NOTICE: In this draft we present a causation theory for cervical dystonia and the outline for the first step of practical research in our project. We will attempt to discuss the practicability of the proposed proceeding with several experts. Our native language is German and for reasons of economy and spontaneity we have not edited the language in this paper. Please excuse the poor English. Do not use this theoretical paper for decisions concerning diagnosis or therapy.

(I) The hypothetically strongest role of neck muscle spindles in the cause of idiopathic cervical dystonia. (II) We propose microneurography of afferent signals from sternocleidomastoid muscle spindles in laterocaput.

Abstract

In part (I) of this paper we explain why distorted sensory feedback from neck muscles would be theoretically sufficient to cause all diagnostically relevant symptoms of idiopathic cervical dystonia.

- The dystonic head postures in cervical dystonia could compensate for an incorrect measurement of head positions caused by distorted muscle spindle feedback. We suppose that the brain could measure absolute positions by setting the contractile endings of intrafusal fibres of antagonistic muscle pairs to the same efferent (fusimotor) activation and by interpreting the resulting ratios of afferent impulse rates. The afferent ratios could be distorted if muscle spindles in a muscle pair do not get equal properties from equal efferent activation. These differences might be due to abnormalities of muscle spindles (or of their innervation) concerning one of the muscles.

- The abnormal cocontraction of antagonistic muscles could be caused by the incompatibility of correct and distorted sensory feedback.

- The situational or task-specific variability of symptom severity in dystonia might be caused by normal sensory variability in the following mechanisms. (1) The normal variability of efferent muscle spindle activation makes the distortion of afferent ratios variable, because “problematic” and normal spindles react differently to the same change of efferent activation. (2) Different feedback types are weighted variably according to situational and task-specific demands (accuracy, importance), so that the dominance of the distorted part of feedback varies in muscle control. (3) Different feedback types might be variably differentiated in muscle control, so that the distorted part of feedback is variably melted with/“diluted” by/conflicting with intact sensory feedback.

- The sensory trick is an attempt to reduce symptoms by setting an extern absolute position to the position of the head can be related correctly, because relative changes of positions can be measured without calculating afferent ratios.

- Brain abnormalities could be interpreted as diffuse and “desperate” secondary compensations that are motivated by the grave impairment of dystonia patients and the impossibility to compensate for muscle spindle abnormalities directly. There are two competing directions: (1) Intensification of movement control represented in the enhancement of brain activities related to muscle control. This well-intentioned intensification can aggravate symptoms, because a) efferent activation of muscle spindles might be enhanced and aggravate distortion of afferent ratios, b) the problematic muscle spindle feedback might be weighted higher, and c) the problematic muscle spindle feedback might be differentiated more sharply from intact feedback types. (2) Making muscle control unsharp, rep-
resented in reduced brain activities, white matter abnormalities and reduced distances of cortical representations. This might anticipate/compensate for the problematic intensification of movement control.

- Enhanced globus pallidus activity could be the most central expression and pacemaker of the problematic intensification of movement control. This might explain the effect of deep brain stimulation “switching off” the GP and fostering by this the unsharpness of proprioceptive muscle control that is hypothetically beneficial for the reduction of dystonic symptoms.
- Distortion of sensory feedback might be the common causal mechanism in etiologically very different types of dystonia. The sensory problem might originate alternately in abnormalities of proprioceptors or of the nerves that transmit afferent/efferent signals, or in abnormal brain functions triggering inappropriate activation of type specific receptors (muscle spindles).

In part (II) we outline basic ideas about the empirical verification of the theory.

- Useful steps would be microneurography of afferent signals from neck muscle spindles, microneurography of efferent signals, and dissection of neck muscle spindles and their innervation after the death of patients. We provide some details for the microneurography of afferent signals from sternocleidomastoid muscle spindles in the CD subtype laterocaput. The recordings would have to be made bilaterally during a task demanding active muscle control. We expect this proceeding to pose several grave problems that have to be discussed.

Introduction to both parts

Patients who suffer from focal idiopathic dystonia use sensory tricks to reduce the severity of symptoms [1,2], and chemical blockage of afferent signals from muscle spindles of dystonic muscles reduced dystonic muscle activity in several types of primary dystonia, including cervical dystonia [3,4,5]. There is a sufficient number of publications in which dystonia is interpreted as a partially sensory disorder [6,7] and in which muscle spindles are hypothesized as playing a role in causal mechanisms [8]. However, nobody has yet attempted to record and analyze the signals from muscle spindles in patients. We suppose that this is because of two reasons.

First, in the current theories dystonia remains a disorder of primarily central nervous character, so that the causal role of muscle spindles remains too weak and too abstract to justify the use of the invasive microneurography technology. The authors of a review combined, for example, the hypothesis of increased IA afferent input (due to dystonic co-contracture) with the hypothesis of a predisposition to a disorder of group-IA afferent processing and a compensatory abnormal processing of group-II afferent information. They supposed this hypothetically faulty sensorimotor processing system to bias the development of inappropriate motor programmes in the basal ganglia [9].

Second, it has been practicable but not easy to discriminate afferent signals from muscle spindles properly during microneurography [10] in normals. Possibly dystonic symptoms would aggravate this problem in patients.

In response to these two problems, we divide this paper into two parts. In part (I), we present a theory in that the causal role of muscle spindles is concrete and strong enough to motivate the use of microneurography in the research of cervical dystonia. The theory is a derivate of our muscle spindle theory concerning the cause of stuttering [11]. In part (II), we present the outline of a future microneurography study of afferent signals for a subtype of cervical dystonia.

However, microneurography might be impossible for physical reasons (e. g. simply because the electrode does not rest in place when the head is moved). This problem could force us to initiate practical research first with another type of primary idiopathic dystonia, to promote technological progress in microneurography, or to motivate the direct research into structural properties of muscle spindles and their innervation in dystonia patients.
(I) The hypothetically strongest role of neck muscle spindles in the cause of idiopathic cervical dystonia

Introduction

It is self-evident that there can be no stronger hypothetical role for muscle spindles in dystonia than the idea of structural muscle spindle abnormalities causing dystonic symptoms in primary normal central nervous mechanisms.

Some years ago, a group of authors has touched on such an idea. According to their hypothesis, the observed abnormal reactions to vibration stimuli may indicate abnormalities of muscle spindle elasticity in patients with dystonia [12]. One author of that group later proposed that these elasticity abnormalities could cause afferent signals to change with muscle fatigue and pose a problem for motor learning: “(...) motor subroutines learned in the fatigued state are used on distorted muscle spindle afferent information and are therefore inappropriate for the unfatigued state, resulting in the affected muscles moving with inappropriate force when initiating the motor subroutine in the unfatigued state” [13]. We think that this hypothesis is not suitable for most types of dystonia. The hypothesis mainly refers to musician’s dystonia while playing instruments, but other dystonic movements are normally not subject to excessive learning and practising causing muscle fatigue, such as head postures in cervical dystonia or glottal movements in laryngeal dystonia. Moreover, we think that changes of mechanical spindle properties due to fatigue are not sufficient to explain the task-specificity or situational variability of symptom severity. Last but not least, the undeniable brain abnormalities [e. g. 14] and the effect of deep brain stimulation [15] have to be explained in a theory of peripheral causation. (On 28.06.2013 Richard Grünewald wrote us that in his hypothesis the fatigue of neck muscle spindles is caused, for example, by twisted positions in sleep. We added his article – that contains also interpretations of central nervous aspects - in the following reference: [14a]).

In 2012 we presented a theory in that abnormalities of certain laryngeal muscle spindles or of their innervations would be sufficient to cause stuttering. In our theory, stuttering could compensate for an abnormally reduced abductor/adductor ratio of afferent impulse rates when this ratio is used to measure the absolute position of the vocal folds. We explained the situational and task-specific variability of symptom severity psychoneurologically by the basically normal variability of feedback processing and of efferent muscle spindle activation. In this model, the brain abnormalities result as (partially contradictory) compensations from the effects of impairment experience on this normal variability. We related stuttering to the abductor type of laryngeal dystonia which we hypothesized to compensate for the inverse defect (for an abnormally enhanced abductor/adductor ratio of afferent impulse rates). Finally, we generalized the hypothetically causal mechanism for other types of idiopathic dystonia as follows: Dystonic symptoms compensate for the distorted ratio of afferent impulse rates from antagonistic muscle spindles during the occasional determination of positions [11].

We think that neither stuttering nor laryngeal dystonia are suitable for a first microneurographic examination of muscle spindle signals in patients. Laryngeal movements in spontaneous speech are quick and therefore could pose a problem for microneurographic measurement and analysis. The attempt to slow down speech movement could artificially change sensory settings in patients. Furthermore, with the current technology, it is necessary to press and passively move the parent muscles in order to identify afferent signals [16], which is hardly possible in the larynx. Therefore we suggest concentrating first on cervical dystonia, and especially on its subtypes laterocaput or laterocollis [17]. We suppose that from the normal symmetry of head posture and from the normal identity of contralaterally paired neck muscle spindles could be derived simple and objective criteria for what could be abnormal in neck muscle spindle signals of patients showing lateral head or neck tilt.
The development of our muscle spindle theory for the cause of stuttering was motivated by the question what defect could be minimally sufficient to produce the diagnostically relevant symptoms. Therefore, in the following, we adapt our muscle spindle theory for cervical dystonia by demonstrating that muscle spindle abnormalities are sufficient to cause the typical dystonic symptoms: 1) The dystonic head posture, 2) the cocontraction of antagonistic muscles, 3) the task-specific and situational variability of symptom severity, and 4) the sensory tricks. Furthermore, we propose a basic interpretation of functional and structural brain abnormalities.

### The dystonic head posture

Microneurographic findings from muscle spindle afferents of muscles that move the foot tip around the ankle have demonstrated that the brain could theoretically “measure” positions by calculating population vectors representing the mean contribution of each muscle population of afferents to the coding of a particular position, and by finally calculating a sum vector [16]. However, we suppose that a hypothetical error in a sum vector which is really used for muscle control would cause inappropriate positions to that all muscles contribute “in harmony” by inappropriate activities. Therefore this model would not be suitable for the explanation of the inappropriate head postures in cervical dystonia that are characterized by dystonic activities of only some muscles and a conflict of muscle activities. We propose the following model.

In order to “measure” the absolute head-to-neck posture exactly the brain could consider several pairs of neck muscles that act as agonist and antagonist in lateral movements, forward/backward movements or in the rotation of the head. The brain might occasionally set the sensory parts of the intrafusal fibres to the same internal prestretching (by equal gamma-activation of the contractile endings) and calculate for each pair the ratio of afferent impulse rates. Without formally calculating a “sum vector”, the brain could derive from the ratios an image of the lateral, forward/backward and rotatory components of the head position. (In an email to one of the authors of a proprioception review [17a] we explained the model detailed as “afferent ratio landscape” [appendix]).

In this model, a normal person would demonstrate during the “measurement” of head-to-neck positions the following microneurographic findings from contralaterally paired neck muscles of identical type (e. g. from the sternocleidomastoid muscles).

\[ f_r = \text{afferent impulse rate (frequency) from the right muscle; } f_l = \text{afferent impulse rate from the left muscle} \]

<table>
<thead>
<tr>
<th>Symmetrical head-to-neck position (without rotation and lateral head or neck tilt)</th>
<th>( f_r = f_l )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lateral head-to-neck tilt to the right side (without rotation and lateral neck tilt)</td>
<td>( f_r &lt; f_l )</td>
</tr>
<tr>
<td>Lateral head-to-neck tilt to the left side (without rotation and lateral neck tilt)</td>
<td>( f_r &gt; f_l )</td>
</tr>
<tr>
<td>Furthermore, the inverse afferent ratios ( f_{l,xr}/f_{l,rx} = f_{l,rx}/f_{r,xl} ) would be equal when the head is tilted to the same degree (x) to the left side (xl) and to the right side (xr)</td>
<td></td>
</tr>
</tbody>
</table>

A patient suffering from laterocaput and demonstrating dystonic activity in the right sternocleidomastoid muscle might have unilateral or bilaterally different muscle spindle abnormalities in
this muscle pair. These asymmetric abnormalities distort the ratio of afferent impulse rates. During the measurement of head-to-neck position, the muscle spindles from the sternocleidomastoid muscle pair produce in the actually symmetrical head position the afferent impulse ratio of lateral head tilt to the left side \( (f_l > f_r) \). When this ratio is processed in the motor control of the sternocleidomastoid muscle pair and the head position is intended to be symmetrical, the right sternocleidomastoid muscle contracts in order to produce the afferent ratio of the symmetrical position \( (f_r = f_l) \). Unfortunately, this well-intentioned compensatory contraction moves the head to the “dystonically” tilted head posture (to the right side).

We do not specify the type of afferent (primary or secondary) that is used for the “measurement” of head positions. Microneurography studies with imposed passive movements revealed differences in the reactions of primary and secondary afferents that could principally be relevant for the ability to encode head positions (e.g., silence during muscle shortening, deceleration response, position sensitivity) [18]. However, we hypothesize that muscle spindles could be gamma-activated specifically for position measurement in the preparation or execution of active movements, which could change spindle properties.

The cocontraction of antagonistic muscles

In our theory for the cause of stuttering we interpreted the cocontraction of antagonistic laryngeal muscles to be a compensatory solution of the hypothetical sensory problem. We proposed that performing a “position maintaining task” for the abducting (PCA) muscle could enhance the efferent activation of its muscle spindles [11]). However, in this theory for the cause of cervical dystonia, we interpret the cocontraction of antagonistic muscles simply as motor conflict resulting of the incompatibility of distorted and normal sensory feedback.

While in our laterocaput example above the ratio of afferent impulse rates from the sternocleidomastoid muscles is distorted, the afferent ratio from other bilaterally paired muscles might be correct (or less distorted). Furthermore, the brain might process correct sensory feedback from mechanoreceptors in the skin surrounding the atlanto-occipital joint, and the visual system (needing to be readjusted) could provide sensory feedback of the inappropriate dystonic head tilt. While the distorted muscle spindle feedback in our example initiates a lateral movement to the right side in order to achieve symmetry in the sternocleidomastoid afferent ratio, these correct feedback types initiate a movement back to the left in order to achieve symmetry, resulting in a conflict of antagonistic muscle activities.

The situational variability of symptom severity

We suggest three hypothetical reasons for the situational variability of symptom severity in cervical dystonia.

First, the abnormal distortion of the afferent signal ratio could be variable because the efferent (gamma-) activation of muscle spindles varies. There are studies demonstrating a normal task-

---

1 This sensory interpretation of antagonistic muscle cocontraction implies an explanation for the restricted efficacy of botulinum toxin therapy in some types of dystonia, for example in the therapy of abductor spasmodic dysphonia [19] (a subtype of laryngeal dystonia). If botulinum toxin injections have to reduce muscle activities that are triggered by the distorted part of proprioception and enable by this the accomplishment of muscle contractions that are triggered by normal sensory feedback, the lack of normal sensory feedback is a problem. Although the normal auditory system provides feedback of the vocal fold position when the vocal folds are appropriately closed for phonation, there is no definite auditory feedback when the vocal folds are open, because a variety of open positions is likely to produce voiceless sounds. More generally, the lack of intact sensory feedback could cause “non-spasmodic” symptoms in that the inappropriate postures are not accompanied by excessive muscle activity, because agonists and antagonists do not “fight” against each other, but contribute in harmony to the production of the inappropriate postures.
specific and situational variability of efferent activation [20,21]. In our laterocaput example, the sensory parts of intrafusal fibres in the right sternocleidomastoid muscle spindles might be abnormally softened, causing the sensory parts of intrafusal fibres in the right and in the left muscle spindles to be prestretched differently by the same efferent activation of the contractile endings, and producing by this mechanism the distorted ratio of afferent impulse rates during the “measurement” of the absolute head position. The difference of prestretching and the concomitant distortion of the afferent signal ratio might be larger when the efferent activation is high. This interpretation implies that a sudden enhancement of efferent activation could cause a sudden dystonic muscle activity.

Second, the information from different feedback systems could be weighted variably in muscle control. We suppose that mechanoreceptors in the skin surrounding the atlanto-occipital joint provide a direct, but rather rough feedback of the head-to-neck-position. On the other hand, the ratio of afferent muscle spindle signals might provide a more calculated, but rather exact feedback of the head-to-neck-position, because the afferent ratio could reflect the real ratio of muscle lengths. Therefore we hypothesize that, in the control of neck muscles, the brain weights the (unfortunately distorted) muscle spindle feedback higher in situations in which a correct head posture is individually estimated to be more important or more difficult to achieve.

Third, the resolution of position measurement could be kept low in motor tasks that are estimated easy or less important. That is, afferent ratios (and other feedback types) indicating similar muscle effects are not sharply differentiated, so that a distorted afferent ratio might be “diluted” and dystonic symptoms might be reduced. On the other hand, the sharp differentiation of feedback types might increase the conflict of antagonistic muscle contractions that results of the incompatibility of distorted and intact feedback.

The sensory trick

The sensory trick [1] in our theory is a voluntary manoeuvre that is intended for keeping the brain from spontaneously calculating the absolute head-to-neck position by (partially distorted) ratios of afferent signals from antagonistic neck muscle spindles and from using these ratios in muscle control. In our laterocaput example from above, the patient might put some fingers of his left hand to the left side of his face and set by this an external absolute position to that the movements of the head can be related correctly (because even the hypothetically softened muscle spindles in the right sternocleidomastoid muscle, when considered without calculating ratios, encode movement directions and changes of muscle lengths correctly). Since in our theory the problematic measurement of the “absolute” head-to-neck-position actually refers to symmetry (as far as the lateral and rotatory components of head movement are concerned), we are not surprised about the fact that a compensatory sensory trick produces asymmetrical changes of brain activities [22]. We propose that the replacement of distorted muscle spindle feedback by intact sensory feedback might occur also task-specifically and contribute to the variability of symptom severity2.

Functional and structural brain abnormalities

In the preceding paragraphs we demonstrated that peripheral sensory abnormalities would be sufficient to cause the diagnostically relevant symptoms of cervical dystonia in a functionally and structurally normal brain. As a consequence, we can interpret the functional and structural brain

2 When, for example, patients with musician’s dystonia learn a new instrument, the new motor programs might first be developed and controlled slowly by the processing of intact auditory and visual feedback at a conscious level, so that dystonic symptoms are reduced. As a result of practice, proprioceptive feedback might get more and more important in quick, exact and unconscious muscle control. So the enhancement of efferent activation might increase the distortion of the problematic afferent ratio again, and the enhanced differentiation of (distorted and intact) feedback types might foster the conflict of antagonistic muscle activities again.

stuttering-and-dystonia.de, 27.06.2013

- 6 -
abnormalities psychoneurologically as secondary and compensatory reactions (similarly to the interpretation that we presented in our theory for the cause of stuttering [11]). There are basically four types of abnormalities that have been revealed for cervical dystonia or other types of primary dystonia: enhanced activities in areas related to movement control, reduced activities in such areas, white matter abnormalities and reduced distances between cortical representations.

**Enhanced activities.** Although dystonic postures and movements in our hypothesis are “well-intentioned” motor compensations of the sensory error, they undeniably impair patients functionally and can cause physical pain. Furthermore, patients suffer from social isolation by the dysaesthetic and by the interpretation as symptoms of a central nervous disorder. The awareness of such problems surely burdens the dystonic movement with an additional estimation of difficulty and importance. As a normal compensatory reaction in our hypothesis, the movement control is automatically intensified. Expressions of this intensification of movement control might be, for example, the enhanced sensimotor and premotor activities in patients with writer’s cramp [23] and the enhanced somatosensory activity in orofacial dystonia [24]. The well-intentioned intensification can worsen the symptoms by the mechanisms that we described in the paragraph concerning the situational variability of symptom severity: a) absolute positions are “measured” more frequently, b) the (unfortunately distorted) muscle spindle feedback is weighted higher than the feedback from mechanoreceptors in the skin, c) the “alerted” brain might enhance the efferent activation and make by this the problematic ratio of afferent impulse rates more distorted, and d) afferent ratios are differentiated more precisely, so that the distorted ratio is not “diluted” by correct ratios or other correct feedback types, but conflicts with them. These mechanisms can result in a screwing aggravation of dystonia. The intensification of movement control causes a symptom worsening that causes a further intensification of movement control, again worsening the symptoms. Things are different during the use of a sensory trick. Increased brain activities related to muscle control may then indicate the artificial concentration on an intact type of sensory feedback and correlate negatively to symptom severity of dystonia.

**Reduced activities.** The bad consequences of the normal, but fatal compensatory intensification of muscle control may provoke an opposing compensatory reaction. Patients try to keep the intensity of movement control artificially decreased in order to reverse the described mechanisms. A direct evidence for the beneficial compensatory role of decreased cortical activities could be the following finding. Writer’s cramp patients did not produce dystonic symptoms in a complex movement task that was likely to cause symptoms when cortical (somatosensory, sensorimotor, premotor) activities were reduced [14]. The fact that cortical activities in a simple motor task were not reduced could indicate that this was not necessary, because in a simple motor task the favourable sensory settings appear spontaneously.

The result of these contradictory and competing compensatory reactions can be a co-existence of enhanced and reduced brain activities [24]3.

**White matter abnormalities** [25]. Such abnormalities exist also in patients who stutter [26a]. They are supposed to indicate reduced connectivity and interrupted flow of information. In our hypothesis for the cause of dystonia we propose two interpretations. First, the compensatory attempt to interrupt pacemaker connections for the unfavourable mechanisms that we described in the paragraph “enhanced activities” could cause white matter abnormalities. Second, the connections between areas that are activated contradictorily (enhanced and reduced) could be automatically interrupted.

**Reduced distances of cortical representations** of fingers have been revealed in the brains of patients with writer’s cramp [26a]. We interpret these abnormalities, similar to reduced cortical activi-

---

3 By proposing an important influence of secondary changes of brain activities and of efferent muscle spindle activation on the symptom severity, our theory principally allows the integration of genetic predispositions that concern abnormal brain activities.
ties, as part of the attempt to compensate for the bad consequences of control intensification by making muscle control artificially unsharp.

The interpretation of brain abnormities as secondary reactions is supported by the observation that certain abnormities were reduced or disappeared after botulinum toxin therapy, for example enhanced somatosensory activity in Meige patients [24] and white matter abnormities in patients who suffer from cervical dystonia / hand dystonia [27]. Although the authors who revealed these effects proposed neurological mechanisms that are triggered by the feedback of reduced muscle activity, we propose an indirect psychoneurological explanation (In our opinion, the physiological properties of muscle spindles [28] do not suggest that they provide “electromyography-like” feedback of muscle activity). By reducing or abolishing the dystonic symptoms, botulinum toxin therapy reduces or abolishes functional and social problems that triggered the compensatory brain abnormities by the mechanisms that we described above. Cancelling the “alert” in the brain may result in a reduced efferent activation of muscle spindles in the dystonic region and reduce by this the distortion of afferent signals even in dystonic muscles that have not been treated with botulinum toxin. Differences in the brain activity normalization following BTX therapy might also be interpreted psychoneurologically. Patients may consider the relief from impairment to be too temporary or to have come too late, so that not all compensatory changes of brain activity disappear; the reduced sensorimotor activity in patients with adductor spasmodic dysphonia [29], for example, might be kept up after botulinum toxin injections to support their beneficial effect.

The most discouraging effect on the research into peripheral nervous causes of primary dystonia might come from theories that introduced an important causal role of abnormal activity in the basal ganglia and that seem to be confirmed by the effectiveness of deep brain stimulation (DBS) of the globus pallidus also in the therapy of cervical dystonia [15]. However, DBS is interpreted to mimic the ablation of the globus pallidus rather than to cause the normal function. In our hypothesis, the abnormal globus pallidus activity could be the most central expression of the attempt to overcome the functional and social problems that result of dystonic symptoms by an intensification of muscle control. So the globus pallidus might be the main pacemaker for the unfavourable compensatory mechanisms that we described above in the paragraph “enhanced activities”. Since DBS is interpreted to deactivate the globus pallidus, this might support the competing compensatory attempt to keep the efferent muscle spindle activation (enhancing the distortion of the problematic afferent ratios), the differentiation of sensory feedback (enhancing the conflict of intact and distorted feedback), and the weight of muscle spindle feedback (competing with other feedback types) artificially reduced. We think that these hypothetical mechanisms of reduced sensory sharpness are also suitable for explaining the side effects of DBS, e. g. the worsening of speech and writing exactness.

Conclusion

We demonstrated that muscle spindle abnormalities would be theoretically sufficient to cause dystonic symptoms as compensations in primarily normal central nervous functions. However, we emphasize that this model could be only one variant of a bigger theory suggesting that the compensation of sensory errors in muscle control might be the common causal mechanism in very different types of primary idiopathic dystonia. Differences could occur in the types of sensory information that are distorted and in the causes of the distortion. In the explanation of adductor spasmodic dysphonia, for example, we suggest that the tension of the vocal folds might be the distorted sensory information (rather than the vocal fold position). Peripheral causes of sensory distortion might not be restricted to the proprioceptors, they may occur alternately in the nerves transmitting afferent and efferent activation of muscle spindles in the dystonic region and reduce by this the efferent efferent muscle spindles in the dystonic region and reduce by this the distortion of afferent signals even in dystonic muscles that have not been treated with botulinum toxin. Differences in the brain activity normalization following BTX therapy might also be interpreted psychoneurologically. Patients may consider the relief from impairment to be too temporary or to have come too late, so that not all compensatory changes of brain activity disappear; the reduced sensorimotor activity in patients with adductor spasmodic dysphonia [29], for example, might be kept up after botulinum toxin injections to support their beneficial effect.

The most discouraging effect on the research into peripheral nervous causes of primary dystonia might come from theories that introduced an important causal role of abnormal activity in the basal ganglia and that seem to be confirmed by the effectiveness of deep brain stimulation (DBS) of the globus pallidus also in the therapy of cervical dystonia [15]. However, DBS is interpreted to mimic the ablation of the globus pallidus rather than to cause the normal function. In our hypothesis, the abnormal globus pallidus activity could be the most central expression of the attempt to overcome the functional and social problems that result of dystonic symptoms by an intensification of muscle control. So the globus pallidus might be the main pacemaker for the unfavourable compensatory mechanisms that we described above in the paragraph “enhanced activities”. Since DBS is interpreted to deactivate the globus pallidus, this might support the competing compensatory attempt to keep the efferent muscle spindle activation (enhancing the distortion of the problematic afferent ratios), the differentiation of sensory feedback (enhancing the conflict of intact and distorted feedback), and the weight of muscle spindle feedback (competing with other feedback types) artificially reduced. We think that these hypothetical mechanisms of reduced sensory sharpness are also suitable for explaining the side effects of DBS, e. g. the worsening of speech and writing exactness.

Conclusion

We demonstrated that muscle spindle abnormalities would be theoretically sufficient to cause dystonic symptoms as compensations in primarily normal central nervous functions. However, we emphasize that this model could be only one variant of a bigger theory suggesting that the compensation of sensory errors in muscle control might be the common causal mechanism in very different types of primary idiopathic dystonia. Differences could occur in the types of sensory information that are distorted and in the causes of the distortion. In the explanation of adductor spasmodic dysphonia, for example, we suggest that the tension of the vocal folds might be the distorted sensory information (rather than the vocal fold position). Peripheral causes of sensory distortion might not be restricted to the proprioceptors, they may occur alternately in the nerves transmitting afferent and

---

4 The interpretation of basal ganglia activity as a secondary pacemaker implies that direct therapeutic normalization of the globus pallidus activity would not result in the elimination of dystonic symptoms, but in a medium symptom severity (between the symptom severity without treatment and the reduced symptom severity resulting from DBS).
efferent signals between the proprioceptors and the brain (structural abnormalities of peripheral nerves have been observed in some patients suffering from laryngeal dystonia [30]). Furthermore, secondary brain reactions influence symptom severity in our theory, so that even structural brain abnormalities could be integrated in this model, if they have effects on these secondary reactions. Finally, sensory distortion could be caused exclusively in the brain, if abnormal brain functions lead to abnormal fusimotor activation of muscle spindles. The latter mechanism might distort the afferent ratio landscapes [appendix] of our model widely and thereby be especially suitable for the explanation of generalized types of dystonia. Most of the mechanisms that we suggested to explain the situational variability of symptom severity work independently of the cause of sensory distortion (variable weight of different feedback types, variable differentiation). Brain researchers might also like the idea that in our model it is not necessary to suppose “magic” central-nervous effects of peripheral botulinum toxin injections for the explanation of post-therapeutic changes of brain activities, because the beneficial peripheral effects of BTX could simply reduce the motivation for diffuse central-nervous compensations.

So we believe that the theory of sensory distortion as a peripherally or centrally caused problem has the potential to unify types of dystonia that are very different in their etiology and in the numbers of muscles and body regions that are affected. On the other hand, we fear that theories hypothesizing abstract central nervous mechanisms as primary causes in all types of dystonia could fail in explaining very restricted symptoms. We suppose, for example, that the theory of disrupted brain circuits would make it necessary to hypothesize a sequence of protecting or compensatory mechanisms in order to explain that symptoms occur only in some muscles, so that common focal types of dystonia would finally require more complicated explanations than rare generalized types of dystonia. This problem could lead to a decline of focal dystonia research, and it might motivate researchers to extend compensatorily the primary symptoms of focal dystonia, risking thereby to damage the social perception of dystonia as a pure movement disorder (some authors, for example, emphasized the importance to reveal “psychopathological symptoms” in patients with cervical dystonia as primary symptoms resulting directly from abnormal basal ganglia activity [31] We think that the methodology of this study was not suitable for refuting the presumption that the - minor - psychic abnormities are only secondary reactions to social problems resulting from dystonic symptoms).

Genetic findings also might serve as arguments against the one-sided research focusing exclusively on central nervous aspects in patients suffering from problems in muscle control. In patients who stutter there have been found gene mutations that are associated also with abnormalities of bone, connective tissue, liver, and spleen [32]. Tissue abnormalities are principally interesting in our theory, because they could change the mechanical properties of muscle spindles directly or by impairing repair mechanisms. Impaired repair mechanisms could also play a role in the cause of peripheral nerve abnormalities (mentioned above) and provide explanations for cases of focal dystonia that occurred after surgery [32a], peripheral injury [32b], or infection [32c]. We believe that hypothesizing mysterious long-distance effects that might cause damage to brain functions is not the obvious thing to do first in these cases.

On the first glance, the fact that chemical muscle afferent blockage (MAB) reduced dystonic symptoms, but did not completely abolish them [3,4,5], could be interpreted as evidence against the primary role of muscle spindles in the cause of dystonia. However, we point out the following. 1. MAB might not have “switched off” the production of afferent signals completely nor corrected the frequency of the afferent signal sufficiently, so that the afferent signals were still strong enough to be “attractive” for the automatic use in muscle control and still distorted enough to cause dystonic symptoms; 2. The brain might have replaced the original muscle pair (of which the distorted afferent signal ratio is processed for muscle control) by another muscle pair of which the afferent signals were similarly distorted; 3. The conflict of antagonistic muscles results in our model from the incompatibility of intact and distorted sensory feedback. This conflict may impair patients sufficiently to cause a compensatory learning process in that the interpretation of the intact sensory feedback is
brought closer to the interpretation of the distorted feedback, so that intact feedback also causes (mild) dystonic postures; 4. Motor programs might not be developed completely from the base of sensory measurements. There might be basic motor programs that become refined and corrected by the use of sensory feedback. When distorted feedback (falsely) causes “corrections” of the basic motor program frequently, to a large extent and always to the same direction, the basic motor program itself might be finally changed. (With respect to lateral head-to-neck positions, the basic motor program could originally establish that symmetrical activities of bilateral neck muscle pairs are likely to produce a symmetrical position when the neck is vertical).

The clarification of the questions whether, and on which level of the sensory system dystonia could be caused by distorted sensory feedback is naturally very important for the development of prevention strategies and new therapies for dystonia patients. Although Parkinson’s disease is clearly a neurodegenerative brain disease, Parkinson research could also get new impulses from this type of dystonia research, since functional abnormalities of proprioception [32d] and structural abnormalities of muscle spindles [32e] have been found in patients with Parkinson’s disease. More generally, investigators of different types of central nervous movement disorders might be interested in the question whether central nervous dysfunctions cause inappropriate motor “decisions” for muscles a) directly in the brain, or b) indirectly by distorting fusimotor activity of muscle spindles and subsequent sensory informations that are finally the base of motor decisions.

In the second part of this paper we will develop first ideas for the empirical verification of our theory.
(II) We propose microneurography of afferent signals from sternocleidomastoid muscle spindles in laterocaput.

Introduction

In our opinion, studies that have already been published do neither support nor refute the idea that there could exist abnormalities of muscle spindles in patients who suffer from primary cervical dystonia. We present the following examples.

Vibration induced illusion of movement (VIIM). Dystonia patients copied the sensation of movement they perceived in the vibrated arm with the other (tracking) arm subnormally. Both arms were non-dystonic body parts [12]. The authors of that paper offered several interpretations, including the hypothesis that the findings might indicate general abnormalities of muscle spindle elasticity in dystonia patients. However, we think that there could occur diffuse changes of efferent (gamma-) activation of muscle spindles as secondary reactions in the alerted brains of dystonia patients, and that changed efferent activation could cause subnormal perception also with structurally normal muscle spindles. Furthermore, the patients knew that vibration produces only the illusion of movement. Like other illusions, this one might also depend on personal considerations at an unconscious level. The reduced perception of illusionary movement could simply indicate that dystonia patients have learned to diffusely distrust their muscle control systems. Since the relatives of dystonia patients might develop comparable feelings of alert and distrust when invited to a VIIM study, the VIIM abnormalities in this group [33] could be due to the same causal mechanisms.

An old case study. In 1976, muscle spindles of a patient with idiopathic torsion dystonia were examined post-mortem [34]. With respect to optical criteria, the morphology and innervation of the muscle spindles showed no abnormalities. We suggest that this study is not very meaningful for the following reasons: (1) it refers to only one patient and one type of dystonia; (2) it is not clear whether or not both spindles of dystonic muscles and their antagonists were examined; (3) a normal look does not necessarily indicate normal mechanical properties and normal functions of muscle spindles.

The ability to adjust a laser pointer. In 2003, a group of authors presented an article entitled “Idiopathic spasmodic torticollis is not associated with abnormal kinesthetic perception from neck proprioceptive and vestibular afferences” [35]. They deduced this from the observation that patients in a dark room were able to adjust the light of a laser pointer (with the help of a joystick) in their head and trunk mid-sagittal directions after head and trunk had been passively rotated in varied combinations (only head, only trunk, and head and trunk) around the vertical axis. For three reasons we believe that these facts do not refute our theory: (1) In part (I) of this paper we hypothesized that there exist several intact feedback types and that the problem with the distorted ratio of afferent impulse rates has motor consequences when the ratio is considered with preference in the active control of the muscle that provides the abnormal afferent signal. Since in the experiment the head is only passively rotated, the defect part of proprioception might be covered by intact sensory feedback; (2) in passive movements the gamma-activation of muscle spindles could be low and produce a small distortion of the afferent signal; (3) a neurologist from the University of Tuebingen (Germany) wrote us that, in the light of clinical experience, approximately one of three cervical dystonia patients does not perceive the dystonic head posture spontaneously and that this lack of perception can occur when the dystonic head tilt is up to 20 degree. Larger dystonic head tilt is usually perceived because the visual system has to be readjusted.

We fear that artificial study designs with passive movements might cause sensory settings that differ markedly from settings in normal situations demanding sensory “measurement” in active muscle control. In the following proposals we attempt to take this into account.
The logical steps for researching into the hypothetical role of muscle spindles in cervical dystonia

With regard to the theoretical approach in part (I) of this paper, we propose four principal steps.

First step. In order to verify whether or not abnormalities in the ratios of afferent impulse rates do exist in primary cervical dystonia and whether or not they are theoretically sufficient to cause the dystonic head posture (as compensation), there should be made microneurographic recordings of Ia / II signals from a dystonic neck muscle and its most direct antagonist. The recordings should be made during a task that requires the “measurement” of the absolute head position in active muscle control.

Second step (in case the first step reveals relevant abnormalities of afferent signals). In order to verify whether the abnormalities of afferent signals are caused in the CNS by abnormal fusimotor (gamma-) activation or peripherally by abnormalities of muscle spindles, there should be made microneurographic recordings of efferent fusimotor signals that are transmitted to the spindles of the most dystonic neck muscle and its most direct antagonist. The recordings should be made under the conditions of the first step (same patients, same movement task). Probably, the gamma-signals would have to be identified by excluding the possibility that the signals could be of other types [36].

Third step (in case the first and the second step reveal relevant abnormalities of afferent signals that are likely to be caused in the muscle spindles themselves). In order to research into structural muscle spindle abnormalities, patients could allow post mortem dissection of dystonic neck muscles and antagonistic muscles. Abnormalities might include, for example, 1) softening of the sensory part of intrafusal fibres / enhanced strength of intrafusal fibres’ contractile endings in dystonic muscles, or 2) hardening of the sensory part of intrafusal fibres / weakness of the contractile endings in antagonistic muscles. There might also exist innervation abnormalities. Demyelination of efferent innervation, for example, could possibly reduce or delay the contraction of intrafusal fibre endings and distort the efferent feedback.

It is self-evident that every step has to be accompanied by examinations of a healthy control group. In the first and the second step, the microneurography of the healthy control group should be made before and the results could be published separately as physiological studies that reveal the normal mechanisms of head position “measurement”.

We believe that the third step would make sense standing alone if the microneurographic steps should prove to be impracticable for technological, anatomic or ethical reasons. However, in the following we attempt to provide some details for the first step in the research of laterocaput. This is the base on that we will start to discuss the practical proceeding with experts.

Microneurographic recordings of afferent signals from muscle spindles of the sternocleido-mastoid muscle pair in laterocaput patients and a healthy control group

Proceeding for the healthy control group that could provide material for a separately published physiological study.

The target muscle. We choose the sternocleidomastoid muscle pair for synchronized bilateral microneurography of afferent signals because this is the most concerned dystonic muscle in laterocaput [17]. The muscle is divided in a part that extends from the caput sternale to the processus mastoideus and a part that extends from the caput clavicular to the processus mastoideus. The muscle is probably sensibly innervated by rami musculares branching off the plexus cervicalis. The first problem that could foil the whole study is the following. We fear that the electrode for microneurography has to be inserted in a part of the neck that is moved when the head posture is changed during the experiment. This could cause the loss of the signal, since the electrodes should not be fixed (in order to avoid injuries of the nerve).
Choice and preparation of subjects. The healthy subjects must not suffer from neural diseases causing the symptoms of a movement disorder and have to give their informed consent as required by the declaration of Helsinki. In order to relieve the recognition of head postures in frontal video recordings, the midline of the neck, the middles of the chin and of the forehead should be marked.

Identification of afferent signals. We derive the following outline of proceeding from the descriptions in two physiological microneurography studies [10, 16]. The subjects are seated comfortably in an armchair. In order to minimize neck muscle activities, the subjects lean their head as relaxed as possible to a cushion on a fixed inclined board to the side opposite to the first needle insertion. The sternocleidomastoid muscle is controlled by recording surface EMG activity. The electrode for microneurography is inserted percutaneously into the sensory nerve of the sternocleidomastoid muscle. The signals are monitored on an oscilloscope and a loudspeaker. “Single units are isolated by adjusting the position of the microelectrode in minute steps”[16]. In order to identify the signal to be afferent and to originate in the sternocleidomastoid muscle, the tendon of the muscle is manually pressed. In order to find out whether the afferent unit is type II or Ia and to exclude afferents from Golgi tendon organs, it is necessary to adapt, for example, some of the methods that have been described for the discrimination of afferents from the finger extensor muscles [10]. Maybe three criteria of this study would be sufficient: 1. the “enhanced response to a slow ramp stretch was considered firm evidence for muscle spindle origin, because similar responses have not been shown to occur with Golgi tendon afferents.” 2. Initial bursts are more common in primary afferents. 3. Most primary afferents (80%) ceased firing during imposed muscle shortening, whereas only 0 to 5% of the secondary afferents did so.

If an afferent unit has been identified, the complete proceeding is carried out on the other side of the neck. The aim is to get a sufficient number of bilateral pairs of afferent units from the sternocleidomastoid muscles that are of equal type (Ia/Ia and II/II), and of sufficient number of mixed type pairs (Ia/II). However, it might be a problem to get a sufficient number of II/II pairs, because there was a clear majority of afferents that have been identified as primary [10]. The following experiment is carried out with every pair of afferents.

Experimental setup. The subjects have been informed that they have to reproduce several head-to-neck positions most exactly, blindfolded and with the trunk bended to one side (in order to avoid that visual or vestibular feedback could provide feedback of the head-to-neck-position). The subjects practise this first in the following proceeding. They regard in succession the pictures of three lateral head-to-neck positions (for example 7, 14 and 21 degrees) and reproduce these positions first with open eyes (with a mirror) and then with closed eyes in both directions (to the left and to the right), with the trunk bended to the left or to the right. If the subjects have learned this, they are blindfolded and the bilateral microneurographic recordings are made during the following proceeding. After some arbitrary movements of the head, the subjects move their head slowly in succession to the middle position, then to the 7, 14 and 21 degree positions on the right side, then back to the middle position and to the 7, 14 and 21 degree positions on the left side and back to the middle position. The subjects press the button of a buzzer every time they have achieved one of the demanded positions most exactly. We think that this could motivate the subjects to set the fusimotor activation clearly for the encoding of positions and make the analysis of the movements from frontal video recordings easier.

Data analysis. The following proceeding is carried out for every pair of afferents. First, the video recording is converted to a diagram showing the instantaneous head-to-neck tilt (horizontal coordinate: time [s], vertical coordinate: head tilt [degree]). In order to make the comparison of left and right positions easier, both directions are drawn as positive and the directions are indicated below the graph. The graph starts 1s before the subjects have pressed the button of the buzzer for signaling that they have achieved the first of the demanded 0 degree positions. Every buzzer signal is indicated in the diagram. Then, the microneurographic recordings are converted to a diagram showing the instantaneous afferent frequency of the right and of the left unit (horizontal coordinate: time
[s], vertical coordinates: frequencies $f_{al}$ and $f_{ar}$ [imp/s]). The buzzer signals are also indicated in this diagram by drawing vertical lines from the buzzer signals in the preceding diagram.

After that, the diagram of every afferent pair is analyzed according to the following questions. 1. Did the muscle spindles produce signals continuously, or at least at the demanded positions that have been indicated by the buzzer signal? 2. Is $f_{al} / f_{ar}=1$ at the 0 degree position? 3. What were the $f_{al} / f_{ar}$ ratios at the 7, 14 and 21 degree positions on the right side and did they increase from one position to the next? 4. What were the $f_{ar} / f_{al}$ ratios at the corresponding positions on the left side and were they equal to the ratios on the right side?

Then the results of all afferent pairs are compared in a combined analysis in order to test whether certain afferent ratios encoded certain head-to-neck positions symmetrically and equally

- in all afferent pairs of a certain combination (Ia/Ia, II/II, Ia/II) in a subject;
- in all afferent pairs of all combinations in a subject;
- in all afferent pairs of a certain combination (Ia/Ia, II/II, Ia/II) in all subjects;
- in all afferent pairs of all combinations in all subjects.

The results of this analysis could be converted to several diagrams (horizontal coordinates: head tilt [degree, positive to the right direction, negative to the left]; vertical coordinates: $f_{al} / f_{ar}$ for head tilt to the right, $f_{ar} / f_{al}$ for head tilt to the left).

**Differences in the proceeding for laterocaput patients**

**Choice of patients.** The patients must have a diagnosis of laterocaput with the sternocleidomastoid muscle being affected. The microneurographic recordings should be made when the effects of the last botulinum toxin injection have been diminished to the greatest possible extent, because botulinum toxin is likely to influence also the intrafusal fibres. The patients should be able to actively change the lateral head position for at least 5 degrees (in order to produce afferent signals for two clearly different positions.

**Identification of afferent signals.** In a previous study it was possible to passively rotate the heads of torticollis patients without causing discomfort or pain [35]. Therefore we hope that it is possible to carry out with laterocaput patients the passive lateral movements of the head that are principally necessary to discriminate primary and secondary afferent signals. However, we do not know whether or not the reactions of the muscle spindles of cervical dystonia patients meet the normal criteria for the correct identification of the afferent signals. In other words: The hypothetical muscle spindle abnormalities (or the abnormal fusimotor activation) that we attempt to research could hypothetically foil the identification of afferent signals that is necessary for research.

**Experimental setup.** Under the same conditions as the healthy control group, the laterocaput patients practice and perform positioning tasks only on the dystonic side. The head-to-neck positions are chosen individually according to the capabilities of the patients. However, it would be best if the healthy controls and the patients could produce some directly comparable positions.

**Data analysis.** It is self-evident that the analysis proceeding must depend on the results of the physiological study with the healthy controls. Principal questions would be the following. 1. Are there abnormalities in the heights of afferent impulse rates? 2. Are there abnormalities in the afferent ratios? The special questions resulting from our hypothesis in the first part of this paper would be: 1. Do the muscle spindles in the sternocleidomastoid muscles of laterocaput patients produce in tilted head-to-neck positions on the dystonic side the sensory feedback of symmetrical position or even a lateral head tilt to the opposite side? 2. Do the muscle spindles produce the sensory feedback of increasing head tilt to the opposite side when the head is actually moved from the dystonically tilted head-to-neck position towards the symmetrical position?
Questions concerning the practicability of the above described proceeding

1. The sternocleidomastoid muscle is probably sensibly innervated by rami musculares branching off the plexus cervicalis. Are there sites that are suitable for the insertion of the electrode for microneurography of muscle spindle afferents?

2. Are there insertion sites that are also accessible in the lateral head-to-neck tilt of laterocaput patients?

3. Is it possible to move a head passively (e.g. slow ramp stretch, passive shortening) without a machine in order to discriminate afferent types?

4. Would passive or active head-to-neck movements move also the electrode in a way that could cause the loss of signal?

5. Are there a sufficient number of laterocaput patients with dystonic sternocleidomastoid muscles who could participate in a study that is for technological reasons likely to be bound to certain universities?

6. Are there a sufficient number of laterocaput patients who are able to produce voluntarily at least two clearly different head-to-neck positions?
Appendix: “Afferent ratio landscapes” in the measurement of positions (return to the main text)

Uwe Proske is one of the authors of a recent review about the proprioceptive senses [17a]. Being interested especially in the question whether or not there is empirical evidence against our physiological model of position measurement, we described it in an email to him (19.02.2014) as follows.

We propose that joint positions could be “measured” by calculating the ratios of afferent impulse rates from pairs of antagonistic muscles that are equally gamma-activated. The number of muscle pairs depends on the complexity (number of components) of joint positions, so that complex joint positions could be represented by “landscapes” formed by afferent ratios. A muscle can contribute signals to more than one afferent ratio.

In comparison to a sum vector model the afferent ratio landscape has some functional advantages that might make it likelier to be the physiological function in the measurement of positions. We will explain these advantages in the following paragraphs.

There are several variables in the production of afferent signals which could make position measurement inexact or ambiguous. Afferent ratios are fractions; therefore variables that appear in the numerator and in the denominator as equal factors can be eliminated.

First of all, the variable efferent (gamma-) activation can be eliminated in our model, since we suppose that this central nervous activation is kept equal in both muscles of antagonistic pairs during the measurement of positions (or at least the efferent activations are kept in a constant ratio) [Intrrafusal fibres can be interpreted as consisting of two springs in series: One spring representing the sensory part and the adjacent spring representing the contractile endings (MileusnicMP, Brown IE, Lan N, Loeb GE. Mathematical models of proprioceptors. I. Control and transduction in the muscle spindle. J Neurophysiol 2006;96(4):1772-88). Gamma activation changes the mechanical properties of the second spring and defines by this how the spindle length is distributed among the springs. In our model, these distributions are equal in both muscles of antagonistic pairs during position measurements, or at least the ratio of these distributions is kept constant].

There are also peripheral variables (in the spindles themselves) that might appear as nearly equal factors in spindles of antagonistic muscle pairs and therefore can be eliminated: 1. fatigue of contractile endings that could alter their reaction to gamma activation, and 2. stiffness changes of the intrafusal fibres’ sensory parts due to multiple stretches within short time.

Furthermore, the equal contraction of contractile endings in spindles of both antagonistic muscles could set the spindles to the same stretch “history” and eliminate by this structural differences (such as slackening) that have been caused by different previous movements.

In the following, we will explain the advantages of grouping afferent ratios to “landscapes” representing complex joint positions.

Afferent ratios indicating similar components of joint positions could be grouped next to each other, so that either the sharp feedback from single afferent ratios or the blurred feedback from a group of similar afferent ratios could be processed by muscle control. This would allow changing the “resolution” of position feedback according to varying difficulty and importance of motor tasks. Furthermore, the loss of an afferent ratio (after a lesion) could be compensated by a “neighboured” ratio [In a sum vector model of position measurement it is necessary to calculate always with all
vectors, and the loss of a vector cannot be compensated. In addition to this, afferent ratio landscapes would make the integration of feedback from mechanoreceptors easier because the spatial distribution of pressure is also more likely to be represented in a landscape.

Afferent ratio landscapes are bundles of coordinates of joint positions. Complex positions of limb ends are composed of several joint positions (fingertip-to-nosetip positions, for example, contain 7 or 8 joint positions). The base of motor learning in our model is collecting afferent ratio landscapes of as much positions as possible. Remembered positions are waypoints in the planning of voluntary movements. Planning a movement (only with muscle spindle feedback) means laying the afferent ratio landscapes of the target position (or those of the first waypoint) over those of the current position and calculating the differences. Executing a movement means transforming the afferent ratios of the current position into those of the target position (or into those of the next waypoint).

We suppose that the algorithms of this model would be less complicated than a sum vector model, because the sensory informations in our afferent landscape model directly indicate how muscle lengths have to be changed.

Uwe Proske wrote in his reply (20.02.2014) that there might be 1) evidence suggesting that for a pair of antagonists it is not the ratio but the difference in afferent firing between them that is used by the brain [37]; 2) evidence suggesting that when a muscle is contracting, including the contraction of spindle intramuscular fibres, as a result of co-activation, those spindles may no longer contribute to proprioception. Thus, vibrating a contracting muscle does not produce the expected illusions of movement and position [38]; 3) evidence suggesting that the regulated property is limb endpoint and a joint-based reference frame is not used [39]. We do not think that these arguments make it necessary to change our model basically, but we have to clarify some details. 1) An afferent ratio in our landscape model might not be represented by one “mountain”, but by two mountains produced by the afferent impulse rates from both muscles of an antagonistic muscle pair. Equal (change of) efferent gamma-activation of the spindles in both muscles multiplies the heights of the mountains by the same factor. A certain difference of afferent impulse rates (unlike a ratio) could only be interpreted as a certain joint position if efferent activity is constant. Therefore we suppose that the brain uses the ratio of afferent impulse rates. The experiment of Gilhodes et al. (illusion of movement in passive limbs following vibration stimuli) [37] was not suitable to reflect special demands that could result of variable efferent activity during active muscle control. 2) Our model does not presuppose that afferent ratios can be calculated all the time. The measurement of absolute positions by the calculation of afferent ratios might be restricted to moments before movement, after movement and to turning points. If reliable feedback from contracting muscles is not available during movement, the afferent impulse rates from the stretched muscles might provide sufficient feedback to calculate the change of position in relation to the latest absolute position. 3) We suppose that there are no special receptors for the perception of limb endpoint positions, so that a limb endpoint frame is a central nervous interpretation of joint position feedback that bundles and couples information from proprioceptors optimized for the quick calculation of the limb endpoint position in automatized motor tasks (such as walking) that normally do not demand differentiated motor decisions for the participating joints. These details are less important in this paper, since we hypothesize mainly the basic contribution of muscle spindle afferents to the joint position feedback and the pathophysiological potential of feedback distortion in the cause of cervical dystonia. We do not know whether or not there exists a limb endpoint frame for the head position in the human brain and suppose that motor decisions that base on such sensory frames would be similarly concerned by distorted muscle spindle feedback when compared to joint based frames.
Strange Numbers (such as [32a]) are due to updates of our theory that forced us to insert new references.


