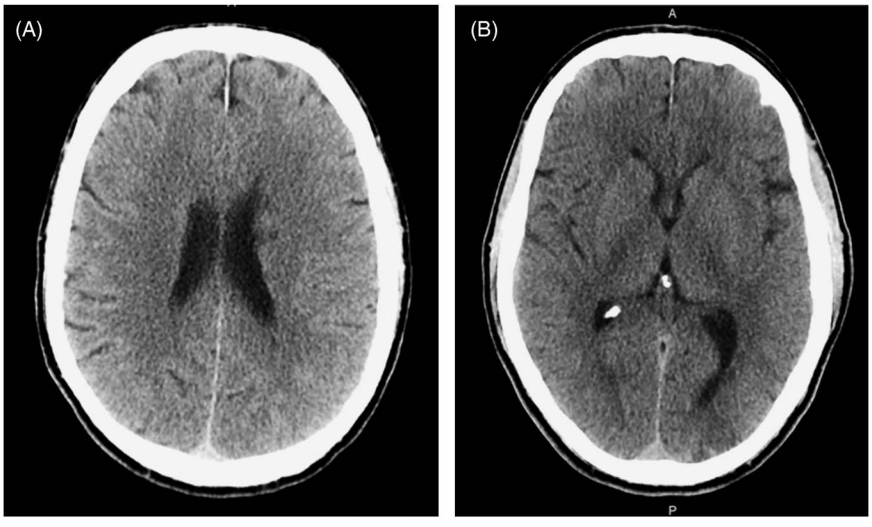

Figure 1: *CT head showing hypodense regions in the head and body of the caudate (A), in addition to a hypodense area in the left parieto-occipital junction (B).*

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	Cawood et al. 2006[1]	Whitfield and Jayathissa 2006[2]	Burns and Randall 2014[3]	Hannah et al. 2014[4]	Ryu et al. 2012[5]
Deficit of interest	Dystextia	Dystextia	Dystextia	Dystextia	Dystypia
Onset	Acute	Acute	Acute	Sub-acute	Acute
Associated symptoms and signs	Left-sided weakness, slurred speech	Headache, dressing apraxia	Slurred speech, right homonymous hemianopsia	Headache, depression	Impaired visuospatial memory
Hemiparesis	Left side	No	Right side	No	No
Language deficits	No	No	Receptive/expressive aphasia	No	No
Etiology	Ischemic stroke	Complex migraine	Ischemic stroke	Meningioma	Ischemic stroke
Imaging	Right internal capsule (genu) lacune	Normal	Left caudate and lentiform nuclei	Paramedian meningioma with hemorrhage	Multifocal, subcortical, predominantly left frontal
Prognosis for dystextia/dystypia	Recovered speed of texting	Full recovery of dystextia	Rapid improvement after IV tPA	-	Mild residual deficits in typing

Figure 2: *Published reports of dystextia and dystypia.*

now pervasive in our society, and deficits in these modern forms of communication may be the most conspicuous symptom on history and a sensitive sign of a presenting neurological disorder.

Dystextia and dystypia have been rarely described as a presentation of ischemic stroke as well as of migraine and meningioma (see Figure 2). In one report, IV tPA was administered after the timing of stroke onset was able to be estimated using the message history on a patient's phone, as there were no witnesses to the onset of his symptoms.[3]

In our patient, the problems with texting, typing, and higher-level language functions arose in part from infarcts to the head and body of the caudate. The mild right visual field neglect and apraxia, likely related to the left parietal lesion, would certainly also have contributed to his deficits. The most common deficits seen after caudate strokes are abulia, restlessness and hyperactivity, dysarthria and mild contralateral hemiparesis.[6,7] Almost all patients with dysarthria and hemiparesis had larger infarcts that also involved the anterior limb of the internal capsule. Lesions of the right caudate also resulted in contralateral neglect and left caudate lesions caused language deficits in a minority of patients in these case series. Connections between cortical association areas in the frontal, parietal and temporal lobes have been described in animal studies, and the caudate is proposed to have cognitive and behavioural functions within the basal ganglia-thalamo-cortical circuits.[8] Our patient did not display neuropsychiatric symptoms, but abulia and restlessness may arise due to interruptions in frontolimbic circuits or connections between the caudate and temporal lobe, respectively.[6] The transient language deficits seen after caudate infarcts are thought to be related to the disruption in connections between the anterior and posterior language areas and the caudate[7] or inputs from the auditory cortex to the head of the caudate.[6] Specific deficits can be related to the vascular territory affected; infarcts involving the anterior lenticulo-striate arteries cause only cognitive and behavioural deficits, while those involving the lateral lenticulo-striate artery territories cause motor and neuropsychological deficits.[7]

In the digital age, neurologists should incorporate questions about electronic forms of communication into their history and consider testing these functions as an addition to the neurological exam in patients with language or cognitive complaints as this may have implications for diagnosis and even treatment options.

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Disclosures

Neil Thomas and Tiago Mestre hereby state that they do not have conflicts of interest to disclose.

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