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Alm, Per A

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Stuttering, emotions, and heart rate during anticipatory anxiety: a critical review

Per A. Alm*

Department of Clinical Neurosciences, Division of Psychiatry, Lund University, Lund, Sweden

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Abstract

Persons who stutter often report their stuttering is influenced by emotional reactions, yet the nature of such relation is still unclear. Psychophysiological studies of stuttering have failed to find any major association between stuttering and the activity of the sympathetic nervous system. A review of published studies of heart rate in relation to stressful speech situations indicate that adults who stutter tend to show a paradoxical reduction of heart rate compared with nonstuttering persons. Reduction of heart rate has also been observed in humans and mammals during anticipation of an unpleasant stimulus, and is proposed to be an indication of anticipatory anxiety resulting in a "freezing response" with parasympathetic inhibition of the heart rate. It is suggested that speech-related anticipatory anxiety in persons who stutter is likely to be a secondary, conditioned reaction based on previous experiences of stuttering.

Educational objectives: The reader will be able to: (1) describe how the autonomic nervous system is modulated by emotional responses; (2) explain how anticipatory fear often results in inhibition of heart rate due to parasympathetic activation; (3) discuss why emotional reactions in persons who stutter may be secondary to negative experiences of speech problems.

Keywords: Stuttering; Emotions; Heart rate; Parasympathetic nervous system; Freezing

The relation between stuttering and emotional factors has long been a matter of debate. Many people who stutter have the experience that stuttering is influenced by their emotional reactions, and this is also a common clinical experience. One way to investigate emotional aspects of stuttering is to measure physiological changes associated with activation of the

* Present address: Department of Clinical Neurophysiology, Lund University Hospital, SE-221 85 Lund, Sweden.

E-mail address: Per.Alm@psykiatr.lu.se (P.A. Alm).

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sympathetic branch of the autonomic nervous system. Two of the most thorough studies in this field (Peters & Hulstijn, 1984; Weber & Smith, 1990) failed to find any significant overall group differences in sympathetic activation between the stuttering and the nonstuttering group, either at rest or in relation to speech or nonspeech tasks. However, speech was associated with relatively large increase in sympathetic activity for both groups. Weber and Smith (1990) showed significant correlations between measures of sympathetic activity and speech dysfluency in persons who stuttered, but the correlations only accounted for between 3 and 17% of the variance in fluency. In brief, this type of research has not been able to show any major association between sympathetic activity and stuttering.

If there is a close connection between stuttering and emotional factors, the nature of this connection is still unclear. Menzies, Onslow and Packman (1999) reviewed the existing research on stuttering and anxiety and came to the conclusion that the lack of evidence for a relationship between stuttering and anxiety may be a result of inadequate research designs regarding definitions and measures of anxiety, insufficient statistical power, or inappropriate speech tasks. They stated "It is our contention that the inadequate literature on anxiety and stuttering is not just an academic problem .... Until the precise nature of the relationship between anxiety and stuttering is understood, fully appropriate treatment of adult stuttering cannot be offered" (p. 8).

An assumption in psychophysiological research about stuttering has been that an increase in anxiety is related to increase in heart rate. In this article it will be argued that *reduction* of heart rate can indicate an emotional response of anticipatory anxiety, and that indications of this type of response have repeatedly been shown in the previous psychophysiological studies of stuttering. First, a brief overview of the functions of the autonomic nervous system (ANS) will be presented, and related to cardiovascular effects of different affective responses. Thereafter, studies of heart rate changes in persons who stutter will be reviewed and possible interpretations will be discussed.

1. The autonomic nervous system

The ANS controls the visceral functions of the body, such as the contractions of the heart, constriction of blood vessels, blood pressure, digestion, and sweating. The ANS consists of two major subdivisions, the *sympathetic* and the *parasympathetic systems*, which cooperate to adapt the bodily functions to different situations and demands (Guyton & Hall, 1996). In many autonomic functions, but not all, the sympathetic and parasympathetic systems have opposite effects, like acceleration versus deceleration of the heart rate, or dilation versus constriction of the pupil. The parasympathetic system is mainly passing through the two *vagus nerves* from the brain stem (Guyton & Hall, 1996). The vagus nerves send autonomic fibers to the heart, lungs, and digestive organs, but also non-autonomic fibers to organs involved in speech, like the larynx, pharynx, and soft palate (Porges, 1995).

The sympathetic system is often discussed in relation to stress and anxiety. Cannon (1915) found that fear, rage and pain tend to result in massive activation of the sympathetic system, a reaction known as the *fight or flight response* (also called the *alarm* or *stress response*). This is a catabolic reaction that prepares the body for action, with increased heart rate, blood pressure, blood sugar, and redirection of blood from the viscera and skin to the muscles

(Guyton & Hall, 1996). Porges (1997) argued that Cannon made a mistake in his focus on the sympathetic system in relation to emotions, neglecting parasympathetic contributions to emotionally induced cardiovascular reactions. Porges stated that this view still influences contemporary researchers. According to Porges also the parasympathetic innervation of the heart is modulated by affective responses in the brain.

1.1. Autonomic coactivation during anticipatory fear

The "traditional" view of the ANS is that the sympathetic and the parasympathetic branches work in a reciprocal manner, so that an increase of activity in one branch is accompanied by a decrease of activity in the other branch (Berntson, Cacioppo, & Quigley, 1991). In this model autonomic activation can be described by a single variable, like "arousal". However, this is not always the case. Berntson et al. (1991) claimed that the activity of the ANS must be described by two separate dimensions, for sympathetic and parasympathetic activation. In some situations the two branches of the ANS are *coactivated*. Coactivation means that an increase of sympathetic activity is paralleled by an increase of parasympathetic activity. In this case a large increase in ANS activity will have a small effect on the heart rate, since the accelerating sympathetic effect is inhibited by the parasympathetic system.

Autonomic coactivation in humans was shown by Obrist, Wood, and Perez-Reyes (1965) in a study of autonomic responses during anticipation of a painful stimulus. Tests with and without pharmacological blockade of the parasympathetic vagal nerve (with atropine) revealed autonomic coactivation starting before onset of the anticipated painful stimuli. The vagal activation resulted in bradycardia (reduction of heart rate) despite simultaneous sympathetic activation. The observation that anticipation of a painful stimulus tends to result in bradycardia was confirmed by a study of Santibanez-H and Schroeder (1988). They found that normal men and women as well as men with "anxiety neurosis" showed bradycardia during the anticipation period before the painful stimulus. (Women with anxiety neurosis tended to show the opposite response in this study, i.e. increase of heart rate.)

This type of reaction has been studied in more detail in rats. Iwata and LeDoux (1988) conditioned rats to associate a tone with an electric footshock. When exposed to the tone the rats showed a marked *freezing response* (see Section 2) with characteristic signs of anticipatory fear (piloerection, hunching of the back, etc.). In contrast, unconditioned rats that had been exposed to a random pattern of tones and footshocks rested quietly when exposed to the tone, showing no signs of fear. However, both groups showed the same increase of heart rate at the sound of the tone. Tests with drugs blocking the sympathetic or parasympathetic fibers revealed that the conditioned group, showing anticipatory fear, actually had a large sympathetic increase that was partly masked by a simultaneous parasympathetic increase. The unconditioned group displayed only sympathetic increase, with no change in the parasympathetic system. These results were confirmed in a similar study by Nijsen et al. (1998).

A relation between bradycardia and threatening stimuli has also been observed in other mammals. Mancia, Baccelli, and Zanchetti (1972) found that cats tend to react with immobility and slight bradycardia when approached by another hostile cat. Kalin, Shelton, Davidson, and Lynn (1996) reported bradycardia in rhesus monkeys during anticipation of

an unpleasant stimulus. In summary, it can be concluded that the widespread assumption of a direct relation between anxiety and increase of heart rate is not correct. Instead anticipatory fear often results in suppression of heart rate.

2. The freezing response: biological background

From an evolutionary perspective emotions can be seen as sets of genetically determined responses which are important to solve specific problems, like avoiding danger, finding food and reproducing. Each emotional state is associated with a specific way of reacting and behaving and the nervous system adapts the organism to the demands of the current situation. Both animals and humans show two contrasting behavioral expressions of fear. One is to freeze and become mute, and the other is to become active and for example scream and run away (Marks, 1987). These different defense mechanisms are also referred to as *freezing* versus *fighting and flight* (Fanselow, 1994). In the literature different terms are used for "freezing", such as "reactive immobility" (Carrive, 1993) and "vigilance reaction" (McCabe et al., 1994). The degree of freezing in an animal can vary gradually from total immobility to different degrees of movement inhibition (Marks, 1987; Blanchard & Blanchard, 1989). When the term "freezing" is used in the following discussion it does not imply that a person or animal is totally immobile. Instead it refers to a state of anxiety with varying degree of inhibition of movement and vocalization. A possible relation between the freezing response and stuttering was suggested by Peters and Guitar (1991).

2.1. The functional role of coactivation during freezing

If an animal notices a possible predator at some distance, the freezing response is activated to avoid being detected, while the animal becomes hyperreflexive and highly reactive to surrounding stimuli (Carrive, 1993). If the predator is coming close, the prey's behavior rapidly changes to either fight or flight, with facilitation of motor behavior. An important part of the freezing state is to prepare the organism for sudden motor activity if attacked. Increased sympathetic activity redirects blood to the muscles and the lungs and increases the blood sugar level. The simultaneous parasympathetic activation inhibits acceleration of the heart. The autonomic coactivation prepares the body for rapid acceleration of the heart, since the fastest way to increase heart rate is to withdraw the parasympathetic brake. Changes in the parasympathetic nerves to the heart reach maximum effect in about 0.5 s, compared with about 4 s for the sympathetic nerves (Berntson et al., 1997). Thus, autonomic coactivation during freezing may be seen as an important part of the defensive mechanisms in mammals.

2.2. Psychological aspects of freezing

Blanchard and Blanchard (1989) suggested that the freezing response is especially associated with situations involving potential or poorly understood threats, without clear information about the best way to act. This reasoning is in line with the proposition of Härtel (1987), that a combination of fear and the feeling of being helpless causes vagal

activation and bradycardia in humans. These types of situations, with potential and poorly understood threats and a feeling of being helpless, may parallel social situations for persons who stutter. The risk of not being able to speak because of stuttering can be regarded as a potential and poorly understood risk. The experience of being helpless in the moment of stuttering is often reported from persons who stutter. It would therefore not be surprising if persons with stuttering tend to react with a tendency for freezing and vagal activation during anticipation of stressful speech situations.

3. Autonomic activity in persons who stutter

Two of the most thorough studies of autonomic reactions in persons who stutter were made by Peters and Hulstijn (1984) and Weber and Smith (1990). Peters and Hulstijn measured skin conductance, pulse volume, and heart rate in 24 persons who stuttered and in 24 persons without speech problems. The measurements were taken before, during, and after tasks like mirror writing, intelligence test, reading aloud, and spontaneous speech. Contrary to what may have been expected, the stuttering group did not show significantly higher physiological responses, either during rest or during the different tasks. The only significant interaction effect between group and task was related to heart rate ($P < 0.01$). However, the largest group difference in heart rate was in the opposite direction to what was expected: During the anticipation period before spontaneous speech, the stuttering group only showed about 0.5 beats per minute (bpm) higher heart rate than during rest, while the corresponding result for the nonstuttering group was 5.2 bpm. That is, the stuttering group showed 4.7 bpm *less* increase of heart rate than the nonstuttering group during anticipation of the speech task. For the same period the measurements of skin conductance and pulse volume showed similar results for both groups.

The lower increase of heart rate in the stuttering group can hardly be explained by lower stress in this group. The stuttering participants reported significantly higher subjective anxiety during the speech tasks compared with nonstuttering participants and skin conductance and pulse volume did not indicate lower arousal in the stuttering group. Instead this result suggests that anticipation of a spontaneous speech task was associated with autonomic coactivation in the persons who stuttered.

Weber and Smith (1990) used similar procedures to those of Peters and Hulstijn (1984), with quite comparable results. During the anticipation period before the speech task, the heart rate of the stuttering group was *reduced* by 1.2%, while the nonstuttering group showed an *increase* of 1.3% (i.e. a group difference of 2.5%). During the same period, persons who stuttered and persons without speech problems showed approximately the same increase in indications of sympathetic activation, with somewhat higher mean increase for the stuttering group. (Figures for stuttering and control groups: reduction of pulse volume [indicates sympathetic activation] 10.9% versus 7.9%, increase of phasic skin conductance 0.15 versus 0.17, and increase of tonic skin conductance 1.04 versus 0.70.) During the speech task, the stuttering group showed 5.7% increase in heart rate compared with 10.4% increase in the nonstuttering group. The increase of the other measures, indicating sympathetic activation, was about the same for the two groups. (Figures for stuttering and control groups: reduction of pulse volume 51.0% versus 46.6%, increase of phasic skin conductance 0.26 versus

0.24, and increase of tonic skin conductance 1.51 versus 1.24.) These results confirm the indications of autonomic coactivation in relation to spontaneous speech in persons who stutter. Interestingly, in both studies the signs of parasympathetic activation were much weaker during anticipation of the reading task compared with anticipation of spontaneous speech. The difference between anticipation of reading and anticipation of spontaneous speech might be related to how the tasks were presented or to differences in level of fear for different speech tasks.

The tendency for persons who stutter to show suppression of the heart rate during speech-related stress is supported by the results from a study by Caruso, Chodzko-Zajko, Bidinger, and Sommers (1994). Changes in heart rate were registered in nine persons who stuttered and nine persons who did not stutter, under three different conditions designed to induce increasing levels of cognitive stress in combination with speech. The three conditions were (a) low stress, self paced reading aloud of the words RED, YELLOW, BLUE, and GREEN at a comfortable speech rate; (b) medium stress, to read the same words as rapidly as possible; (c) high stress, to name the screen color of the words as fast as possible, when the screen color and the word name were incongruent (i.e. if the word RED was shown in blue color, the correct verbal response would be "blue").

The mean heart rate, during the tasks, of the persons who stuttered was significantly *lower* than that of the persons who did not stutter ($P < 0.05$), with increasing difference as the task became more stressful. Stuttering subjects had 7.6 bpm lower heart rate in the low stress condition, 12.9 bpm lower in condition in the medium stress condition, and 20.5 bpm lower in the high stress condition. Increasing stress was related to increasing percentage of dysfluencies (see Fig. 1). In summary, the study indicates a relation between increased stress, increased dysfluency, and increased suppression of heart rate.

Also older psychophysiological studies of stuttering have found this type of "paradoxical" cardiovascular responses. Golub (1952) studied heart rate changes during reading in 26

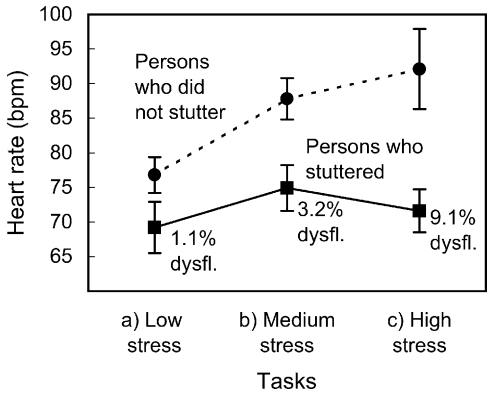

Fig. 1. Based on data from Caruso et al. (1994). Heart rate in beats per minute (bpm) for persons who stuttered (solid line) and persons without speech problems (dashed line), during reading in three conditions: (a) self-paced reading (low stress); (b) speed reading (medium stress); (c) speed reading + cognitive stress (high stress). The percentage of dysfluencies during reading is indicated for the stuttering group. The error bars represent standard errors of the mean.

persons who stuttered and 28 persons who did not stutter. It was found that the mean heart rate was significantly lower during passages with stuttering compared with passages with fluent speech. Berlinsky (1954) investigated changes in heart rate in 14 persons who stuttered and 14 persons who did not stutter, during anxiety inducing conditions with and without speech. The stuttering group showed lower heart rate in all conditions (with and without speech), but showed higher skin conductance compared with the nonstuttering group. It is interesting to note that the results of this study led the author to the conclusion that "stuttering acts as a cathartic activity relieving the anxiety of the stutterer"—a logical conclusion if regarding reduced heart rate as a sign of reduced anxiety. A similar interpretation was suggested by Dabul and Perkins (1973): They found that a difficult speech task involving stuttering reduced the mean systolic blood pressure in a group of persons who stuttered. Dabul and Perkins concluded that this result "is congruent with the psychodynamic implication that stuttering fulfills a need" (p. 590), but the authors did not exclude alternative explanations, like physiological effects.

4. Alternative explanations of reduced heart rate?

As mentioned above, Obrist et al. (1965) observed reduction of heart rate and increase of vagal activation during anticipation of conditioned unpleasant stimuli. Obrist et al. discussed how this observation could be interpreted, with the basic assumption that vagal responses are *not* related to emotions like anxiety. Suggested mechanisms like "conditioned pressor response", respiratory changes, or "orienting response" were found not to be congruent with the observations.

In persons who stutter it is conceivable that the heart rate is affected by effects of muscular tension. It is known that the so-called *Valsalva maneuver*, a forced expiration against a closed glottis, causes complex changes of heart rate and blood pressure (Ganong, Lange, & Lange, 1993). This may be relevant in stuttering, since a tense stuttering block could act as a Valsalva maneuver. The Valsalva maneuver results in increased abdominal pressure, which compresses the veins and thereby reduces the blood flow to the heart. The reduced flow leads to decreased arterial pressure and inhibition of baroreceptors, causing increased heart rate and constriction of peripheral vessels. When the Valsalva maneuver is released, the normal flow to the heart is restored but the peripheral vessels are still constricted, which leads to a rise of blood pressure above normal and a following reduction of heart rate below normal (Ganong et al., 1993). In summary, this mechanism could lead to an increase of heart rate during tense blocks or tense holding of breath, and a subsequent drop in heart rate during the periods after tense straining.

If this was the cause of the reduced heart rate seen in persons who stutter, then the reduction of heart rate should have been accompanied by signs of constricted peripheral vessels. The measurement of pulse volume in the finger is sensitive to constriction of peripheral vessels (Hugdahl, 1995) and should then be expected to covary with the reduction of heart rate. However, when looking at the data from Peters and Hulstijn (1984) and Weber and Smith (1990) this is not the case. During the anticipation period before spontaneous speech the pulse volume was almost exactly the same for persons who stuttered and persons who did not stutter. There are no indications in these studies that the reduction of heart rate in relation

to speech situations was an effect of peripheral vasoconstriction, and therefore, it seems unlikely that the reduction of heart rate was a result of frequent Valsalva maneuvers.

Another possibility could be that the findings of relatively reduced heart rate in relation to stuttering are part of a general reduction of heart rate. Available data give somewhat contradictory information about the "baseline" heart rate for persons who stutter, but in brief summary, they do not indicate generally low heart rate. For example, in the study by Peters and Hulstijn (1984), the mean heart rate during "rest" and during anticipation of the four different non-speech task was between 0.6 and 4.8 bpm *higher* for persons who stuttered compared with persons who did not stutter.

In summary, it seems to be difficult to find any non-emotional mechanism that could explain the observed reduction of heart rate. Autonomic coactivation during anticipatory anxiety appears to be a likely explanation.

5. Discussion

The reviewed psychophysiological studies of stuttering suggest that many adults who stutter tend to react with anticipatory anxiety and autonomic coactivation in stressful speech situations. This type of autonomic reaction is characteristic of a freezing response, which implies some degree of inhibition of motor activity and vocalisation. As mentioned above, a possible relation between the freezing response and stuttering was suggested by Peters and Guitar (1991). They proposed that observed co-contraction of antagonistic muscles in the larynx and in articulatory structures in persons who stutter could be part of a freezing response, with the function of silencing vocal output.

However, it is important to emphasize that emotional modulation of the severity of stuttering would not necessarily imply that emotional factors are the basic cause of stuttering. For example, it may be the case that certain types of instability in the speech system make the speech sensitive to quite normal emotional reactions. In such a case it would be reasonable to refer the cause of stuttering to the instability of the speech system and to regard the emotions as a modulating factor. Findings by Miller and Watson (1992) suggest that people who stutter do not have increased general anxiety. The increase in anxiety seems to be restricted to speech related situations and could be regarded as a rational response to negative experiences of communication. This view leads to the conclusion that the freezing reaction indicated in the reviewed studies of stuttering is likely to be a secondary, conditioned reaction, based on previous negative experiences of stuttering. It is also important to note that the reviewed indications of autonomic coactivation are based on group means for adults and may be limited to a subgroup of persons who stutter. Clinical experience suggests that anxiety is an issue for some but not all adults who stutter.

The distinction between two contrasting behavioral expressions of fear (Marks, 1987) with inhibition versus activation of motor activity (freezing versus fight/flight) might explain some paradoxical reports regarding the relation between stuttering and stress. For example, Bloodstein (1995) mentioned two cases from World War II. Two stuttering persons reported that they could only manage oral communication fluently when the situation was very dangerous. This is an extreme example, but similar experiences seem fairly common among persons who stutter. It is possible that acute danger, when the necessary course of action is

clear, may result in a fight/flight-reaction with facilitation of motor activity and vocalisation, and that this reaction ameliorates stuttering.

If emotional reactions like freezing and fight/flight affect the severity of stuttering, what would be the mediating mechanism? It seems likely that emotional states can affect the speech fluency through pathways in the central nervous system. Limbic structures like the amygdala and hypothalamus may exert strong influence on several levels of the nervous system, such as the brain stem, the basal ganglia and the cortical level. However, a detailed discussion of possible mechanisms for emotional influence on speech is beyond the scope of this article.

For future studies of emotional reactions in persons who stutter it is important to consider autonomic coactivation as a possible sign of anxiety. One way to get an indication of coactivation may be to measure the PQ interval in the electrocardiogram. Nijsen et al. (1998) found that the duration of the PQ interval (in rats) is mainly influenced by parasympathetic activity, which has a prolonging effect. During conditioned fear and freezing, rats showed a slight increase in heart rate combined with prolongation of the PQ interval. The authors claim that this combination of increased heart rate and increased PQ interval can only be explained by autonomic coactivation.

In summary, the main conclusions of this review is that (a) anticipatory anxiety can result in coactivation of the sympathetic and the parasympathetic systems, with parasympathetic suppression of the heart rate; (b) many adults who stutter tend to react with anticipatory anxiety in stressful speech situations, with a relative decrease of heart rate; and (c) there are indications that this increase of anxiety usually is limited to speech related situations, and may reflect negative experiences of previous stuttering.

CONTINUING EDUCATION

Stuttering, emotions, and heart rate during anticipatory anxiety: a critical review

QUESTIONS

1. What are the functions of the autonomic nervous system?
   a. To control the muscles of the speech organs
   b. To regulate the visceral functions of the body, such as heart rate, blood pressure, and digestion
   c. To support automatic control of speech articulation
2. How is activation of the sympathetic nervous system related to activation of the parasympathetic nervous system?
   a. The sympathetic and the parasympathetic systems are always reciprocal, so that a sympathetic increase will result in a parasympathetic decrease
   b. The two systems are always co-activated, working together
   c. The activation of the two systems may vary independently
3. Which are the basic behavioral expressions of fear, in human and other mammals?
   a. To freeze and become mute, or to become active and for example scream, run, or fight
   b. To fight or to run away
   c. To play dead and become mute

4. What has been a common conclusion from psychophysiological studies of speech-related anxiety and stuttering (the studies by Peters and Hulstijn (1984) and Weber and Smith (1990))?
   a. That stuttering is likely to be caused by emotional reactions, resulting in strong autonomic activation
   b. That persons who stutter do not show stronger autonomic reactions than non-stuttering persons
   c. That persons who stutter show a paradoxical reduction of sympathetic activation, resulting in reduced heart rate
   d. That persons who stutter tend to show a strong reduction of parasympathetic activation, resulting in increased heart rate
5. How should existing studies of heart rate in persons who stutter be interpreted according to this paper?
   a. That they tend to show a relative reduction of heart rate in relation to stressful speech situations, which is likely to be an indication of anticipatory anxiety
   b. That there is no relation between heart rate and anxiety in persons who stutter
   c. That they tend to show reduction of heart rate in relation to stressful speech situations, which indicates a paradoxical reduction of anxiety

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