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Alleviation of Acquired Stuttering with Human Centremedian Thalamic Stimulation

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Summary: Despite many investigations, the cerebral mechanism for stuttering remains unknown. Recently, increased attention has been paid to acquired stuttering of adult onset in the hope that the events associated with it might provide clues to the biological mechanism underlying stuttering. This attention has focused exclusively on the cortical substrates. We present our observations of acquired dysfluency, presumably of subcortical origin in a neurosurgical subject with intractable pain. The stuttering was relieved by thalamic electric stimulation. The effect of thalamic stimulation on the stuttering suggests that the pathophysiology of transient asynchronisation in the balancing and sequencing of multiple impulses is amenable to a diffusely orchestrated functional tuning of the thalamic and brainstem implicated subcortical structures and pathways.

Cortical pathologies in adults often produce stuttering (prolongations, involuntary repetitions and hesitational blocks). The

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onset of acquired stuttering in adults can occur with dysarthria, aphasia and head trauma.[1-5] Clinically, acquired stuttering differs from the developmental form; speech blocks are not confined to word initial sounds and syllables alone.[2] It is not associated with secondary symptoms such as articulatory struggles, anxiety, tension facial grimaces and contortions. The acquired rhythm disturbances are usually transient; however, they may persist if the underlying pathology is fixed and bilateral.[3] The anatomical mechanism for acquired stuttering has been primarily attributed to the cortical substrates.[1-5] Very little attention has been given to subcortical structures. This study presents acquired stuttering symptoms of subcortical aetiology and suggests that electrical discharges in a malfunctioning brainstem may generate speech blocks. It also demonstrates that attenuation of the discharge by thalamic stimulation alleviates the stuttering.

Methods and procedures

Patient History

A 61-year-old male with normal intelligence (WAIS) had a 20 year history of trigeminal pain which started as tic douloureux. During a period of 18 years he was treated with a variety of medications and bilateral trigeminal ganglion blocks. He underwent three trigeminal rhizotomies on the right and one on the left. The pain became progressively worse. Additional symptoms were absence attacks, tremor, photophobia, inattention, double vision and stuttering. The speech blocks consisted of syllable or word reiterations, or sound prolongations, tonic blocks and hesitations. The stuttering progressively deteriorated. He described his blocks as predictable spasmodic episodes of varying lengths affecting his ability to articulate. The rhizotomies and the drugs adversely affected his speech. Sansert was the only drug which smoothed the dysfluency blocks; however, it adversely affected his kidney functions.

In a preoperative assessment there was no aphasia, memory or neuromotor speech impairment but there was a marked speech dysfluency. The stuttering was measured by controlled conversation and a specific reading task; equal amount of dysfluency was noted on

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both tasks. On reading, he stuttered on 58 out of 172 words. The patient showed no adaptive effect on repeated readings, anxiety or any secondary mannerisms. His dysfluencies primarily consisted of mostly predictable motoric articulatory spasms.

Thalamic stimulation electrode implant for chronic pain (anesthesia dolorosa)

A chronic stimulation electrode was stereotactically implanted[6] in the left centremedian nucleus for the relief of chronic pain. The site of stimulation is illustrated in the sagittal plane (fig 1), according to the atlas by Schaltenbrand and Baily.[7] The tip of the stimulating electrode was 3 mm in length and 0.5 mm in diameter. Unipolar stimulation was used with the indifferent electrode implanted in the infraclavicular subcutaneous tissue. A transmitter antenna, attached to a 9v battery operated stimulator, was placed over the receiver antenna to activate the stimulation electrode. The subject was instructed to self-stimulate three to four times per day for 20 minutes. Stimulation parameters (50 Hz, 2.5 V, 200 μsp) were below threshold for sensory motor alterations. The stimulation of the left thalamus had a remarkably beneficial effect on the speech dysfluencies. There also was a considerable reduction in the pain.

Stimulation evoked changes in metabolic activity of the brain

The effect of electric stimulation in the left centre-median nucleus upon the cerebral metabolic activity was evaluated by Single Photon Emission Computed Tomography (SPECT), using 3.5 millicuries of radiopharmaceutical I-123 lodoamphetamine 8. Two (pre- and post-thalamic stimulation) SPECT studies, using the radiolabelled amine N-isopropyl-p-(123 I) lodoamphetamine (IMP), were performed. Data collected was started 20 minutes after IMP injection. Transverse, sagittal and coronal images were displayed as 8 mm thick (two pixel thick) sections. The transaxial images were in the CT orientation (10-15 degrees from the orbital-meatal line). The SPECT images revealed a considerable metabolic increase bilaterally in the thalamus from the left CM stimulation (fig 2) with no metabolic alteration at the cortical level.

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Result

Within the first three weeks of daily self-stimulations, there was a noticeable reduction of the stuttering (syllable/word reiterations, hesitations and sound prolongations). After eight weeks of continuous selfstimulations, only three instances of hesitation and two word repetitions were noted in a ten-minute conversation. He described the effect of stimulation as smoothing on motor-speech spasms. On a scale of 1-10 (with 1 being normal and 10 being maximum deficit), he rated his pre-thalamic surgery level of dysfluency as 8-9 and after the surgery as 1 to 2. At present he is free of any perceptible dysfluency and has remained free of a stutter for the past two and a half years of self-stimulation.

Comment and discussion

These observations suggest that cortically generated motor speech schemas are integrated through subcortical structures, such as the thalamus, for fluency, accuracy synchronisation motor commands and rhythmic continuity. The mini discharges which were considered secondary to fifth cranial nerve denervation and which were thought to account for this patient's anesthesia dolorosa[6] were also most likely to start the stuttering. Bilateral abnormal electrical discharges if present may be attenuated since SPECT studies[8] revealed bilateral increased blood flow and metabolism in response to the unilateral stimulation. The beneficial effect of thalamic stimulation on the stuttering suggests that the pathophysiology of transient asynchronisation in the balancing and sequencing of multiple impulses is amenable to a diffusely orchestrated functional tuning of thalamic and brainstem subcortical structures and pathways. These observations further stress the importance of thalamic and neighbouring subcortical structures in providing electrophysiological generators for the modulation and tuning of motor speech mechanisms.

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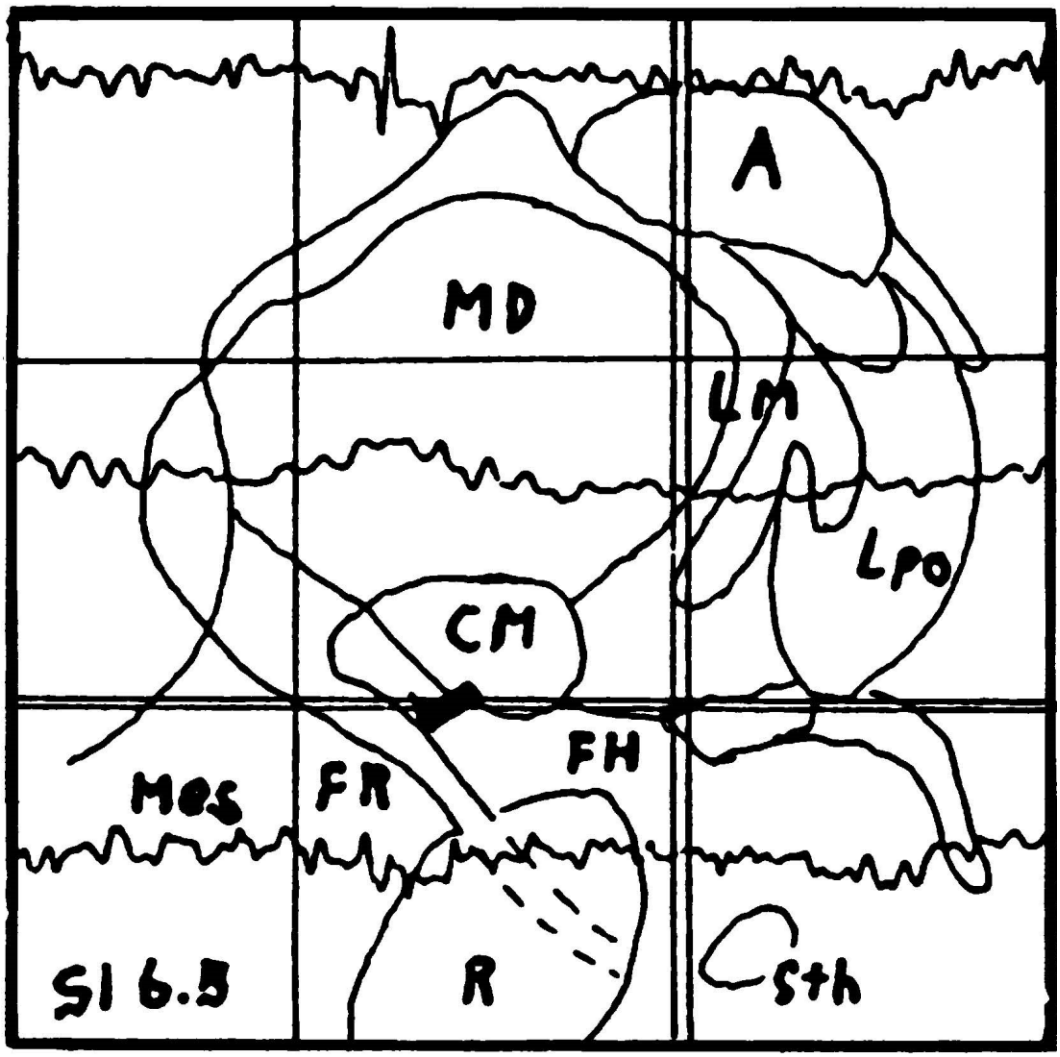

Fig 1. Electric stimulation site illustrated in the sagittal view of the mesodiencephalon 6.5 mm from midline. Squares are 10 × 10 mms, horizontal double line is horizontal zero plane and the vertical double line is the mid thalamic frontal zero plane. A, anterior thalamic nucleus; CM, centremedian nucleus; FH, fiel H of Forel; FR, fasciculus retroflexus; LM, lamina medularis interna; Lpo, lateralis Polaris; MD, medial dorsal nucleus; Mes, mesencephalon; R, red mucleus; Sth, subthalamic nucleus.

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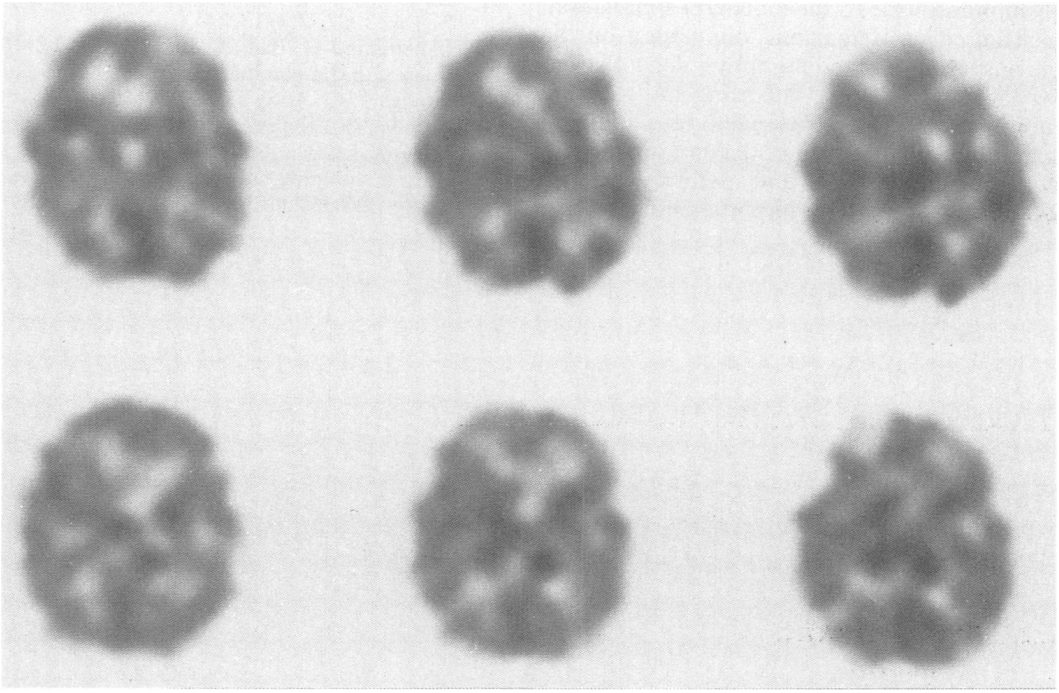

Fig 2 Comparable transaxial nonstimulation (upper) and stimulation (lower) SPECT images: a noticeable thalamic increase in the bilateral metabolic activity is evident after the stimulation (lower images).