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Research Article

# Nice Side Effect of the Bruxism Treatment by Botulinum Toxin: Stopping Tinnitus

**Dominique Batifol[1*], Marie de Boutray[1], Sophie Galmiche[1], Patrick Jammet[1], and Pierre-Jacques Finiels[2]**

[1]*Department of Maxillofacial Surgery, University Hospital Gui de Chauliac, France*
[2]*Department of Traumatology and Spine Neurosurgery, Alès General Hospital, France*

***Corresponding author**
Dominique Batifol, Department of Maxillofacial Surgery, University Hospital Gui de Chauliac, 80 avenue Augustin Fliche, 34295 Montpellier cedex 5, Tel: 33-686862618; Fax: 33-466360107; Email: dominique.batifol@dbmail.com

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**Abstract**

Injections in the manducatory muscles to treat bruxism relax the temporal and masseter muscles with non-negligible quantities of Botulinum Toxin. That is to say, there is always some diffusion of the product.

Incidentally, we noticed that, some of our patients, suffering from tinnitus, experienced relief.

Botulinum toxin has already been used to treat tinnitus with palatal injections. But here, we are speaking of a sort of "welcome side effect". In this paper, we try to explain this effect, even if it was not the aim of our initial study and if the number of patients concerned is small.

The studies, which have been going on for 15 years now, show the effect of botulinum toxin on a large number of neurotransmitters, explaining its action on pain and inflammation, and perhaps not only on them….. !

## INTRODUCTION

Tinnitus corresponds to a hearing sensation not bound to a sound generated by a vibration outside the body and not audible by the surrounding people. European, Japanese or American prevalence would be around 10% of the adult population, increasing with age. We do not know exactly what causes tinnitus: an advanced hypothesis is that they could result from an attempt of the brain to compensate hearing loss by increased activity, which, in return, generates phantom or neuropathic pain. During the process of a study carried out for another pathology (bruxism), we noticed a potential activity of botulinum toxin in such a case of condition, which seems to be interesting to be reported on.

Bruxism is a para-functional activity with repetitive, involuntary contractions of the jaw-lifting muscles. Bruxism can be centric by clenching the teeth or eccentric by grinding the teeth. Manducatory muscles are controlled by reflex nerve pathways, under brain control. During sleep, this abnormal chewing action can be intense, because the reflex part is active, without brain control [1]. Botulinum Toxin type A (BoNTA) is now a well-known therapy for involuntary contractions, and considered one of the most efficient treatments of bruxism. A lot of people suffer from this disease, and stress increases bruxism: it is thus estimated that 80% to 95% of the occidental population (depending on the study), have had bruxism once in their lives.

A lot of patients have severe bruxism and need to be treated by BoNTA. Some of them have other symptoms, such as migraine headache, cervical pain, orthoptic disorders, tinnitus …

With the treatment of bruxism, some of these symptoms can be resolved, even if the 2 pathologies are of different origins and are not related.

## MATERIAL AND METHOD

This is a retrospective study on 288 patients treated for bruxism in the department of maxillofacial surgery, Montpellier University Hospital (France), with BoNTA. All the patients were injected in the masseter and temporal muscles. The dosage depended on the intensity of the contractions. Our aim was to relax muscles to protect the teeth and the temporomandibular joint (TMJ), to decrease the pain in the muscles themselves and all the pain induced in the surrounding areas (migraine, cervicalgia,...). The injections of BoNTA ( BOTOX* : Allergan Pharmaceuticals, Westport, Ireland ) were carried out in the masseter and temporal muscles, the dosage in the masseter ranging from 30 U to 100 U and in the temporal from 10 U to 50 U (Fig 1 and 2). Patients had to be careful to avoid touching the injection zones. The anatomical region of the ears contains very

small elements and a very small quantity of BoNTA is enough to have an effect.

Files were analyzed for two years: 2015 and 2016, with the emphasis on secondary pathologies.

## RESULTS

Among these patients, 17 had tinnitus. Most of them only came to treat bruxism, a small number of them had heard from their ENT specialist that BoNTA could sometimes decrease tinnitus.

After the beginning of the treatment, 14 patients had a decrease in (3 cases) or a cessation (11 cases) of the tinnitus.

## DISCUSSION

Tinnitus is usually divided into two categories: somato-sensory modulating tinnitus and pure somato-sensory tinnitus. Whilst the former often have an auditive origin, somato-sensory tinnitus is linked more to problems in the cranio-cervical area than to problems with the hearing system. [2-4]. Their appearance is linked to one or several factors:

- cranial and/or cervical trauma,
- dental, cervical or maxillary manipulation,
- chronic pain in the head, neck or shoulder girdle,
- unsuitable posture at rest, whilst walking, at work or during sleep,
- day time and/or nighttime bruxism.

There is no consensus in publications about the role of these various factors in the origin of tinnitus. Thus, epidemiological studies show that in their acute stage, cervical traumas are not associated with tinnitus. Only 10 to 15% of patients with a history of true cervical sprain would experience tinnitus or other otologic symptoms [4]. These delayed effects could be explained by abnormal muscular tension linked to the postures adopted by patients to avoid pain following the cervical trauma. These postures could also bring about lesions to the temporomandibular joints (TMJ) and post-traumatic malocclusion problems, which cause a dysfunction in the masticatory muscles which can, in the long term, bring about tinnitus. However, for certain authors, cervical sprains do not cause TMJ dysfunction. As for the role of the TMs in tinnitus pathogenesis of tinnitus, it is unlikely that this symptom is directly caused by a mechanical relationship between the masticatory system and the middle ear. In theory, TMJ pathologies could bring about a middle ear dysfunction via the anatomical connections between these structures. But recent anatomical studies do not confirm the possibility that traction of the various ligaments of this joint could bring about movements in the middle ear auditory ossicles. Furthermore, it is known that, due to a joint innervation by the trigeminal nerve, neuromuscular dysfunction in the masticatory muscles can bring about reflex hypertonia in the middle ear muscles, along with myoclonia of the palate which can cause tinnitus. [3].

In rare cases, irregular tonus in the soft palate tensor muscle can bring about a dysfunction in the eustachian tube, which can then cause congestion in the ear and tinnitus.

It has also been shown that contraction or specific palpation of certain muscles which regulate existing cases of tinnitus can cause tinnitus in healthy individuals who never previously suffered tinnitus. In a controlled study, *Sanchez et coll.* achieved a regulation of the intensity and pitch of the tinnitus in around 60% of patients by means of specific actions in the cranio-cervical area [5]. At the same time, the authors showed that these same manipulations brought about temporary tinnitus in 14% of the individuals without symptoms who were tested. Repeating this type of treatment would increase its efficacy but would not change the patient's perception of the discomfort felt on a day-to-day basis. These phenomena could be explained partly by the interaction between the auditory system and the somatosensory system, and partly by the role of neuroplasticity in the mechanism of tinnitus. It has currently been well established that the afferences of the head and neck muscles, particularly conveyed by the second cervical nerve and the trigeminal nerve (fifth cranial nerve) interact with the auditory afferences at the level of the dorsal cochlear nucleus (1st relay of the auditory pathways). The latter, located in the brain stem, has a decisive role in tinnitus. Through these anatomical connections, somatosensory influences regulate the sensitivity of the central auditory pathways; bring about an anomaly in neuronal activity which, in turn, may result in the perception of tinnitus. Thus, according to the theory of neuroplasticity, tinnitus may be the result of an abnormal interaction between the different sensorial modalities, the sensory-motor systems and the cognitive and emotional networks. The uncovering of "latent" synapses, the reduction in inhibition and the creation of new connections thanks to new axon growth are early manifestations of this neuroplasticity.

They bring about a lateral distribution of neuronal activity and stimulate the development of areas of hyper-responsiveness in the central nervous system. In the longer term, a re-organisation of the tonotopic receptive areas is observed in certain relays of the auditory pathways like the cochlear nucleus, the lower quadrigeminal body and in the auditory cortex. These phenomena, based on the neuronal plasticity of the central auditory system and on the possibility of it being activated by pathways which are not specifically auditory, would thus be responsible for somatosensory tinnitus.

Even if we do not yet know the origins of tinnitus, it seems that tinnitus results from altered or cross-modal synaptic activity [3]. Some cases of tinnitus had a toxic origin, like excessive release of glutamate in the ear. Severe noise can cause damage to the inner ear with "changes in glutamatergic or GABAergic neurotransmission or neuroinflammation" [6]. N-methyl-D-aspartate receptors (NMDA-Rs) are located at each synapse in the lower auditory pathway. They are characterized by a slow and long-lasting excitatory response upon glutamate activation, promoting protective and neurotrophic roles following acute insult by regulating AMPA-R expression and assisting in the restoration of synaptic inputs. This contrasts with chronic damage where overactivation of NMDA-Rs is implicated in neuronal death [7]. These functions are thought to be involved in auditory diseases, including noise-induced hearing loss, neural presbycusis, and tinnitus via aberrant excitation. This explanation is coherent with Aoki's publication [8], explaining the antinociceptive action of BoNTA: as stated, more than an

inhibition of acetylcholine release, BoNTA "inhibited several of the neurophysiological and neurochemical effects…, including glutamate release". It is also stated by other, later, publications [9].

Despite the significant unmet clinical need for a safe and effective drug for tinnitus relief, there is currently no approved drug on the market to treat this kind of pathology [10]. The effect of BoNTA on tinnitus is nevertheless already known. First reports came at the beginning of the 21th century, one of the first of them reporting successful treatment with electromyography-guided intramuscular injections of type A botulinum toxin in a woman who developed distressing tinnitus related to contractions of re-innervated masticatory muscles, caused by a post-polio syndrome that gave rise to an acoustic resonance phenomenon transmitted to the middle ear as an audible sound [11]. A lot of papers put the emphasis, during the same period, on objective tinnitus, coupled with forced eyelid closure syndrome [12], or on essential palatal tremor [13-16]. Each article described intra-palatal botulinum toxin injection. Only a few reports put the emphasis specifically on botulinum toxin injections in auricular muscles [17] or administration near the stapedial muscle [18]. Our results would seem to be unique as they stated that efficacy could be gained in the treatment of tinnitus by directly injecting the temporal and masseter muscles, whatever the primitive cause of tinnitus was. This study is limited by its conception and methodology: it is a retrospective review of a prospective database, and as such a recall bias and selection bias may be present. Furthermore, tinnitus was a relatively rare pathology in this series which was not designed specifically for studying its treatment. Larger, prospective studies are required to fully understand the implications of our findings, and especially to know if our injections of BoNTA acted on glutamate release, or on other neurotransmitters.

## CONCLUSIONS

Botulinum toxin could be a valuable treatment for tinnitus, whatever its origin, by simply acting on the easy to reach masseter and temporal muscles. In our study, the originality is that the effect on tinnitus is a side effect of the injection due to the high dosage of toxin put into the particular muscles against severe bruxism and diffusion of the product.

Confidence in our findings is reinforced by the fact that our analysis matched results validated in other studies in different ways.

## REFERENCES

1. Batifol D, Harding-Kaba B, Finiels PJ, Goudot P. Does Botulinum Toxin could reveal pre-existing asymmetry of jaw-lifting muscles in the treatment of bruxism? Pan Arab Neurosurgical J. 2013; 17.
2. Chrbolka P, Paluch Z, Alusik S. Current perspectives of tinnitus and its therapeutic options. European Geriat Med. 2015; 6: 170-174.
3. Haider HF, Hoare DJ, Costa RFP, Potgieter I, Kikidis D, Lapira A, et al. Pathophysiology, Diagnosis and Treatment of Somatosensory Tinnitus: A Scoping Review. Front Neurosci. 2017; 28: 207.
4. Londero A, Avan P, Bonfils P. Subjective and objective tinnitus: clinical and therapeutic aspects. EMC. 2008; 20.
5. Sanchez TG, da Silva Lima A, Brand o AL, Lorenzi MC, Bento RF. Somatic modulation of tinnitus: test reliability and results after repetitive muscle contraction training. Ann Otol Rhinol Laryngol. 2007; 116: 30-35.
6. Manohar S, Dahar K, Adler HJ, Dalian D, Salvi R. Noise-induced hearing loss: Neuropathic pain via Ntrk1 signaling. Mol Cell Neurosci. 2016; 75: 101-112.
7. Sanchez JT, Ghelani S, Otto-Meyer S. From development to disease: diverse functions of NMDA-type glutamate receptors in the lower auditory pathway. Neuroscience. 2015; 29: 248-259.
8. Aoki KR. Review of a proposed mechanism for the antinociceptive action of botulinum toxin type A. Neurotoxicology. 2005; 26: 785-793.
9. Láinez MJ, Piera A. Botulinum toxin for the treatment of somatic tinnitus. Prog Brain Res. 2007; 166: 335-338.
10. Langguth B, Elgoyhen AB. Current pharmacological treatments for tinnitus. Expert Opin Pharmacother. 2012; 13: 2495-2509.
11. Scolozzi P, Carrera E, Jaques B, Kuntzer T. Successful treatment of a postpolio tinnitus with type a botulinum toxin. Laryngoscope. 2005; 115: 1288-1290.
12. Kaffenberger TM, Mandal R, Schaitkin BM, Hirsch BE. Palatal botulinum toxin as a novel therapy for objective tinnitus in forced eyelid closure syndrome. Laryngoscope. 2017; 127: 1199-1201.
13. Olthoff A, Laskawi R, Kruse E. Successful treatment of autophonia with botulinum toxin: case report. Ann Otol Rhinol Laryngol. 2007; 116: 594-598.
14. Penney SE, Bruce IA, Saeed SR. Botulinum toxin is effective and safe for palatal tremor: a report of five cases and a review of the literature. J Neurol. 2006; 253: 857-860.
15. Slengerik-Hansen J, Ovesen T. Botulinum Toxin Treatment of Objective Tinnitus Because of Essential Palatal Tremor: A Systematic Review. Otol Neurotol. 2016; 37: 820-828.
16. Stidham KR, Solomon PH, Roberson JB. Evaluation of botulinum toxin A in treatment of tinnitus. Otolaryngol Head Neck Surg. 2005; 132: 883-889.
17. Lee K, Chang J, Park S, Im GJ, Choi HJ, Kim JH, et al. Bilateral muscular tinnitus due to myoclonus of extrinsic auricular muscles. Auris Nasus Larynx. 2015; 42: 173-175.
18. Liu HB, Fan JP, Lin SZ, Zhao SW, Lin Z. Botox transient treatment of tinnitus due to stapedius myoclonus: case report. Clin Neurol Neurosurg. 2011; 113: 57-58.

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