SHORT REPORT

Repetitive speech disorder resulting from infarcts in the paramedian thalami and midbrain

Kazuo Abe, Ritsuko Yokoyama, Shiro Yorifuji

Abstract
A repetitive speech disorder resulting from infarcts in the paramedian thalami and the midbrain is reported. Although the speech disorder seemed like stuttering, the compulsory repetitions, constant rate and monotonous tone were not associated with ordinary stuttering. Since repetition was restricted to the first syllable, the speech disorder in our patient could be distinguished from palilalia. The extrapyramidal system is considered responsible for repetitive speech disorders resulting from infarcts in the paramedian thalami and the midbrain but without good reason. Repetitive speech disorder in patients with infarcts in the supplementary motor area (SMA) have similar clinical features to our patient. It is suggested that interruption in the propretic system to the SMA is a possible cause of “stuttering like repetition”.

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Stuttering is characterised by difficulty in producing the next expected sound.1-3 Although congenital stuttering is well known, acquired stuttering resulting from cerebrovascular disease also occurs. Various types of cerebral infarcts can cause acquired stuttering, but stuttering resulting from infarcts in the paramedian thalami and paramedian midbrain has not been previously reported. We report the first case, and discuss its unique clinical features compared with those of other speech disorders resulting from infarcts in the same anatomical location.

Case report
A 38 year old man had been suffering from cardiovascular disease. He was admitted to a hospital and had coronary angiography. After the angiography, he was comatose and developed a tetraparesis. He had dilated non-reactive pupils. Doll’s head movements were preserved. A CT scan 24 hours later showed low density areas in the paramedian thalami and in the midbrain. Three weeks later, he started to respond with simple words to verbal commands. However, he was still drowsy and seemed apathetic. Deep tendon reflexes were slightly increased and Babinski’s sign was noted on both sides. Two months later, he began to speak in long sentences but showed compulsive repetition of syllables, almost always the first syllable, and never words or phrases. Neurological examination at this stage revealed that he was still apathetic and lay on the bed if other instructions were not given. He had isocoric pupils and preserved light reaction. He had disturbed eye movements on right lateral gaze and vertical gaze. He had preserved doll’s head movements and Bell’s phenomenon. Other normal cranial nerve functions were found. He had slightly increased deep tendon reflexes and positive Babinski’s sign on both sides, and normal muscle strength with mild rigidity in the hand joints. There was increased jaw jerk and positive sucking reflex. Coordinate movements were normal. The patient could walk a short distance unaided, but with fluctuating and compulsive gate. Sensory functions were almost normal.

Repetition in his speech was restricted to syllables; repetition of words or phrases was not observed. Repetition of syllables was exclusively observed in spontaneous speech or in reply to a question and was rarely observed when he repeated words spoken to him, or when he read sentences (table). There were no other speech disorders, such as perseveration, literal or verbal paraphasia. For example, when asked what his breakfast was, his answer was “go-go-go-go-go-gohan to misoshiru sorenini ninn-ninn-ninn-ninn-ninn-ninn-nin-ni-ni-ni-ni-nishimetayatsu” (gohan = rice, to = and, soren = and, misoshiru = miso soup, nishimetayatsu = boiling foods).

Mild disorientation to place and time was noticed. Digit span was 6 forward and 3 backward. Recent verbal and visual memory were disturbed. Calculation and writing were also disturbed. Right left disorientation, apraxia and agnosia were absent.

Table Frequency of stuttering is evaluated in four modalities; repetition of spoken phrase, reading phrases, naming, and spontaneous speech. Stuttering is the most frequently observed in spontaneous speech.

<table>
<thead>
<tr>
<th>Category</th>
<th>Frequency</th>
<th>Rare if any</th>
<th>Seldom</th>
<th>Sometime</th>
<th>Every sentence</th>
<th>Every two syllables</th>
<th>Every syllable</th>
</tr>
</thead>
<tbody>
<tr>
<td>repeat</td>
<td>5% (1/22)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>reading</td>
<td>9% (2/22)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>naming</td>
<td>28% (8/28)</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>speech</td>
<td>58% (14/24)</td>
<td></td>
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</tr>
</tbody>
</table>
Labouboty findings including complete
blood count and serum calcium, blood urea
nitrogen, creatine phosphokinase, alkaline
phosphatase, and glutamic transaminase were
normal. An ECG was normal.

The basic rhythm on an EEG was 8 Hz
alpha waves with complete alpha attenuation,
and sporadic 5–6 Hz theta waves were seen
bilaterally. A brainstem auditory evoked
potential (BAEP) showed no abnormal find-
ings. MRI showed infarcts in the bilateral
medial thalami and in the midbrain (fig).

The repetitive speech disorder has contin-
ued for more than two years, although his
daily activity gradually improved. Instability
in walking was slightly improved, but there
was no improvement in right eye movement.

Discussion

Stuttering can be defined as a deviation of
speech attracting attention of speakers or lis-
teners because of interruption of the normal
rhythm of speech by involuntary repetition,
prolongation or arrest of sounds. Acquired
stuttering, mainly resulting from cerebral
infarcts, has been described in disorders in
the corpus striatum, the dominant temporal
lobe, the dominant parietal lobe, or in the
dominant operculas.1–3 Various types of
speech disorders resulting from infarcts in the
paramedian thalami and the midbrain have been
reported, but stuttering has not been
described. Castaigne4 studied patients with
paramedian thalamic and midbrain infarcts
and described some patients with mutism. He
did not, however, report patients with repeti-
tive disorders. Yasuda5 reported a patient
with repetitive speech disorder resulting from
infarcts in the paramedian thalami and mid-
brain and they diagnosed it as palilalia. The
repetition shown by their patient was not
restricted to the first syllables and showed
gradually increasing rate with reduced loud-
ness. In contrast, our patient showed repeti-
tions restricted to the first syllables. Restricted repetition to the first syllable is a
characterised feature of stuttering.5 Our
patient did not demonstrate an increasing
rate with reduced loudness, which is known
as “palilalia aphone”. Instead, he repeated the
first syllables at a constant rate and loudness.
His repetitive speech can therefore be distin-
guished from palilalia or logoclonia.

The repetitive speech disorder in our
patient was unique for its extraordinary fre-
cuency of repetitions. He repeated the first
syllables of words no less than seven times at
a slow and constant rate, which was unlike
stuttering. Conversely, palilalia resulting from
infarcts in the paramedian thalami and mid-
brain showed repetitions of syllables or parts
of words more frequently than repetitions of
words or phases, which does not fit the crite-
rion for palilalia.5–6 Since repetitive speech
disorders resulting from infarcts in the para-
median thalami and midbrain have charac-
teristic clinical features, it is natural to
 distinguish them from stuttering and palilalia.
We therefore propose to call this repetitive
speech disorder “stuttering like repetition”.

Although the pathogenesis of “stuttering
like repetition” is unclear, it seems that it
results from extrapyramidal dysfunction2,8 as
in palilalia. “Stuttering like repetition”, how-
ever, has clinical features which differ from
those of palilalia, and our patient did not
have extrapyramidal signs. We consider that
the causative lesion for “stuttering like repeti-
tion” can not be restricted to the extrapyra-
midal system. Nagafuchi reported a patient
with stuttering resulting from infarcts in the
supplementary motor area (SMA), and the
clinical features of their patient were compat-
ible with ours.8 As some patients with infarcts
in the SMA demonstrated uncontrollable
interruption of speech or utterance, the SMA
might play a significant role both in initia-
tion and in control of spontaneous speech.10–12

The infarcts of our patient anatomically
located in the centromedial thalami and to the cerebral peduncles which are part of the projection system to the SMA.13 We concluded that interruption of the projective system to the SMA might cause “stuttering like repetition”.

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