Am XX.XX.XXXX via email to:

[title, name]
[department]
[university]

StuDyst

Research proposal: Search for neurovascular conflicts of the recurrent laryngeal nerve /vagus nerve in patients with laryngeal dystonia and stuttering

Dear [title, name],

With my brother as co-author I published the hypothesis that a characteristic falsification of laryngeal proprioception would suffice to cause stuttering (Schuster and Schuster, 2012). We stutter ourselves.

Our hypothesis implies a causal relationship of stuttering to forms of dystonia in which distortions of proprioception have already been suspected. In a discussion paper, we have specified the mechanisms of our causation hypothesis for a lateral subtype of cervical dystonia, because in this type relatively simple assumptions and predictions for abnormalities of afferent muscle spindle signals can be made.

However, according to several experts, microneurography technology is not (yet) suitable for empirically testing this hypothesis by recording afferent muscle spindle signals. Therefore, we will focus directly on nerve abnormities that could distort proprioception by altering the efferent and afferent signal transmission between proprioceptors (muscle spindles) and the brain.

As with hemifacial spasm, neurovascular conflicts have already been found and surgically treated in patients with cervical dystonia (PubMed list). In patients with laryngeal dystonia, a peripheral nervous cause was also considered four decades ago after the discovery of demyelinated nerve fibers in the recurrent laryngeal nerve (Bocchino and Tucker, 1978). However, there is still no publication on the search for neurovascular conflicts.

We therefore suggest that patients with laryngeal dystonia as well as stuttering patients should be screened on both sides for neurovascular conflicts of the the recurrent laryngeal nerve (e.g. in the loop around the aortic arch or the subclavian artery). Attention should also be paid to other abnormal contacts in the course of the nerve (e.g. penetration of the ligamentum suspensorius). The course of the recurrent laryngeal nerve is quite complicated and can vary individually in details. It might be even better to start search for neurovascular conflicts at the vagus nerve which passes the carotid sheath between the common carotid artery and the internal jugular vein. As a low-threshold introduction to the topic, we suggest testing the imaging procedure (MRI with 3D visualization) first on one of us (Steffen Schuster). Relevant results could be published in a case study. Would you be interested in carrying out this first step?

Please note that on the second page we respond to some foreseeable objections and are happy to answer further questions. We would be very pleased if you were interested in this topic and look forward to your reply.

Yours sincerely,

Steffen Schuster Frank Schuster

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Please write to Steffen Schuster, schustersh@stuttering-and-dystonia.de

Four predictable objections:

1. Objection: Hemifacial spasm has unilateral symptoms. Bilateral symptoms - such as in laryngeal dystonia and stuttering - are generally regarded as an argument for causation in the brain.

Answer: Normal phonation settings of the larynx are always symmetrical. It can therefore be assumed that a rationalized brain does not properly separate the left and right laryngeal muscles in sensory and motor terms. According to this assumption, a one-sided sensory disorder after processing in the brain also causes motor symptoms on both sides. In any case, it has already been discovered that unilateral laryngeal botulinum toxin injections in patients with laryngeal dystonia also affect the muscle activity of the other side (Ludlow, 1990).

2. Objection: Neurovascular conflicts have a disturbing effect especially in the exit zone of the cranial nerves, because the myelin sheaths are not completely developed and therefore susceptible to damage. This does not apply to the laryngeal recurrent nerve. In addition, the rather "slow" symptoms of laryngeal dystonia (in contrast to the "shooting" contractions in hemifacial spasm) do not correspond to the common notion that electrical impulses between nerve fibres jump over due to damaged insulation.

Answer: Our sensory causation hypothesis does not require dramatic effects of neurovascular contacts. Sufficient are conduction disorders that lead to faulty fusimotor activation of proprioceptors (muscle spindles) or change the sensory information (impulse frequency) of the signals sent by proprioceptors to the brain. In addition, studies on the effect of neurovascular decompression in cervical dystonia do not show that it only works in patients with clonic symptoms.

3. Objection: Abnormal brain activity was found in dystonia patients, so it is not worth looking for causes outside the brain.

Answer: A study also found abnormal brain activity in hemifacial spasm (Tu et al., 2015) and drug therapies have some effect. Nevertheless, the very good effect of microvascular decompression in most cases undoubtedly proves the causation through a neurovascular conflict.

4. objection (by Roger J Ingham, University of California): *"I enjoyed your revival of the idea that stuttering might be a consequence of a problem located within the larynx. That idea was explored in a number of studies during the 1970s. However, it essentially came to an end with the discovery of alaryngeal stutterers - persons who stutter who then have a laryngectomy but their stuttering persists. The first documented case was reported by Tuck in 1979 (Tuck, A.E. (1979). An alaryngeal stutterer: A case history. Journal of Fluency Disor-ders, 4, 239-243.). Other reports followed. I'm not sure, therefore, how your theory would accommodate such findings."*

Answer: Stuttering can hypothetically be reduced to a problem of laryngeal control, because stutterers articulate voicelessly fluidly. The laryngectomized patient of Tuck (1979) does not stutter without a voice either, but when using the esophageal voice. The subsequent study by Wingate (1981) also seems to refer only to the use of the esophageal voice. This replacement voice is produced by the cricopharyngeal muscle, which - like the inner laryngeal muscles - is also controlled by the recurrent laryngeal nerve. This could lead to control problems similar to those of the "real" laryngeal voice, so that stuttering after laryngectomy is not an argument against our research. To clarify this aspect we will propose the study "Do stuttering patients speak fluently when using an electronic voice prosthesis?"