

## SHORT REPORT

## Subcortical infarction resulting in acquired stuttering

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### Abstract

**Stuttering is an uncommon presentation of acute stroke. Reported cases have often been associated with left sided cortical lesions, aphasia, and difficulties with other non-linguistic tests of rhythmic motor control. Three patients with subcortical lesions resulting in stuttering are discussed. In one patient the ability to perform time estimations with a computerised repetitive time estimation task was characterised.**

**One patient had a pontine infarct with clinical evidence of cerebellar dysfunction. A second patient had a left basal ganglionic infarct and a disruption of timing estimation. A third patient had a left subcortical infarct and a mild aphasia. These findings expand the reported distribution of infarction that can result in acquired stuttering. Subcortical mechanisms of speech control and timing may contribute to the pathophysiology of acquired stuttering.**

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**Keywords:** stuttering; stroke; subcortical; infarction

Stuttering has been defined as a disruption of the fluency of verbal expression characterised by the involuntary repetition or prolongation in the utterance of sounds and syllables.<sup>1</sup> Acquired stuttering is infrequent after stroke. Evidence for the physiological basis of stuttering has come from findings of acquired stuttering in adults after vascular lesions or traumatic lesions, as well as from studies of developmental stutterers. Most cases of acquired stuttering with stroke are reported to result from left cortical or bilateral cortical lesions with associated aphasia.<sup>2,3</sup> Subcortical stroke leading to acquired stuttering has been infrequently reported in the literature. We present three cases of acquired stuttering resulting from subcortical infarction and investigate the timing behaviour of one patient, using a computerised repetitive time estimation task.

### Methods

Three cases of acquired stuttering were evaluated with brain MRI. A computerised repetitive time estimation task was performed with

patient 2 and in normal control subjects. Patients were instructed to repetitively press a mouse button in a target interval of 10 to 13 seconds from the prior response. After each response, one of two tones was sounded to indicate whether the response was inside or outside of the target interval. Testing was performed in three blocks of 18 intervals each.

### Results

#### PATIENT 1

A 53 year old right handed man with a history of diabetes and hypertension presented with a 2 day history of episodic vertigo and ataxia. On examination he had a left internuclear ophthalmoplegia, dysarthria, left facial droop, jaw tremor, right dysmetria, and gait ataxia. Cognition and swallowing were normal. Language function disclosed stuttering consisting of rapid speech with consistent repetition of every syllable with intermittent aphonia but no initial prolongations. Stuttering was present on spontaneous speech and with repetition. He showed intermittent velopharyngeal incompetency during connected speech utterances. He was able to decrease the frequency of his stuttering by speaking slowly. Brain MRI showed a linear band of T2 signal hyperintensity in the left rostromedial pons consistent with small vessel infarction (fig 1 A).

#### PATIENT 2

A 54 year old right handed woman with a history of diabetes, hypertension, myocardial infarction, and breast and ovarian cancer awoke with dysarthria. On examination her spontaneous speech, reading, and repetition were slow but grammatically correct with initial prolongations of consonants and occasional repetitions of initial syllables without circumlocutions. Cognitive function was intact. A mild, right facial droop and slowness of right finger movements with preserved strength were noted. Otherwise motor, sensory, and cerebellar examinations were normal. A computerised test of timing behaviour<sup>4</sup> showed impaired ability to make accurate time estimations (fig 2). T2 weighted brain MRI demonstrated infarction in the left putamen extending to the caudate and corona radiata (fig 1 B).

#### PATIENT 3

A 63 year old left handed woman with a history of hypertension and hypercholesterolaemia

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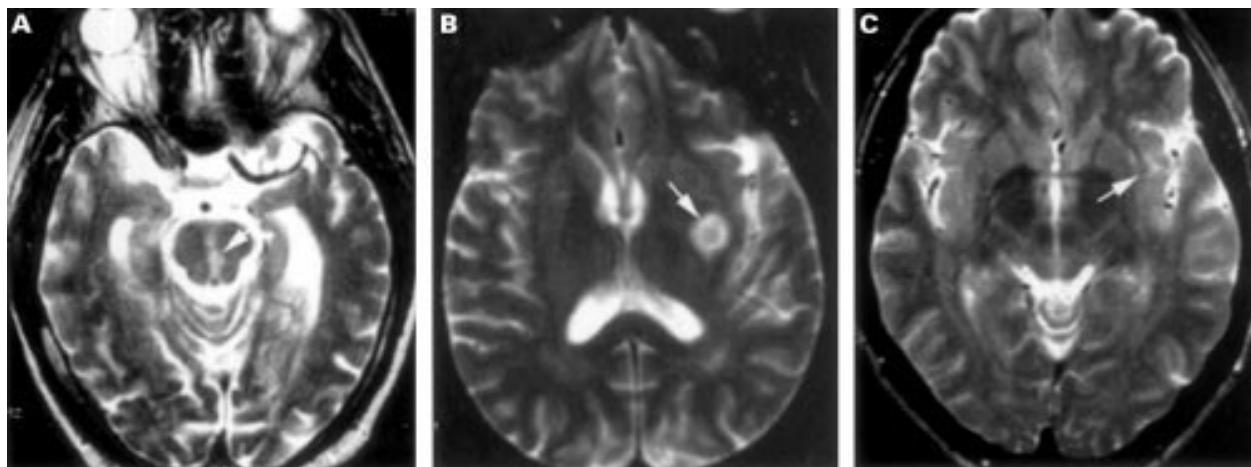


Figure 1 (A) T2 weighted brain MRI of patient 1 showing signal abnormality consistent with infarction in the left rostromedial pons. (B) T2 weighted brain MRI of patient 2 showing signal abnormality in the left putamen, corona radiata, and caudate, consistent with infarction. (C) T2 weighted brain MRI of patient 3 showing a small subcortical infarct in the left corona radiata and a small left lateral putamen and subinsular infarct.

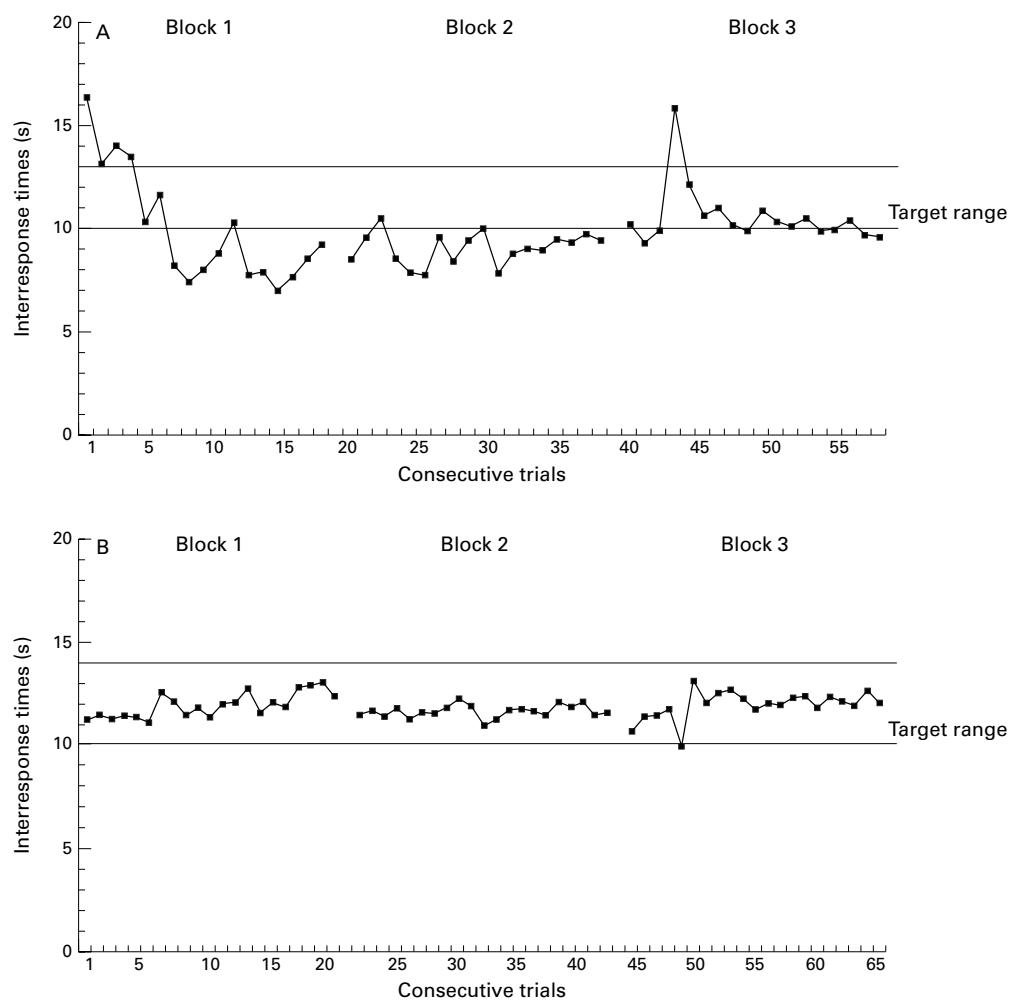


Figure 2 Computerised repetitive time estimation task in (A) patient 2 and in (B) a normal control subject.

experienced a sudden onset of a change in vision, right hand weakness, and difficulty finding words. On examination her comprehension, reading, and writing were intact. Stuttering consisted of repetition of initial syllables during spontaneous speech with occasional initial prolongations. Transcranial Doppler showed increased flow velocities in both middle cerebral arteries. Brain MRI disclosed a small

subcortical infarct in the left corona radiata and a small left lateral putamen and subinsular infarct.

### Discussion

Although most reported cases of acquired stuttering result from left cortical or bilateral cortical lesions with associated aphasia<sup>2,3</sup> (table), the cases we present here show that acquired

## Reported cases of acquired stuttering after stroke with brain imaging available

Reference	Location of stroke
Helm et al 1978 <sup>2</sup>	Right frontal-perisylvian No lesion No lesion Left frontoparietal Left frontal Right parietal Left frontal, temporal, parietal, occipital Left temporoparietal Left frontotemporal Bilateral diffuse Left frontoparietal No lesion No lesion
Rosenbek et al 1978 <sup>3</sup>	Right medial, anterior frontal Right temporal Right frontoparietal watershed Right subcortical Bilateral medial thalamus and midbrain Left centrum semiovale Left striatocapsular Left frontotemporoparietal Medial left occipital
Donnan 1979 <sup>5</sup>	
Rosenfield et al 1980 <sup>6</sup> Ardila and Lopez 1986 <sup>7</sup> Fleet and Heilman 1989 <sup>8</sup> Soroker et al 1990 <sup>9</sup> Abe et al 1993 <sup>10</sup> Lanoe et al 1994 <sup>11</sup> Kono et al 1998 <sup>12</sup> Grant et al 1999 <sup>13</sup>	

stuttering may also result from infarction in subcortical structures including the pons. A pontine infarct as seen in our first patient has not been previously reported to result in acquired stuttering. The persistence of this patient's stuttering on every syllable sets him apart from our other two patients. A patient with bilateral thalamic and midbrain infarcts was reported to show a repetitive speech disorder similar to stuttering.<sup>10</sup> This case was notable for a high number of repetitions of syllables. The authors concluded that as the clinical features of their patient's stuttering were similar to the stuttering of patients with infarcts of the supplementary motor area, thalamic and midbrain projections to the supplementary motor area may have been disrupted. Additionally, alleviation of acquired stuttering with thalamic stimulation has led to the suggestion that the brainstem reticular formation, diencephalon, and basal ganglia are involved in the motor execution of speech through their projections to the frontal cortex.<sup>14 15</sup> We propose that disruption of connections from the brainstem reticular formation to the supplementary motor area in our first patient may have resulted in acquired stuttering.

Studies with PET and SPECT, focusing primarily on developmental stutterers, have implicated multiple neural mechanisms in the generation of stuttering.<sup>16-20</sup> These neural mechanisms include a widespread overactivation of the motor systems in both the cerebral and cerebellar hemispheres with a right predominance, a lack of normal activation of the left, anterior, superior temporal phonological circuits, and a deactivation of a circuit between the left frontal and temporal cortex.<sup>19</sup> However, studies in developmental stutterers have also suggested that subcortical structures may play a part in stuttering. Brain PET studies of developmental stutterers have showed left caudate hypometabolism during stuttering and fluent choral reading.<sup>20</sup> It is important to be cautious when attempting to infer analogous mechanisms from studies of developmental stutterers as they do not have structural brain lesions and a recent study did not show

consistent differences in blood flow at rest compared with controls.<sup>21</sup>

Other mechanisms have been proposed for the generation of acquired stuttering with subcortical infarcts. These include impaired regulatory callosal transmission,<sup>9</sup> damage to circuits connecting the basal ganglia to the cortex,<sup>12</sup> and also cortical dysfunction resulting from subcortical infarction.<sup>11</sup> The last hypothesis was substantiated in one patient with a left striatocapsular infarction by showing cortical deactivation visualised on SPECT imaging.<sup>11</sup> In our second and third patients, it is possible that intrahemispheric connections were disrupted by the extension of the lesion into the subcortical white matter. Alternatively, lesions of the left basal ganglia in these two patients may have led to stuttering independent of disruption of intrahemispheric connections.

Difficulty with non-linguistic skilled motor tasks has been described in patients with acquired stuttering, including difficulty with rhythmic tapping and copying three dimensional figures.<sup>2 22 23</sup> Our second patient demonstrated marked impairment of repeated time estimations. Disruption of the timing of speech control may play a part in generating stuttering.

Multiple mechanisms may exist which can generate acquired stuttering given the diversity of lesions that have been described. Our three patients expand the localisation of lesions that can result in acquired stuttering and show that subcortical mechanisms of speech control and timing may contribute to the pathophysiology of acquired stuttering.

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