

Generative music processes in Parkinson's disease:

facilitation of motor behavior by internally
generated stimuli

R. Harris

2/1/2011

***Abstract** Prosodic anomalies in the speech of patients suffering from Parkinson's disease (PD) are generally attributed to rigidity and brady- and hypokinesia, especially of the laryngeal musculature. However, the inability of patients to recognize or produce prosodic distinction between questions and statements points at impairment of higher cognitive function implicated in language. Recently, a similar inability of congenital amusics to recognize prosodic distinction has been demonstrated, suggesting that musical pitch discrimination and prosodic discrimination in speech are interrelated. Musical stimuli are known to facilitate motor behavior such as gait in PD. Similarly, one could expect music to facilitate other forms of motor behavior, for example speech. Indeed, singing and rhythmic tapping have been used to facilitate speech in the treatment of expressive aphasia (Melodic Intonation Therapy). In the first case, music functions as an external cue, in the second as an internal cue. If in PD, as in expressive aphasia, the impairment of verbal expression is restricted to the language domain, generative music ability might remain intact. In that case, whistling a familiar or improvised tune might still reveal regularity of pitch level and a normal statistical distribution of scale tones. The possible dissociation of speech and music production in PD patients would provide insight into the role of music as an internally generated stimulus in cerebral motor control. Alternatively, if such dissociation cannot be demonstrated, one may infer the existence of a common musical-linguistic neuronal source.*

1. INTRODUCTION AND RATIONALE

Parkinson's Disease (PD) is a progressive neurological disease with motor symptoms including rest tremor, rigidity and bradykinesia. It is characterized by progressive necrosis of dopaminergic neurons primarily in the substantia nigra pars compacta, but also in other areas of the brain (Goberman & Coelho 2002). The speech dysfunction resulting from PD is typically classified as hypokinetic dysarthria, the term hypokinetic referring to reduced amplitude of movement.

Prosody is the term applied to the natural variations in pitch, intensity, and rhythm occurring during running speech. The frequently documented higher fundamental pitch (F_0) in speech production among PD patients, i.e. higher speaking voice, is generally attributed to rigidity of the laryngeal musculature, resulting in increased stiffness of the vocal folds. In addition, various researchers have also found a low mean F_0 difference during the production of question-statement pairs and differentiation between noun phrases (a black *board*) and compound nouns (a *blackboard*), compared with normal speakers.

Individuals with basal ganglia disease exhibit abnormally reduced sensitivity to the emotional significance of prosody in a range of contexts which cannot be attributed to changes in mood, emotional-symbolic processing or estimated frontal lobe cognitive resource limitations in most conditions (Pell 2003). Several findings point to the possibility that hypokinetic dysprosody is not simply a manifestation of the rigidity underlying brady- and hypokinesia (Penner et al. 2001). In the domain of speech-associated motor control, it has further been observed that speakers with PD have increased difficulty with heterogeneous sequences, where they have to plan a complex sequence of gestures, switching motor programs along the way (Marsden 1989; Ackermann et al. 1995; Ho et al. 1998). It has also been observed that PD patients with no indication of dementia had difficulty generating word lists independent of their articulatory status (Gurd et al. 1998), suggesting that there might be a cognitive component above the basic motor disorder.

Given the fact that a variety of sensory cues may either enhance or disrupt the natural sequences of movement in PD, music might act as one of such stimuli. While there is an overall consensus that Freezing of Gait (FoG) does not occur randomly, and that sensory cues can both trigger and ameliorate FoG in PD, the relative contributions of the modality of sensory cues to this phenomenon and of other more internally-generated modulation, such as by attention or emotional stimuli, remain unclear (Rahman et al. 2008). In this respect, the interaction of music and motor control can be studied along two general approaches: (i) music may be used as a stimulus for motor performance, in e.g. gait, which is essentially independent from it,

and (ii) music may be an intrinsic component of motor performance, which is the case in singing and dancing.

The strong embedding of music as a general cerebral function may be inferred from the fact that various studies in healthy subjects have demonstrated that without absolute pitch the original pitch level of well-known melodies can nevertheless be accurately reproduced (Halpern 1989). In addition, in maternal singing, song repetitions across time reveal constancy in pitch level (tonality) and tempo (Bergeson & Trehub 2002). It thus seems possible that the susceptibility of Parkinson patients to musical cues might allow them to maintain pitch and tempo in the musical domain.

Hypothesis

Normal individuals without perfect pitch nevertheless accurately replicate the pitch level and tempo of familiar tunes when singing. While the speech of PD patients is characterized by a higher mean fundamental frequency F_0 (higher speaking voice), lesser prosodic variability and slower tempo, intrinsic effects of music on PD patients might include:

1. maintained ability to replicate pitch level and tempo while humming or whistling familiar tunes
2. normal distribution of scale tones while humming or whistling improvised tunes
3. maintained ability to replicate the tempo of a familiar song while reciting the lyrics in the rhythm of the song

These findings would demonstrate that music may act as an internal stimulus facilitating cerebral control of the motor components in speech production.

2. OBJECTIVES

To obtain more fundamental insight into the sensorimotor integration in Parkinson's disease; in particular the relationship between specific cognitive impairments, rigidity and brady- and hypokinesia and prosodic and melodic anomalies.

3. STUDY DESIGN

1. As a baseline measurement PD patients and healthy controls will first be requested to perform (once) a spoken task: state their name, place of residence, address, date and place of birth in a normal speaking voice, using complete sentences, for example:
My name is John Doe.
I live in Groningen.
My address is 21 Main Street.
I was born on February 29, 1964 in ten Boer.
2. The second task will be to recite two stanzas of the lyrics of a familiar song in the rhythm of the song. As a stimulus for this task, patients will be asked to bring a familiar song to mind which they have come to know by heart through frequent exposure, for example:

Zie ginds komt de stoomboot uit Spanje weer aan.
Hij brengt ons Sint-Nicolaas, ik zie hem al staan.
Hoe huppelt zijn paardje het dek op en neer,
hoe waaien de wimpels al heen en al weer.

Zijn knecht staat te lachen en roept ons reeds toe:
'Wie zoet is krijgt lekkers, wie stout is de roe!'
Oh, lieve Sint-Nicolaas, kom ook eens bij mij
en rijd toch niet stilletjes ons huisje voorbij!
3. The third task is to hum or whistle the same song twice.
4. The fourth task is to sing both stanzas of the song by heart (with lyrics).
5. Finally, the fifth task is to hum or whistle continuations to interrupted phrases. Stimuli will consist of a minimum of five recordings of antecedent phrases. Subjects will be asked to hum or whistle improvised continuations when the phrase is interrupted.

Verbal and musical performance will be digitally recorded. Characteristics such as syllables, musical tempo and pitch will be quantitatively analyzed (see statistical analysis). Combined duration of the test is estimated to be fifteen to thirty minutes

and can be performed in conjunction with the patient's appointment with the neurologist.

4. STUDY POPULATION

4.1 Population (base)

PD patients from the Neurology ward of the UMCG and healthy controls not suffering from congenital amusia, matched for age and gender. Patients and controls are older than eighteen.

4.2 Inclusion criteria

PD patients (n = 15) will be selected from a database from the 'werkgroep bewegingsstoornissen' from the department of Neurology of the UMCG. Both patients with a recent diagnosis as patients with more advanced symptoms will be selected. There is no age limit.

Patients will be tested, either without taking Parkinson medication since onset of the day of testing or at end of dose, i.e. just before next dose of medication is regularly taken.

Healthy controls (n=15) with similar age and gender will be recruited.

4.3 Exclusion criteria

Neurological diseases other than Parkinson; absolute deafness; congenital amusia.

5. METHODS

5.1 Study parameters/endpoints

5.1.1 Main study parameter/endpoint

Mean pitch; mean inter-onset interval; statistical distribution of scale tones; mean squared (pitch) interval.