

SHORT REPORT

Cerebellar voice tremor: an acoustic analysis

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Abstract

Patients with cerebellar disease may exhibit tremulous phonation as part of their dysarthria. The results of an acoustic analysis of cerebellar voice tremor in a patient with hereditary ataxia and presenting with a purely cerebellar syndrome are reported. Analysis included computation of speech intensity contours, fundamental frequency contours, and spectral parameters from sustained productions of vowels and voiceless fricatives. Fundamental frequency contours during sustained phonation of vowels showed rhythmic oscillations at a rate of about 3 Hz. No concomitant periodicity could be detected for the parameters characterising voiceless fricative production. The results indicate an impairment of phonatory control in relation to the maintenance of a constant isometric activity of the internal laryngeal muscles. Cerebellar voice tremor may therefore be classified as a form of postural tremor.

A variety of tremors have been described in cerebellar lesions and diseases.¹ Among other tremors cerebellar patients sometimes exhibit tremorous phonation as a component of their dysarthria.^{2,3} To date no quantitative studies of cerebellar voice tremor are available to provide information about the underlying mechanism. Tremulous activity of different muscle groups may contribute to quavering speech, for example, respiratory, laryngeal, neck, and orofacial muscles. Cerebellar degeneration can result in tremor of the head and/or tongue.^{4,5} It is unclear, however, if these motor disturbances are responsible for the tremulous voice and speech in cerebellar patients. On essential voice tremor it has been shown that tremor of the respiratory muscles may result in rhythmic modulation of sound pressure level and hence contribute to quavering speech.⁶ But in cerebellar patients comparable evidence is lacking. Furthermore, laryngeal control in cerebellar voice tremor has not yet been studied.

We report the results of an acoustic analysis of voice tremor in a patient with chronic cerebellar atrophy.

Case report

A 63 year old woman presented with severe ataxia of gait and stance, marked limb ataxia

and dysarthrophonia at clinical examination. At the age of 50 years the patient had noticed difficulties in walking and in speech as the first symptoms of cerebellar disease. During follow up no extra-cerebellar signs or symptoms developed apart from a slight impairment of vibration sense. Computerised tomography (CT) and magnetic resonance imaging (MRI) showed a diffuse atrophy of the cerebellum; the brainstem and cerebrum were not affected. Cerebrospinal fluid (CSF) investigations, somatosensory evoked potentials following median and tibial nerve stimulation and acoustically evoked brainstem potentials disclosed no abnormalities. Electro-oculography documented cogwheeled smooth pursuit, saccadic dysmetria and disturbed VOR cancellation. Inspection of the oral cavity at rest and during phonation did not reveal palatal myoclonus.

The patient had a reduced speech rate (3.5 syllables per second in sentence repetition) and "scanning speech". Her conversational pitch was high (with an average of 260 Hz in sentence repetition) and her voice was weak, rough, and breathy. During running speech, voiced segments were occasionally realised as voiceless, whispery sounds. When asked to sustain a vowel sound tremulous phonation could be heard, sometimes intermittently and sometimes constantly. In contrast, the productions of voiceless fricatives (/f/, /sh/, /s/) did not show tremulous quavering. Neither tremorous activity of the orofacial and pharyngeal muscles nor tremor of the limbs, the head or the trunc was observed during sustained vowel sounds.

A sister of the patient, a paternal aunt and her son, had also suffered from a cerebellar syndrome with onset at the age of about 50 years. Autosomal dominant purely cerebellar hereditary ataxia was diagnosed.

Speech recording and processing

Speech testing included sustained phonation on /u/ and /a/ "as long as possible on a single breath", the production of single isolated vowels (/a/, /u/, /i/, /y/; six times each for "approximately two seconds", in a randomised order), and the sustained production of voiceless fricatives (/f/, /sh/, /s/; "as long as possible on a single breath"). Whereas the vowel production tasks examine the patient's faculty of maintaining a stable laryngeal configuration, the fricative task requires the maintenance of stable articulatory configurations of lips and jaw (/f/), tongue blade and jaw (/s/), and tongue dorsum and jaw (/sh/),

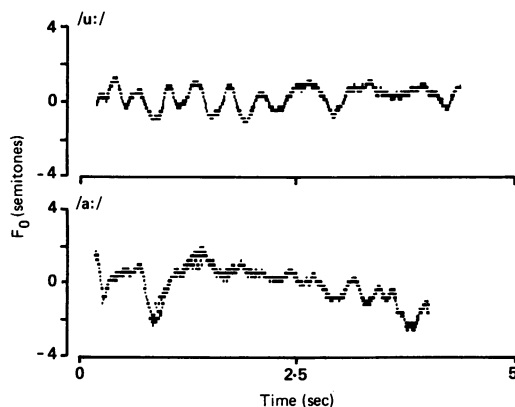
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Figure 1 Time course of fundamental frequency (F_0) during sustained production of the vowels /u:/ and /a:/. According to generally accepted conventions F_0 is measured logarithmically (that is, in semitones).



respectively. In both tasks, a constant transglottal flow must be maintained through respiratory control.

The examination was performed in a sound-treated test booth and recorded on magnetic tape using high quality equipment. The audio recordings were low-pass filtered at 9 kHz, digitised at a 12-bit resolution and a sampling frequency of 20 kHz, and fed into a LSI 11/73 laboratory computer for off-line analysis. Acoustic analysis included the computation of the time course of fundamental frequency (fundamental frequency contour) for vowels (in Hz), fricative spectra (512 point FFT every 12.8 ms, hamming window) and speech intensity contours (in dB) (for further details see ⁷).

The *speech intensity contour*, an acoustic correlate of perceived loudness, reflects the acoustic energy of the emitted sound during sustained phonation and sustained fricative production. Alterations of respiratory pressure should show up in the intensity contours of both vowel and fricative tasks.

Fundamental frequency measures the rate at which the vocal cords open and close during phonation. The fundamental frequency determines the pitch of speech and is primarily sensitive to alterations in vocal fold stiffness.

The *fricative spectrum* describes the distribution of energy over the frequency range during the production of sustained voiceless fricatives. The shape of the fricative spectrum reflects the airflow characteristics associated with the articulatory configuration during fricative articulation.⁸ It can therefore be used

as a measure of articulatory stability of fricative production over time. In this study, the spectral shape of fricatives was modelled by the spectral centre of gravity. This parameter provides an index of the locus of energy concentration along the frequency axis and varies with the anterior-posterior position and the configuration of the tongue. Thus it should reflect alterations of the articulatory configuration required in the production of sustained /f/, /s/, and /sh/, respectively (see ⁷).

Results

Figure 1 presents fundamental frequency contours during sustained phonation on /u/ and /a/. The high jitter in these contours is an acoustic correlate of irregular vocal fold oscillations and of perceived roughness of the voice. More importantly, the two contours demonstrate an intermittent rhythmic modulation of vocal pitch over a range of approximately two semitones. Similar contours were obtained for the isolated vowel productions ($n = 24$) as well. Power spectrum analysis disclosed a dominant frequency of 2.8 Hz.

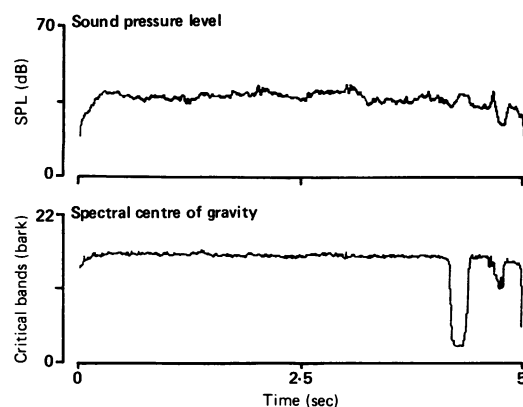
During the patient's sustained production of voiceless fricatives no rhythmic modulation of either sound pressure level or spectral centre of gravity was present (fig 2). The sudden drops in the centre of gravity contour of /sh/ seen in fig 2 reflect episodes of spontaneous voiced phonation, probably due to involuntary vocal fold adductions.

Discussion

In a patient with diffuse cerebellar atrophy and presenting with audible voice tremor acoustic analysis disclosed rhythmic oscillations of sound intensity and fundamental frequency of about 3 Hz during sustained phonation. The frequency of cerebellar voice tremor measured in our patients is similar to that reported for other forms of cerebellar kinetic and postural tremor. Most authors cite a frequency of about 3 Hz for intention tremor.¹ The postural tremors of the lower limbs in alcoholic cerebellar degeneration⁹ and the postural anterior-posterior sway in standing patients with anterior lobe atrophy of the cerebellum¹⁰ reveal the same rhythmicity. Finally, Mai *et al*¹¹ observed a force tremor with a frequency of about 3 Hz during maintenance of isometric finger contraction.

In our patient fluctuations of fundamental frequency could be detected, that is, oscillations of pitch contributed to quavering speech. Fundamental frequency measures the rate at which the vocal folds open and close. The vibrations of the vocal folds is a purely passive process resulting from the interaction of subglottal air pressure built up by the respiratory system and vocal fold tension set by the internal laryngeal muscles.¹² Therefore, both fluctuations of subglottal air pressure and fluctuations of vocal fold tension are possible causes of voice tremor. Further, through the coupling of larynx and tongue by extrinsic laryngeal muscles even changes in articulatory configurations

Figure 2 Time course of sound pressure level (SPL) and spectral centre of gravity during sustained production of /sh/ (30 Hz low-pass filter). Both measures are given in logarithmic units, that is, in dB and barks, respectively.



may result in fundamental frequency alterations. Thus rhythmic modulation of fundamental frequency is ambiguous concerning the underlying pathomechanism.

Oscillatory changes of subglottal air pressure sufficient for the generation of voice tremor should be reflected in the speech intensity contour, irrespective of whether the vocal folds are in an adducted or in an abducted position. In our patient the speech intensity contours of the sustained voiceless fricatives showed no rhythmic modulation, suggesting that the respiratory system did not contribute to any significant extent to the observed tremor. Even the spectral characteristics of the fricatives remained stable, meaning that no tremulous activity was present in the articulators either. In line with these results auditory evaluation of sustained voiceless fricatives did not reveal any tremulous sounds. Thus cerebellar voice tremor in this patient indicates an impairment of phonatory rather than articulatory or respiratory control. During sustained phonation the internal laryngeal muscles are in a state of isometric contraction and thus the cerebellar voice tremor has to be considered as a postural tremor.

Holmes suggested that cerebellar postural tremor reflects voluntary corrections compensating for the drifting of hypotonic muscles.¹³ Recent experimental work emphasises instead the significance of stretch reflexes in the generation of postural tremor.^{14,15} It is known that during isometric contraction muscle spindles show high dynamic sensitivity to small length changes.¹⁶ Laryngeal muscles are densely supplied with muscle spindles, whereas lips and tongue muscles have few or none. During

position maintenance of lips and tongue while realising sustained fricatives (/f/, /sh/, /s/) no rhythmic oscillations could be detected. Thus the results of the analysis of cerebellar voice tremor fit into current conceptions of cerebellar postural tremor.

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