

Stuttering and sensory gating: A study of acoustic startle prepulse inhibition

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Abstract

It was hypothesized that stuttering may be related to impaired sensory gating, leading to overflow of superfluous disturbing auditory feedback and breakdown of the speech sequence. This hypothesis was tested using the *acoustic startle prepulse inhibition* (PPI) paradigm. A group of 22 adults with developmental stuttering were compared with controls regarding the degree of PPI. No significant differences were found between the stuttering adults and the control group; the groups showed similar means and distribution. Likewise, no relation between the degree of PPI and the effect of altered auditory feedback on stuttering was found. In summary, the results of the study indicate that there is no relation between stuttering and PPI.

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1. Introduction

Stuttering is a speech motor disorder of poorly understood etiology. It has been suggested to be related to anomalies of the basal ganglia functioning (Alm, 2004; Rosenberger, 1980; Victor & Ropper, 2001), but also to increased reflex gain in brain stem nuclei (Zimmermann, 1980). It is clear that stuttering in some way is related to the auditory functions, as demonstrated by the often dramatic reduction of stuttering by different types of altered auditory feedback (Kalinowski, Armson, Roland-Mieszkowski, Stuart, & Gracco, 1993; Van Riper, 1982). The *acoustic startle prepulse inhibition* (PPI) paradigm is a method to investigate aspects of the basal ganglia, the brain stem, and auditory processing (Feifel, 1999; Swerdlow & Geyer, 1999), which makes it a potentially interesting method for the investigation of the pathophysiology of stuttering.

The most common way to measure startle responses in humans is to elicit an eyeblink reflex by means of a surprising brief noise, and to measure the magnitude of the eyeblink using electromyography (EMG) of the orbicularis oculi contraction. The term “prepulse inhibition” refers to the phenomenon that a weak sound preceding the loud sound, by 15–400 ms, usually results in a diminished startle response (Blumenthal, 1999). Deficiency in this inhibitory function has been found in schizophrenia, Huntington’s disease, obsessive-compulsive disorder, and Tourette syndrome (Blumenthal, 1999). It should be noted that the degree of PPI is independent of the magnitude of the startle reflex (Swerdlow, 1998). PPI is a quite stable phenomenon, which has been shown to be active even during sleep (Silverstein, Graham, & Calloway, 1980). Startle prepulse inhibition has not yet, as far as the author knows, been tested on persons with stuttering.

The inhibitory effect of the prepulse is exerted in the pons, but the degree of PPI is assumed to be determined by descending forebrain circuits from the basal ganglia (Swerdlow & Geyer, 1999). Pharmacological facilitation of dopamine activity tends to reduce PPI, whereas blockage of

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dopamine receptor type D2 with antipsychotic medication tends to increase PPI (Swerdlow and Geyer). This is in line with the observations mentioned above, of decreased PPI in schizophrenia and Tourette syndrome, two disorders which are treated with D2 antagonists. The D2 antagonist haloperidol is the medication that has the best documented effect on stuttering (Brady, 1991). Furthermore, there is a report that 45% of persons with Tourette syndrome stuttered as children (Ludlow, 1993). Another link between stuttering and dopamine comes from a brain imaging study using FDOPA-PET (Wu et al., 1997), intended to measure the rate of dopamine synthesis in the brain. The three persons who stuttered showed about three times higher uptake of FDOPA in parts of the basal ganglia compared with the controls.

PPI has been interpreted as an expression of *sensory gating* or *perceptual filtering* (Blumenthal, 1999), that is, a function for inhibition of disturbing stimuli. Stuttering tends to be improved when manipulating the auditory feedback, with frequency shift, delay, or masking noise (Burke, 1969; Kalinowski et al., 1993; Van Riper, 1982). Van Riper (1982) claimed that he had authenticated a case where an adult male, with severe stuttering since childhood, “immediately stopped stuttering completely after an accident in which he became completely deafened” (pp. 383–384). One hypothesis is that the effect of altered auditory feedback on stuttering is related to an attenuation of the effective auditory feedback. If this is the case, stuttering might result as a consequence of disturbing superfluous auditory feedback due to impaired sensory gating. In summary, several lines of reasoning lead to the possibility of a relation between stuttering and impairment of PPI.

The present study is an attempt to investigate if PPI is lower in persons who stutter than in controls, and if low PPI is related to a positive effect of altered auditory feedback in stuttering persons (as a test of the hypothesis that the effect of altered auditory feedback on stuttering is related to impaired sensory gating).

2. Method

2.1. Participants

2.1.1. Exclusions criteria

Exclusion criteria for control persons were personal history of stuttering or cluttering, stuttering or cluttering in close relatives, neurological or psychiatric disorders, or use of medication affecting the nervous system. One control person and one person with stuttering were excluded because of noted impairment of hearing (based on an interview). Five stuttering persons were excluded because of antidepressant medication.

2.1.2. Interrupted tests

One woman with stuttering chose to refrain from the startle test because of suspected sensitivity to loud sounds. One female participant in the control group interrupted the

startle test after the first startle trial because of perceived discomfort. This implies that one sound-sensitive person from each group was excluded.

2.1.3. Age and sex

Twenty-two stuttering persons (17 males, 5 females, age 19–58, mean age 38.8 years) and 22 controls without speech problems, matched for sex and mean age (age 24–60, mean age 39.2 years).

2.1.4. Recruitment and diagnosis

Eleven of the stuttering participants were previous patients at a clinic of phoniatrics, 8 were members of a local support group for stuttering persons, and 3 were recruited after they had contacted the research team. All cases showed symptoms of stuttering according to DSM-IV-TR diagnostic criteria (American Psychiatric Association, 2000), and regarded themselves as having problem with stuttering.

2.1.5. Altered auditory feedback test

Only participants with marked stuttering during normal auditory feedback were included in the analysis of the effect of altered auditory feedback (AAF index), resulting in a total of 15 stuttering participants (10 males, 5 females).

2.1.6. Ethical approval

The study was performed as part of a larger study of stuttering, approved by the Lund University Research Ethics Committee.

2.2. Apparatus and stimuli for evaluation of startle

The pulses consisted of 50 ms periods of 106 dB (A) white noise with nearly instantaneous rise time, presented with a background of 56 dB (A) continuous white noise. The purpose of the background noise was to facilitate startle responses (Putnam & Vanman, 1999). The prepulses consisted of white noise with a peak sound level of 71 dB (A) and 10 ms rise time from 56 to 71 dB (A). The total prepulse duration was 50 ms, with onset 90 ms before the pulses. The stimuli were presented by Ear Tone 3A insert earphones with plastic tubes to the ears (E-A-R Auditory Systems). The frequency characteristics of the insert earphones resulted in a frequency range of approximately 70–6000 Hz. The sound level was calibrated using the sound level meter Brüel and Kjaer type 2209. Before each test the sound level was checked using the sound level meter Quest Technologies 2100 (with calibrator QC-10).

2.3. Procedure

The test of startle was included as one part of a larger test battery. The participants were seated in a comfortable chair and gave written informed consent that they had the right to discontinue participation at any time, if there was any part of the tests that they did not approve. Because a

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