Schuster SH, Schuster FM.
A muscle spindle abnormality in one laryngeal muscle would be sufficient to cause stuttering.
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History of submission and revision / Original text before revision

NOTICE: This paper does not include/substitute the above-referenced journal article. The content and the structure of the original manuscript have been changed considerably during the revision process.

Corresponding author: Steffen Schuster, Römmelesweg 20, 71394 Kernen, Germany.
E-mail: schustersh@stuttering-and-dystonia.de
Our native language is German and for reasons of economy and spontaneity we have not edited the language in all parts of this supplementary paper. Please excuse the poor English.

History of submission and revision

We submitted the first version of the paper to the Journal of Fluency Disorders on 29.08.2011 with the following cover letter.

‘Dear Editor:

My brother and I herewith submit a paper in which we hypothesize that stuttering could be the self-evident compensatory symptom of a peripheral defect in the laryngeal muscle spindles. This topic involves a basic understanding of stuttering and stuttering therapies, as well as the social image of patients who stutter.

If our paper should compete with other submissions for publication, we would like you to take into consideration that there are few journals that encourage the submission of hypotheses, and that we cannot initiate empirical research (microneurography, dissection) on our topic directly for three reasons. First, the hypothesis is too complicated to elucidate in the short introduction of an empirical study; second, the hypothesis cannot simply be derived from papers that have already been published; and third, we cannot verify our hypothesis in practical research because our academic backgrounds are not medical, but technical.

In our paper we relate stuttering causally to laryngeal dystonia. The paper contains basic interpretations which we would like to refer to in a future paper concerning the cause of cervical dystonia because the neurological journals dedicated to research on movement disorders either restrict the length of theoretical papers or exclude hypotheses from submission. So publication of this paper in the Journal of Fluency Disorders would hopefully be an important step to the integration of stuttering research into dystonia research.

We are looking forward to your decision.

Kind regards,

Steffen Schuster, corresponding author

P.S. We are patients ourselves, and our experience with attempting to play an active role in research is of interest to patient organizations. Therefore, we reserve the right to publish details of the submission, revision and decision correspondence after publication or rejection, insofar as the rights of Elsevier do not preclude this.’

On 30.08.2011 the Chief Editor of the Journal of Fluency Disorders rejected the paper. In our reply (15.09.2011) we commented on the reasons for rejection and on his advice.

Dear Ashley,

This supplementary paper has been available on our website www.stuttering-and-dystonia.de since 11.06.2012
Thank you for your email in which the above-referenced manuscript was rejected. I have read your explanation and advice and would like to point out the following:

(1) You missed a review, citation or discussion of current stuttering theories and wrote that "arguments from studies on dystonia may not be at all relevant to stuttering, which is quite a different problem". We did not submit our paper as a review and stated clearly that we developed our theory "by answering a coherent sequence of questions in which we looked for the simplest hypothetical causation mechanism that is the closest to normal function". The current stuttering theories played no role in this method, and the hypothetical relationships to dystonia theories are a result of this method. We cited papers of authors who already hypothesized that stuttering could be a type of dystonia, and stuttering shows typical characteristics of dystonia, such as co-contraction of antagonistic muscles and task-specificity. There are fluid transitions from the symptoms of spasmodic dysphonia to the "real" tonic/clonic stuttering [Angerstein, W. (2009). Überlegungen zur Klassifikation und Diagnostik Spastischer/Spasmodischer Dysphonien. Dystonie aktuell, 31, 19-20].

(2) You wrote that "further, right hemisphere overactivity is not necessarily an abnormality in people who stutter". We do not consider right hemisphere overactivity to be necessarily an abnormality in people who stutter. We interpreted all brain abnormalities psychoneurologically as non-causal and secondary compensatory attempts that follow the negative social experiences of patients who stutter. This implies the possibility that not all patients show all attempts and that anxiety may play a motivating role.

(3) You wrote: "Also, you did not cite the work of Smith, Denny et al. (1996). Activity of Intrinsic Laryngeal Muscles in Fluent and Disfluent Speech. Journal of Speech and Hearing Research, 39, 329-348". In our paper, we interpreted the co-contraction and the "conflict" of the posterior (PCA) and lateral (LCA) cricoarytenoid muscles. Smith and Denny et al. (1996) did not make EMG recordings of these muscles because they "opted to minimize the invasiveness". They further proposed that their selection of muscles [CT and TA] "may be questioned, as an important issue considered by Freeman and Ushijima (1978) was the abnormal coactivation of PCA and adductors". Please consider that the findings of Smith, Denny et al. (1996) are not relevant for our theory, thus we did not cite that paper.

(4) You wrote: "The mark of a robust theory paper is how it handles evidence that may disagree with the theory being argued. There is also the matter of cause and effect that needs to be discussed in some detail. The work of Ludlow and Loucks (2003). Stuttering: a dynamic motor control disorder. JFD, 28, 273-295 may also be of interest". Evidence against our theory would arise from studies demonstrating that abnormalities of laryngeal muscle spindles of the PCA or LCA muscles (a) do not exist in patients who stutter or (b) do exist, but are not sufficient to cause stuttering. We did not find any publications concerning this topic. The fact that there exist various stuttering theories that are contrary to our theory is not evidence against our theory.

(5) You wrote: "JFD is primarily a research journal. While it does accept review papers, your paper did not meet the criteria of a review". As mentioned above, we never intended to submit a review. The description of JFD in the "author information pack" evokes the impression that JFD encourages the submission of various article types: "As the official journal of the International Fluency Association, the journal features full-length research and clinical reports; methodological, theoretical and philosophical articles; reviews; short communications and much more". We were surprised...
not to find this variety in the dropdown menu of article types after we had started electronic submission, so we had to submit our paper as a research paper.

(6) You wrote: “Your figures were difficult to understand and far too many for the topic”. [...] Four of the five figures refer to the types of stuttering events which we characterized. We think that short sequences of numbered video frames are easy to understand and the only way to demonstrate the details of laryngeal movement in a print journal.

(7) You wrote: “If you wish to pursue your work, it may be beneficial to consider collaborating with a scientist who could assist you both in formulating testable hypotheses and developing research designs to test your hypotheses”. Our theory contains testable hypotheses, such as the existence of elasticity abnormalities in muscle spindles of the PCA or LCA muscles. However, the direct initiation of practical research would only postpone the publication. Our theory is necessary to understand the origin of the hypotheses and to discuss research results. It does not fit in a short introductory paragraph. We suspect that it would be difficult to collaborate with others before the basic theory has been published.

Because almost all journals accept only theoretical papers that are reviews of theories, it is difficult to motivate new research directions by publishing new theories that are developed by systematic re-interpretation of symptoms. Our next step will be to submit our manuscript to Medical Hypotheses.

Yours sincerely,
Steffen Schuster, corresponding author’

On 26.09.2011 we submitted the (nearly unchanged) paper to Medical Hypotheses. Suspecting that the editor could recommend that we should submit our paper to a more specialized journal, we added a copy of the e-mail above to the cover letter. On 05.01.2012 we received the editorial decision that our paper requires major revision. The Chief Editor stated that Figures 2 - 5 were difficult to understand and far too many for the topic as also suggested and commented by the Journal of Fluency Disorders. He recommended that we follow instructions to authors and restrict our manuscript to a core hypothesis. He also wrote that too many complicated arguments would have no effect on readers of our paper and would not be suitable for publication. On 28.02.2012 we submitted our revised paper with the description of our changes.

‘Dear Dr Manku:

Thank you for giving us the opportunity to revise our paper. With respect to your criticisms and advice, we have changed our paper as follows:

1. We have deleted four of five figures.

2. Instead of developing the hypothesis step-by-step by answering a complicated sequence of questions, we refer to the preceding research and present the core hypothesis early and prominently.

Stuttering events compensate for a sensory problem that arises when the abductor/adductor ratio of afferent impulse rates from the posterior cricoarytenoid and lateral cricoarytenoid muscle spindles is abnormally reduced and processed for the occasional determination of the vocal fold position.

This supplementary paper has been available on our website www.stuttering-and-dystonia.de since 11.06.2012
3. We cannot change the fact that we have to explain how a small defect could cause a complex syndrome. In order to make our paper less complicated, we transferred arguments from the development to the evaluation of the hypothesis. We are sure that reviewers of our paper would pose the critical questions that we answered in the evaluation, but we will shorten this part of our manuscript if it is necessary for acceptance.

4. We have reduced the length of the text radically from 3,600 to approximately 2,500 words (including the abstract but excluding references).

With these changes, we hope to have improved the chances of having our paper accepted.

Yours sincerely

Steffen Schuster, corresponding author'

On 22.03.2012 our paper was accepted for publication.

In the revision we changed all parts of the original manuscript. In the revised paper we seem to derive the core hypothesis rather quickly and intuitively from own experiences with laryngeal botulinum toxin injections, own laryngeal observations and new publications concerning the role of muscle spindles in the cause of dystonia. In the original paper we developed the hypothesis systematically: ‘Engineers concentrate on the symptoms of a dysfunction and search first in a systematic theoretical procedure for the smallest possible defect that could be sufficient to cause these symptoms. They do not proceed to a more complex causation hypothesis until the first one has proven to be wrong in practical research. Following this pattern, we developed an idea of what could be minimally sufficient to cause the symptoms of stuttering [...] We developed our hypothesis by answering a coherent sequence of questions that gradually approached the details of causation. In our replies, we always chose the simplest mechanisms that were the closest to normal functions. If there was no common idea about the normal function, we proposed the simplest one’. This is the boring but rational method to that we committed ourselves later in the research principles on our new website www.stuttering-and-dystonia.de. Since these principles are essential to us (and might be essential to future sponsors), we present in this paper the original version of our manuscript.

Steffen Schuster     Frank Schuster     11.06.2012
ABSTRACT

We developed a theoretical model for the cause of stuttering by answering a coherent sequence of questions in which we looked for the simplest hypothetical causation mechanism that is the closest to normal function. Following this method, we hypothesize that stuttering is a behavior that is used to compensate for a sensory problem that could arise when the ratio of afferent impulse rates from the posterior cricoarytenoid and lateral cricoarytenoid muscle spindles is distorted and processed for the occasional determination of the vocal fold position. Abductor and adductor spasmodic dysphonia (laryngeal dystonia) could also be caused by related sensory defects in the peripheral nervous system. Abnormities of brain activity and white matter may represent only secondary compensatory attempts. The evaluation of our hypothesis would need invasive and difficult practical research on laryngeal muscle spindles using techniques such as microneurography, muscle afferent block, or dissection.

1. INTRODUCTION

Stuttering is the symptom of a disorder that is still not fully understood. Modern research into the cause of stuttering is dominated by brain research and has been conducted using empirical methodology. The brains of patients who stutter have been compared with control groups of individuals with normal speech production. All functional abnormalities that correlate with stuttering severity and all structural abnormalities could be causally relevant. The disorder remains abstracted in the word stuttering, because the peripheral symptoms play no role and do not need to be explained. Some authors hypothesized, for example, that disruption in the cortical and subcortical neural systems or the imbalance of anterior forebrain and postrolandic activities play a role in the cause of stuttering [1, 2].

Our academic backgrounds are technical. Engineers concentrate on the symptoms of a dysfunction and search first in a systematic theoretical procedure for the smallest possible defect that could be sufficient to cause these symptoms. They do not proceed to a more complex causation hypothesis until the first one has proven to be wrong in practical research. Following this pattern, we developed an idea of what could be minimally sufficient to cause the symptoms of stuttering. Our theories may provide motivation toward a more practical approach to research on this topic.

2. METHOD

We developed our hypothesis by answering a coherent sequence of questions that gradually approached the details of causation. In our replies, we always chose the simplest mechanisms that were the closest to normal functions. If there was no common idea about the normal function, we proposed the simplest one. Because our first language is German, we used German standard literature for the general aspects of stuttering symptoms [3, 4].

3. THE HYPOTHESIS

3.1. Stuttering contains abnormalities of respiratory, laryngeal, pharyngeal, lingual, and orofacial speech movements. Which elements could be relevant for researching the causal problem of movement control?
Following our principle of maximal simplicity and normality, we first chose only one causally relevant element of speech movement. Symptoms are reduced when patients whisper and disappear completely when they articulate silently [3]. Stuttering shows the task specificity that is also demonstrated by patients with spasmodic dysphonia (laryngeal dystonia), such as normal fluency while singing [5]. Therefore, we concentrated in the following discussion on the idea that a problem of laryngeal control could be sufficient to cause stuttering. In our opinion, stuttering after laryngectomy [3] does not refute this old idea [6] because it is a rare phenomenon. It is also tied to use of the cricopharyngeal muscle that replaces the function of the glottal muscles following surgery [4]. The cricopharyngeal muscle could be subject to control problems that are similar to those of the glottal muscles.

3.2. If stuttering could be caused by a problem of laryngeal control, is the problem sensory or motor?

We decided to focus on only one type of laryngeal control problem. We chose the sensory problem for two reasons. First, sudden deafness and artificial elimination of auditory feedback can eliminate the symptoms of stuttering [4]. It is unlikely that a causal motor problem could be solved immediately by a sensory change. Second, the motor programs for reading a text aloud to oneself or to a listener are identical. A hypothetical error of such a motor program could not explain the fact that many patients show symptoms only in the latter case [3], although it seems possible that situational differences in the subjective demand for the sensory monitoring of speech movements also make the motor manifestation of a laryngeal sensory defect situational.

3.3. If stuttering could be caused by a sensory laryngeal problem, could it be the symptom of the problem or a symptom of its solution?

Stuttered speech is a sequence of both disfluent and fluent events. Thus, it is self-evident that every stuttering event must contain the solution that enables the transition to the following fluent part. We hypothesize that the solution portion is 100%. That is, we hypothesize that stuttering events are not disorders that cause the interruption of fluent speech, but rather that they are compensations that are repeatedly necessary to enable the fluent parts of speech. By not interpreting stuttering events as interruptions, we have no problem in explaining the stuttering events at the beginning of speech movement; and, by not interpreting them as a disorder, we have no difficulty in explaining the principal ability of stutterers to produce fluent speech parts.

3.4. If stuttering could solve a sensory problem of laryngeal muscle control, what type of internal sensory feedback “measurement” could be the problem?

Sensory feedback from the larynx could be used for “measuring” relative changes of vocal fold position, the speed of these changes, or the absolute position of the vocal folds. We decided to focus on only one type of “measurement”. We chose the determination of the absolute vocal fold position, because this measurement is most likely to be done only occasionally; therefore, it is most likely to cause occasional stuttering events as a solution to the problem.

3.5. If stuttering could solve a sensory problem concerning the occasional determination of vocal fold position, is it a peripheral or a central nervous system problem?
We first presumed a problem at the “starting point” of the sensory information; that is, the problem lies in the production of afferent signals in laryngeal proprioceptors. By presuming a peripheral defect, we have no difficulty in explaining the fact that changes of central speech control in “speech therapies” cannot cure the causal problem even when the therapies occur during childhood when the central control mechanisms are developing [7]. A peripheral defect would also explain why therapeutic speech techniques have to be used consciously in order to suppress the remaining symptoms of stuttering.

3.6. If stuttering could solve a peripheral sensory problem concerning the occasional determination of vocal fold position, what is the problem?

Laryngeal behavior during stuttering events is characterized by cocontraction of antagonistic muscle groups and a “conflict” of adductory and abductory movements of the vocal folds [8]. Consequently, we searched for the smallest sensory problem that could be solved by this abnormal laryngeal behavior. We first developed the simplest model of the normal determination of vocal fold position, because we did not find a common idea for this action.

The posterior cricoarytenoid (PCA) muscles and the lateral cricoarytenoid (LCA) muscles, respectively, abduct and adduct the vocal folds by rotating the arytenoid cartilages. The interarytenoid (INT) muscles contribute to the adduction by drawing the arytenoid cartilages together (Fig. 1). We suppose that the brain simply processes afferent feedback from muscle spindles of these muscles for occasional determination of the vocal fold position. However, there is no absolute correlation between afferent impulse rates from muscle spindles and muscle lengths. For example, afferent frequency increases with increasing muscle length, but a certain frequency does not directly indicate a particular muscle length. The brain changes the muscle spindle sensitivity according to specific situations and tasks by changing the efferent (gamma) activation of the contractile spindle endings, that is, by changing the internal prestretching of the muscle spindles [9]. Thus, we presume that the brain achieves definite sensory feedback of the vocal fold position by setting the muscle spindles of abductor and adductor muscles occasionally to the same internal prestretching (equal efferent gamma-activation) and by deriving the vocal fold position from the ratio of the afferent impulse rates.

In this model, a sensory problem occurs when equal efferent activation by the brain does not cause equal pre-stretching in abductor and adductor muscle spindles during the “measurement” of the vocal fold position. This problem may be due to partial abnormalities of muscle spindles (or of their innervations). In patients who stutter, certain abnormalities could reduce the abductor/adductor ratio of afferent impulse rates. Consequently, the distance between the vocal folds and the glottal midline is perceived as larger than it actually is. Hypothetical abnormalities may include: (1) spindle hardening or a weakness of the contractile endings in the PCA muscle spindles, or (2) spindle softening or an enhanced strength of the contractile endings in the LCA muscle spindles.

3.7. If the problem could be an abnormally reduced abductor/adductor ratio of afferent signals from muscle spindles, how could stuttering solve this problem?

Because the distorted signal ratio falsely indicates a vocal fold distance that is too large, the primary compensatory consequence of this sensory error would be a motor error: an extended adductory movement of the vocal folds. However, adductory movement is stopped when the vocal folds touch each other; that is, adduction ends before the adductor muscles are lengthened enough and before the adductor muscles are shortened enough to produce (in the distorted proprioception) the signal that the vocal folds are appropriately closed for phonation. The extended adductory activity then inhibits the ability to abduct the vocal folds for the produc-
tion of the following voiceless sound. Consequently, patients must attempt to solve the senso-
ry problem itself through compensation, by enhancing the efferent activation of the PCA
muscle spindles in order to increase their internal pre-stretching. Naturally, it is impossible to
voluntarily activate certain muscle spindles in the larynx. Therefore, we hypothesize that pa-
tients have to perform an additional motor task during the problematic “measurement” of vo-
cal fold position. This additional motor activity automatically causes an enhanced sensitivity
of the PCA muscle spindles. We suppose that a suitable motor task for that purpose is to make
the abducting PCA muscles maintain and “defend” the current vocal fold position against fur-
ther adduction. Because the activities of laryngeal and orofacial muscles are normally coordi-
nated, the special laryngeal pattern (i.e. coactivation of antagonistic muscles) also appears in
the electromyographic measurement of orofacial muscles [10].

3.8. If stuttering could indicate that a “position maintaining” task for the PCA muscles is
used as a compensation to enhance the sensitivity of their muscle spindles, is there evi-
dence for the originally well-intentioned character of an adductory inhibition? [In the
submission to the Journal of Fluency Disorders this part of our manuscript included some
references to video clips].

We did not find any published study showing “frame-by-frame” laryngeal behavior during
stuttering events. We have laryngeal behavior video recordings (via a fiberoptic
nasolaryngoscope) that were made in 2006 of one author (SS). He was 5 years old at the onset
of stuttering and 37 years old at the time of the recording. To produce the speech samples, he
talked spontaneously about travel and childhood memories.

Figure 2 shows laryngeal behavior at the beginning of the stuttered German word “g-
ganz” frame by frame at 25 frames per second. In the first transition from voiceless “g” to
voiced “a” there is continuous adductory movement, but this transition is interrupted (“gₐ”).
The second transition attempt is characterized by a short adductory stop. With this adductory
inhibition, the transition from “g” to “a” is accepted and followed by the pronunciation of the
complete word “ganz”.

Figure 3 shows the same pattern of laryngeal behavior during the pronunciation of the stu-
ttered German word “f-früheste”. In the first transition from voiceless “f” to voiced “r” we see
continuous adductory movement, but it is interrupted (“fᵢ”). The second, accepted transition
(after a quick inhalation) is characterized by an adductory stop that makes the transition 3/25s
longer; this “gap” does not disturb the perceived fluency of the production.

It is self-evident that this type of compensation implies a dilemma. The more that the ab-
ductor/adductor ratio of afferent signals from the muscle spindles is abnormally reduced, the
more the efferent activation of the abducting PCA muscle spindles has to be enhanced to
compensate and the stronger the “position maintaining task” that enhances efferent activation
for the PCA muscle spindles has to be created. Therefore, the original attempt to solve a sen-
sory problem can falsely appear to be a motor problem that blocks speech by an inappropriate
“conflict” of adductory and abductory movement of the vocal folds. Figure 4 shows such a
conflict in the beginning of the stuttered German word “k-kchhkhkhkthurz”.

Because the solution of the sensory problem is triggered by an abnormal activation of the
abducting PCA muscles, patients try to remain in or to turn back to the production of voice-
less sounds. However, vowels at the beginning of a word demand an immediate vocal fold
adduction. Thus, the false feedback of a vocal fold distance that is too large (when the vocal
folds are already closed) can first trigger the compensatory reaction that we have already
mentioned in section 2.7; that is, the speaker compensates through extended activation of the
adductory muscles. Figure 5 shows that the ventricular folds have a tendency to close in the
stuttered beginning of the word “liiiinterlaken” (Interlaken; a city in Switzerland). This pri-
mary and unsuccessful attempt to compensate competes with the attempt to enhance the efferent activation of the PCA muscle spindles as described, so that there is another conflict of adduction and abduction that “shakes” the larynx. Our hypothesis implies the possibility that the sensory problem cannot always be resolved by producing the appropriate afferent signal for the appropriate vocal fold position. It could sometimes be resolved by cancelling the “measurement” of the vocal fold position in the brain.

3.9. We have developed a hypothetical causation mechanism. However, why are the frequency and severity of the compensatory stuttering symptoms often situational and task-specific?

In the past, the situational and task-specific inconsistency of stuttering severity led to several theories of stuttering as a psychogenic disorder [3]. We explain that inconsistency by the normal variability of movement control.

First, there might exist basic motor programs for phonatory movements that work without sensory feedback when the demand for accuracy is low. Inversely, the frequency of the problematic “measurement” of vocal fold position may correlate positively with the perceived importance and difficulty of the subjective speech task. Additionally, the unconscious tolerance to accept the (false) feedback of an inappropriate vocal fold position without stuttering as compensation may correlate negatively with it.

Second, the level of efferent activation of laryngeal muscle spindles and their pre-stretching during the “measurement” could vary by situation and be due to fatigue. The distortion of the afferent signal ratio and the severity of compensatory stuttering might then vary concomitantly if the structurally changed spindles and the intact (or less changed) spindles in the antagonist muscle react differently to the same change of efferent activation.

Third, speech tasks with prolonged phonation (e.g. singing) may enable patients to restrict the occasional “measurement” of vocal fold position to the voiced sounds and to process auditory or vibratory feedback, indicating that the vocal folds are appropriately closed for phonation. Prolonged vowels and conscious speech control are also common patterns in so-called “speech therapies”. These methods often make it difficult to maintain the learned “speech techniques” against the normal desire for spontaneous speech [3].

3.10. How could central nervous system abnormalities be interpreted in a model of peripheral causation?

We hypothesize that central nervous system abnormalities are secondary compensatory trials resulting from the normal variability of movement control that we have already explained in section 3.9.

In our hypothesis, stuttering is a physiologically successful compensation. However, it appears as a speech disorder that objectively burdens harmless communication situations with problems and stigmatization. Therefore, it is likely that the intensity of speech control becomes automatically enhanced as a normal secondary compensation. Unfortunately, the well-intentioned enhancement of speech control activities can aggravate stuttering by: (a) enhancing the frequency of the problematic measurement of vocal fold position, (b) reducing the acceptance of the sensory problem without compensation and c) enhancing the efferent activation of muscle spindles if this aggravates the distortion of the problematic afferent feedback. This fatal mechanism may provoke an opposing compensation: the attempt to keep movement control intensity artificially reduced. We suppose that the enhanced or reduced activities in brain areas related to speech control [2] could simply represent the contradictory compensations. Our interpretation does not imply that stuttering severity must always correlate positively with all activities of speech control. If patients attempt to replace the problem-

This supplementary paper has been available on our website www.stuttering-and-dystonia.de since 11.06.2012
atic sensory feedback by concentrating on another feedback type, their brains might show enhanced somatosensory or sensorimotor activity that correlates negatively with stuttering severity.

The transfer of several speech control functions to the right brain hemisphere [2] could represent the attempt to interrupt unfavorable pacemaker connections. The processing of auditory feedback for final control of the composed speech movements, for example, may normally act as a pacemaker for the preceding processing of partially problematic muscle spindle feedback from the larynx. Therefore the spatial separation of these functions could be helpful. Naturally, this separation is not as effective as the complete elimination of auditory feedback that can enable patients (in our hypothesis) to ignore laryngeal feedback completely. We suppose that the white matter abnormalities indicating interrupted connections [1] could also represent an attempt to isolate and deactivate functions that are problematic due to the processing of distorted laryngeal feedback or due to their pacemaker function for such events.

3.11. What does our hypothesis imply for related disorders?

Stuttering shows typical characteristics of dystonia, such as co-contraction of antagonistic muscles and task-specificity. Therefore it has been hypothesized to be a type of dystonia [11] and related to spasmodic dysphonia [5, 12]. There are fluid transitions from the symptoms of spasmodic dysphonia to the “real” tonic/clonic stuttering [13] [This reference was not part of the manuscript we submitted to the Journal of Fluency Disorders]. Following again our principle of maximal simplicity, we suppose that abductor and adductor spasmodic dysphonia could also be compensations for peripheral sensory defects in the larynx. Although stuttering (in our theory) is caused by an abnormally reduced abductor/adductor ratio of afferent signals that falsely indicates a vocal fold distance that is too large, abductor spasmodic dysphonia might be caused by an abnormally enhanced abductor/adductor ratio of afferent signals that falsely indicates a vocal fold distance that is too small during occasional “measurement”. Patients compensate for this sensory error by an extended activity of the abducting PCA muscles, moving the vocal folds to the inappropriately lateral position that falsely produces the afferent signals of an appropriate position. Therefore, stuttering and abductor spasmodic dysphonia (in our hypothesis) compensate for opposite sensory problems, but both compensations contain abnormal activations of the PCA muscles. In this model, adductor spasmodic dysphonia could compensate for an abnormally reduced perception of vocal fold tension, mainly by enhancing the activity of the thyroarytenoid muscles [14].

3.12. Is there evidence against our theory?[This part of our manuscript was not included in the paper that we submitted to the Journal of Fluency Disorders]

Evidence against our theory would arise from studies demonstrating that abnormalities of laryngeal muscle spindles of the PCA or LCA muscles (a) do not exist in patients who stutter or (b) do exist, but are not sufficient to cause stuttering. We did not find any publications concerning this topic. The fact that there exist various stuttering theories that are contrary to our theory is not evidence against our theory.

4. CONCLUSION

Hypothetical muscle spindle abnormalities in the posterior cricoarytenoid (PCA) or in the lateral cricoarytenoid (LCA) muscles would be sufficient to cause stuttering. Some authors have already hypothesized that afferent feedback from muscle spindles plays a role in the cause of dystonia [15] and that it is distorted [16, 17]. By relating stuttering clearly to dystonia, by in-

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interpreting brain abnormalities clearly as secondary and non-causal, and by describing hypothetical details of causation, we emphasized the importance of practical research on such abnormalities. Simultaneous video recordings of laryngeal behavior and microneurographic recordings of afferent/efferent signals to and from the PCA and LCA muscle spindles during stuttered speech could be compared to recordings from a non-stuttering control group. However, there might be technical difficulties in discriminating the signals of interest from other signals or ethical considerations due to the invasiveness of the recording technique. Alternatively or additionally, patients who stutter could allow the post-mortem dissection of muscle spindles, enabling microscopic examination for structural abnormalities and mechanical examination for abnormalities of elasticity.

We suppose that, in contrast to patients with adductor and abductor spasmodic dysphonia, patients who stutter are not impaired by enhanced muscle activities that may result from the sensory problem; rather, they are impaired by the solution of the sensory problem itself. Therefore, our hypothesis implies that botulinum toxin injections could not be very helpful in stuttering therapy. The less effective and less accepted injections to the vocal folds [5] may have reduced stuttering by causing a new feeling of laryngeal abnormality that motivated patients to concentrate on intact, conscious feedback types for laryngeal control. From the existence of muscle spindle abnormalities would arise new ideas for therapy research. Perhaps the blockage of distorted afferent feedback from PCA and LCA muscles could cause the brain to either: (a) “switch off” the demand for the measurement of vocal fold position (comparable to the mechanism we hypothesized for the elimination of auditory feedback), or (b) process another type of feedback. The latter effect would be problematic if the patients would be forced to unnaturally control speech at a conscious level. In writer’s cramp, a subtype of dystonia, muscle afferent block by lidocaine/ethanol injections has shown some effect [18].

Our interpretation of stuttering might provide moral support to patients who do not want to replace their stuttered spontaneous speech with fluent but artificial speech techniques under the pressure of stigmatization. They could say, “Stuttering is not necessarily a mysterious central nervous system disorder. Stuttering could be the self-evident compensation for a peripheral defect, comparable to limping with a shortened leg.”

REFERENCES

FIGURES

For the reason of copyright we do not show figure 1 in this supplementary paper.

Fig. 1. Effects of muscle contractions on the arytenoid cartilage and vocal fold positions. Abbreviations: INT, interarytenoid muscle; LCA, lateral cricoarytenoid muscle; PCA, posterior cricoarytenoid muscle.
Fig. 2. Laryngeal behavior in the stuttered German word “g-ganz”, frame by frame (25 frames per second). The upper frame sequence shows the first, interrupted transition from “g” to “a”; the lower sequence shows the second, accepted transition.

Fig. 3. Laryngeal behavior in the stuttered German word “f-früheste”, frame by frame (25 frames per second). The upper frame sequence shows the first, interrupted transition from “f” to “r”; the lower sequence shows the second, accepted transition.
Fig. 4. Laryngeal behavior in the stuttered beginning of the German word “k-kchhkchhkchhkurz” (frame by frame; 25 frames per second). This sequence shows a “conflict” of adduction and abduction.

Fig. 5. Laryngeal behavior in the stuttered beginning of “liiiinterlaken” (frame by frame; 25 frames per second). The ventricular folds nearly close.