

ORTHODONTICS:

Botulinum Toxin A is an effective in cases of oromandibular dysfunction even if previous bite splint therapy has proved unsuccessful.

Author: Gobel H et al.

Source: Cephalalgia 2001;2 1(4):514-515 (1 Page).

Type: Abstract

Abstract

Full text of abstract:

Objectives: Oromandibular dysfunction is a frequent cause of the development of chronic headache and of chronification of tension-type headache or an increase in the frequency of migraine attacks. The patients are typically found to have parafunctions and painful masticatory muscles at rest or on palpation. A conventional therapy is to fit a bite splint. This therapy has disadvantages, however: the bite splint is time-consuming to fit and unpleasant to wear, and its therapeutic efficacy is frequently suboptimal. This study set out to investigate whether treatment with botulinum toxin A was successful in patients with oromandibular dysfunction even if previous bite splint therapy showed no effect.

Methods: Fourteen patients with oromandibular dysfunction according to the IHS criteria and previous unsuccessful therapy with a bite splint were included in the study. They were treated with Botulinum Toxin A (Botox), a total of 100 MU being injected in three injection sites each in the left and right masseter and two sites each in the left and right temporal muscle. In each case ten MU was injected into locally pressure-sensitive trigger points. Efficacy was continuously recorded with the aid of a standardized pain diary.

Results: In eleven of fourteen patients there was a reduction of pain after 12+/-4 days. After 28+/-11 days there was found to be a plateau phase of pain alleviation with an average pain reduction of 82% of the initial value. In six patients the pain disappeared completely. The pain reduction lasted for an average of 104+/-24 days. Five patients reported a reduction in migraine days by an average of 74% per month. All patients requested a follow-up injection. Four patients reported side effects during the first ten days in the form of an aching sensation (like post exercise muscle soreness) in the muscles treated. No other side effects were reported.

Conclusions: Botulinum Toxin A is effective in the treatment of painful oromandibular dysfunction even if previous bite splint treatment has failed to achieve a therapeutic effect. The functionality of the masticatory apparatus is maintained. Tolerance is very good. By contrast, with cost-intensive and time-intensive bite splint therapy, treatment with botulinum toxin A may be regarded as an important therapeutic option for oromandibular dysfunction. 0 Refs.

The use of botulinum toxin for the treatment of temporomandibular disorders: Preliminary findings.

Author: Freund B et al.

Source: J Oral Maxillofac Surg 1999;57(8):916-921.

Type: Article

Abstract:

Purpose: The aim of this study was to evaluate the response of patients with temporomandibular disorders to Botulinum toxin A (BTX-A) therapy.

Methods: The 15 subjects enrolled in this uncontrolled study were diagnostically categorized and treated with 150 units of BTX-A. Both masseter muscles received 50 units each under electromyographic (EMG) guidance. Similarly, both temporalis muscles were injected with 25 units each. Subjects were assessed at two-week intervals for eight weeks. Outcome measures included subjective pain by visual analog scale (VAS), measurement of bite force, interincisal opening, tenderness to palpation, and a functional index based on multiple VAS.

Results: All mean outcome measures, with the exception of bite force, showed a significant ($P = .05$) difference between the preinjection assessment and the four follow-up assessments. No side effects were reported.

Conclusions: BTX-A injections produced a statistically significant improvement in four of five measured outcomes, specifically pain, function, mouth opening, and tenderness. No statistically significant changes were found in mean maximum voluntary contraction or in paired correlation of factors such as age, sex, diagnosis, depression index, or time of onset. There is a discussion that follows by GT Clark, DDS, MS. He concludes that, for now, botulinum toxin injections should be restricted to cases with true muscle spasm disorders (dykinesias and dystonias) and not used for myofascial pain or fibromyalgia outside of an experimental setting where proper control conditions are included and informed consent of the subjects is provided. 30 Refs.

Uses of botulinum toxin:

Author: Girdler NM.

Source: Lancet 1997 Mar 29;349(9056):953.

Type: Letter

Abstract:

Full text of letter to the Editor: Naumann and Brin and colleagues describe the therapeutic use of botulinum toxin.

It is astonishing that one of the most lethal biological toxins known to man is proving to have increasing therapeutic value. The ability of this toxin to produce chemical denervation of muscle means that it has exciting potential use in many neuromuscular and ophthalmic disorders. The author has used botulinum toxin to successfully alleviate the debilitating facial pain that arises from the chronic muscle spasm of facial arthromyalgia. This condition afflicts over 25% of the population at some stage of their life. Spasm in the muscles of mastication leads to trismus, facial pain, and limitation of jaw function. Although of huge importance to dental, oral, and maxillofacial surgeons this condition is poorly recognized by rheumatologists and orthopedic specialists. Current treatment options including bite guards, anti-inflammatory analgesics, physiotherapy, tricyclic antidepressants, and surgery have a variable success rate. The author obtained informed consent from a thirty-four year old man with a six-year history of facial arthromyalgia that was unresponsive to standard treatment. Two-hundred-fifty units of botulinum toxin type A (Dysport, Speywood Pharmaceuticals Ltd, Maidenhead, UK), dissolved in 1.25 mL saline were injected into the bulk of each masseter muscle. The solution was dispersed by massaging the muscle for 20 minutes. At review ten days later the patient reported minor discomfort and bruising for 24 hr, followed 48 hr later by complete cessation of facial pain. Jaw function had improved dramatically and he had been able to eat his first pain-free meal for 6 years. At six-month review the patient still had painless jaw function and there were no signs of recurrent muscle spasm or joint dysfunction. Botulinum toxin prevents acetylcholine release and causes functional neuromuscular denervation. It temporarily paralyses the affected muscle and provides relief from the pain of chronic muscle spasm. This treatment could give relief to patients with intractable facial arthromyalgia that has failed to respond to standard management regimens. 4 Refs.

Injections of botulinum toxin for the treatment of deep bite of the lower lip after traumatic brain injury (GER).

Author: Weinsheimer C et al.

Source: Neurol Rehab 1997;3(3):180-1.

Type: CASE REPORT

Abstract: This German article has an English abstract only.

A case is described involving a female patient suffering from cerebral brain damage who bit her lower lip. The wound was self-inflicted because the patient's bite and suction reflexes were still intact. After standard treatment of the wound did not promote healing, botulinum toxin type A was injected into the masseter muscle and the temporal muscle. This treatment resulted in complete healing of the wound. 6 Refs.

Morphological and functional-changes in masseter after administration of botulinum toxin for treatment of masseteric hypertrophy.

Author: Manisali Met ai. Source: J Dent Res 1996 May;75(5):1164.

Type: Abstract

Masseteric hypertrophy has been treated by surgical reduction of the muscle bulk until the recent introduction of intramasseter injection of botulinum toxin. The early results of this non-surgical technique have been very encouraging, but the quantification of the treatment effect has not been well researched. Ten patients with bilateral or unilateral hypertrophy were studied in an attempt to establish the efficacy of botulinum toxin in the correction of contour deformity. All patients received twenty mouse units of botulinum toxin directly instilled into the affected muscle(s) followed by a further 100 mouse units after a two week interval. Quantification of effects on contour were recorded using laser scans, photographs and ultrasound scans. Functional effects were monitored using bite force measurements and electromyographic recordings. Results show a significant rapid reduction in muscle bulk with minimal impairment of function although a transient weakness of facial muscles was seen following 4 of the 20 treatments.

Conclusion: From this study it appears that botulinum toxin is a safe, simple, effective treatment for masseteric hypertrophy. 0 Refs.

Botulinum toxin in oromandibular dystonia brain injury. The treatment of following traumatic brain injury.

Author: Yen S et al.

Source: Arch Phys Med Rehabil 1996 Sep;77:955 Poster 64.

Type: Abstract

Abstract:

Full text of meeting poster: The authors report that perioral injections of botulinum toxin are useful in oromandibular dystonia (OMD) in patients with traumatic brain injury (TBI). A sixty-four year old man suffered a gun shot wound to the left parietaloccipital area and right maxillary intra-orbital region, followed by extensive debridement of left hemisphere. He presented with right hemiplegia, right masseter spasticity, and was unable to open his mouth. Ten units of botulinum toxin were injected under EMG guidance in right masseter. Twenty-four hours post-injection, the patient regained oral motor movement allowing him to smile, open his mouth and protrude his tongue. A nineteen year old man with anoxic encephalopathy, secondary to near-drowning presented severe OMD, causing him to bite his tongue continuously. No passive or active oral mandibular movement was possible. Five units of botulinum toxin were injected under EMG guidance in distal masseter, proximal masseter and temporalis, bilaterally. Two days post-injection, the dystonia of the masseter and temporalis muscles were sufficiently relieved to allow passive opening of the patient's mouth, decrease the tongue biting and permit oral hygiene. These cases demonstrate that botulinum toxin can

be useful in the treatment of OMD in patients with TBI by improving oral-motor movement and facilitating oral hygiene. 0 Refs.

Oromandibular dystonia treated with botulinum toxin: report of case.

Author: Heise GJ et al.

Source: J Oral Maxillofac Surg 1995;53(3):332-7.

Type: CASE REPORT

Abstract:

The authors describe a case report of a 34-year-old woman complaining of right temporomandibular joint (TMJ) pain and severe muscle spasms that frequently prevented her from opening her mouth. She had been aware of the problem for 15 years. A bilateral sagittal split osteotomy was performed to achieve proper occlusion, eliminate the appliance dependency, and possibly alleviate the muscle spasms. Minor postsurgical orthodontic treatment was also planned. Three days later the patient was seen for a complaint of right-sided facial pain, but examination did not reveal any significant findings other than the continued muscle fasciculations. Two days later the patient was taken back to the operating room where rigid fixation was reestablished using three 2-mm screws. Twelve days after the original osteotomy the patient again exhibited a mobile right proximal segment apparently as a result of the uncontrolled oromandibular dystonia. The Neurology service subsequently injected sixty units of botulinum toxin A into the right masseter and thirty units into the right temporalis muscle. This significantly reduced the dystonia over the next forty-eight hours. The patient was returned to the operating room a few days later for further administration of thirty units of botulinum toxin A into the masseter and placement of two six-hole miniplates with ten two-mm monocortical screws, and skeletal maxillomandibular fixation. Two years postoperatively, the patient continues to be observed. Her OMD is well controlled with injections of botulinum toxin every three months. Her apertognathia is of minimal concern because she has no difficulty eating. Also attached is a discussion by MF Brin, A Blitzer and CF Stewart regarding this article. Included in this article is a table on the differential diagnosis of oromandibular dyskinesias. 83 Refs.

Treatment of chronic masseter pain with botulinum toxin injections.

Author: Airolidi RL et al.

Source: J Dent Res 1993;72(Abstr Spec Issue):337.

Type: Abstract

This study investigated the effect of botulinum toxin on chronic masseter pain associated with parafunction. Five patients between the ages of twenty and sixty-nine years (mean = thirty-nine) with long histories of unilateral masseter pain refractory to self-administered physical therapy and bite splint underwent one injection of twenty units (= eight ng) botulinum toxin A (Oculinum @) in the afflicted muscle under EMG control. Patients were examined one week before and 2, 6, 16, and 24 weeks after the injection. At each visit patients reported the pain intensity (VAS scale) and underwent a clinical examination of masticatory, neck muscles, TMJs and jaw mobility. In addition the surface EMG of both test and control masseters was recorded ten times with reproducible electrode position during maximum clench (two s each). EMG RMS values were averaged over trials. The session's mean RMS value of the test masseter was divided by the mean RMS value of the control masseter obtained by averaging the values of all trials to analyze whether or not the EMG RMS varied over time. Three patients reported that the pain improved after two weeks and the other two after six weeks. However, neither the VAS scores for pain intensity nor the palpation scores decreased significantly during the examination period ($p > 0.05$, Friedman Test). The median values of the EMG RMS ratios were 0.5 at beginning, 0.29 at two weeks, 0.27 at six weeks, 0.29 at four months, and 0.69 at eight months. These values did

not vary with statistical significance between sessions ($p > 0.05$, Friedman Test). The results suggest that the subjective improvement in pain was non-specific and not related to the botulinum toxin treatment. 0 Refs.

Brief Report: Jaw dystonia triggered by biting into hard food.

Author: Lagueny A et al.

Source: Mov Disord 1991 Jan;6(2):174-176.

Type: Article

Abstract:

The authors report an unusual case of eating dystonia induced by attempts to bite into hard food and resulting in prolonged jaw opening spasms. Mastication was severely impeded. Simultaneous bilateral electromyogram (EMG) of the masticatory muscles during mastication of hard food showed an abnormal prolonged activity in the anterior digastrics with an abnormal co-contraction of these muscles during contraction of the jaw-closers and a prolonged inhibition of the jaw-closers until the hard bolus was softened. This action dystonia seems to be triggered when more than a certain pressure is exerted on the peridontal mechanoreceptors and could result from a defective central command control of their sensory inputs. Botulinum toxin was injected into both digastrics and lateral pterygoids and three months later there was no mandibular pain, however, eating time was not reduced much and mastication was improved only mildly. 11 Refs.

DENTISTRY

Treatment of bruxism with botulinum toxin. Tratamiento del bruxismo con toxina botulinica (SPA).

Author: Palazo Garcia R et al.

Source: Rehab (Madr) 2001;35(4):253-255.

Type: CASE REPORT

Abstract:

This Spanish article has an English abstract only. Bruxism, or rhythmic grinding of the teeth, is generally considered as a parasomnia, but it can also be found as a pyramidal release syndrome after a cerebrovascular accident that occurs with the locked-in syndrome. The case of a fifty year old woman who suffered vertebro-basilar infarct that evolved with coma and tetraplegia and who was in a vegetative state for one year is presented. She received respiratory physiotherapy and passive mobilizations for the first 22 months. This was assessed after, when she presented aphasia, spastic tetraparesis with right predominance and intense bruxism. Oral anti-spastic medication was initiated and succeeded in partially controlled the limb tone, but not the bruxism. It was decided to use botulinic (Sic) toxin for this. After the injection of 100 IU Botox in each masseteric muscle and of fifty IU at three months, it was possible to go from a continuous bruxism with difficulty to open the mouth to isolated episodes of nighttime tooth grinding and capacity to performing orolingual praxia. 10 Refs.

Bruxism and masticatory myalgias: Use of botulinum toxin.

Author: Fross RD.

Source: Mov Disord 2000;15(Suppl 2):35.

Type: Abstract.

This is the full text of the meeting abstract from the International Conference 1999: Basic and Therapeutic Aspects of Botulinum and Tetanus Toxins in Orlando, FL, Nov 16-18, 1999.

Temporomandibular joint (TMJ) disorders are often associated with bruxism, teeth grinding, and jaw clenching. When chronic, this involuntary jaw muscle contraction leads to masticatory myalgias and associated muscular headaches; teeth become damaged, and TMJs undergo accelerated degenerative changes. One method to stem this cycle of deterioration includes focal chemodenervation of the muscles of mastication using botulinum toxin injections. This paper reports the use of botulinum toxin in eighteen patients with chronic bruxism and masticatory myalgias. Fourteen women and four men had been experiencing chronic temporal and jaw muscle pain and tenderness for at least three years, and all used oral splints. Three patients had had local trigger point injections into jaw muscles, with limited and transient benefit. Five women had had TMJ arthrocenteses at least once. Six patients experienced frequent migraine headaches; eleven experienced temporal headaches in addition to jaw myalgias. Seven were aware of involuntary jaw clenching while awake; most described observer-reported evidence of grinding while asleep. All had palpable tenderness in some muscles of mastication (temporalis, masseter, pterygoid). No involuntary movements were seen during examination. On initial examination, most had some limitation of jaw opening, and masseter or temporalis hypertrophy. Eleven had findings of TMJ tenderness or crepitation. All were treated with intramuscular injections of botulinum toxin A; average dosages were twenty units per temporalis (range 10-50), thirty units per lateral pterygoid (range twenty to forty), and sixty units per masseter (range thirty to eighty). Injections were bilateral in seventeen patients; only five patients received lateral pterygoid muscle injections routinely. Injections were repeated every three months. Eleven patients have been receiving injections for two years, all others from one to five years. Injections were performed by palpation technique (without EMG guidance). Side effects were minimal, with one intramuscular hematoma, post-injection tenderness in seven patients, and transient weakened chewing strength in thirteen patients (while only one patient changed food texture for several weeks). All patients reported significant reduction in the use of pain medications (eleven were able to discontinue regular analgesic use), with jaw pain reduction from an average pre-injection score of eight (out of ten), to an average post-injection score of two, lasting an average of ten weeks but never returning to pre-injection levels. The patients with migraine headaches reported an average reduction of headache frequency by forty percent. It can be concluded that botulinum toxin injections provide a significant reduction in masticatory myalgias in patients with bruxism. 0 Refs.

Unilateral temporalis muscle hypertrophy managed with botulinum toxin type A.

Author: Isaac AM.

Source: Br J Oral Maxillofac Surg 2000Oct;38(5):571-572.

Type: Case Report

Abstract: Full text of letter less figures.

The authors have recently treated a case of idiopathic unilateral temporalis muscle hypertrophy with botulinum toxin type A (BtA). Treatment was successful and there were no appreciable side effects. The beneficial result persisted for 12 months. The condition has previously been described as one that requires supportive treatment, with surgery confined to biopsy. Its management has been largely conservative, with the detection and correction of parafunctional jaw movements that lead to reduced muscle hypertrophy. There have been no previous reports of successful treatment with BtA. Temporalis muscle hypertrophy is less common than masseteric hypertrophy, which has been successfully treated by intramuscular injection of BtA. Other conditions such as strabismus, blepharospasm, spasmodic torticollis, laryngeal dysphonia and hemifacial spasm have also been treated successfully. BtA inhibits acetylcholine vesicle release that is caused by the light chain moiety uniting with the SNAP-25 protein complex in the intracellular substrate adjacent to the motor end plate membrane. The muscle recovers when new neuromuscular junctions form after a period of two to four months. The treatment is then repeated if necessary.

CASE REPORT: A thirty-five year old white man presented with a gradually increasing painless swelling over the left temporal region, which had initially been noticed by his relatives eight months previously. The lesion had not changed

except by increasing in size, and no other abnormality had been noticed. That the patient was otherwise fit and well except for mild psoriasis, which was treated with calcipotriol cream. Some eight years previously he had suffered from mild 'migraine-type' headaches for which no drugs had been prescribed and which had resolved spontaneously after a year. There had been no history of temporomandibular joint dysfunction, masticatory parafunctional habits, mental illness or emotional disturbances. On examination there was an obvious, non-tender, firm, diffuse swelling over the left temporal region, which extended across the entire site of the temporalis muscle. The lesion became more prominent when he clenched his teeth. There was no clinical evidence of parafunctional jaw movements or habits. A computed tomogram of the region showed only generalized mild enlargement of the left temporalis muscle with no underlying bone abnormality. The lesion was biopsied under general anesthesia through a small scalp incision, and histological examination showed normal striated muscle tissue. After the biopsy, the hypertrophied muscle was injected with 500 units of BtA (Dysport, Speywood Pharmaceutical Ltd). The toxin was placed relatively deep within the muscle mass at several sites to take account of the anatomy of the deep temporal nerves, which normally enter on the deep aspect of the muscle. The patient was reviewed postoperatively and complained of mild discomfort in the area that resolved after a few days. Further review appointments were arranged one week and eight weeks postoperatively. At the later review the muscle contour had returned to normal and there was little obvious function of the left temporalis muscle when he clenched his teeth. He was very pleased with the treatment result. This case suggests that the treatment of temporalis hypertrophy with BtA could be an effective option to conservative treatment or surgical intervention, where the muscle bulk is disfiguring. Although BtA is expensive (cost is £170 for 500 units), intramuscular injection can be done in the outpatient clinic and costs compare favorably to those of operation.

There have been reports of tolerance to BtA developing after repeated intramuscular injections, which is associated with the development of antibodies to BtA. In such cases, Botulinum toxin type F (BtF) is an alternative for the treatment of blepharospasm, torticollis, and other disorders. However, the duration of action of BtF is shorter than that of BtA. It may be necessary to try the alternative toxin to treat this patient should tolerance develop to BtA. To the authors' knowledge temporalis hypertrophy has never previously been reported as being successfully treated with BtA, and it may well be beneficial.

Refer to AN- 2001030291 for correct correspondents' address (Anne Marie Isaac). Also see letter &om M Clark and D Kippel (AN- 2002020262.) 7 Refs.

Cosmetic oral and maxillofacial surgery options.

Author: Niamtu J.

Type: Article

Abstract Background:

Dentistry and its related specialties have made exponential increases in the functional and cosmetic treatment of the maxillofacial region. Oral and maxillofacial surgeons historically have been involved in functional and cosmetic rejuvenation of the face, and newer technologies have enhanced the ability to make patients look and feel better.

Methods: Cosmetic oral and maxillofacial surgery is being taught in residency programs, and is included in the oral and maxillofacial surgery board examinations and represents a part of contemporary oral and maxillofacial surgery. The author discusses common facial rejuvenation procedures with an emphasis on newer treatment technologies.

Results: Many oral and maxillofacial surgeons have the ability to improve the esthetics of the maxillofacial area and related structures. The large number of aging baby boomers and technological advances in cosmetic facial surgery have made these procedures easier to perform and more popular than ever.

Conclusions: A global diagnosis and treatment plan to include facial esthetics can enhance cosmetic dentistry and serve to name the work of the restorative dentist. The oral and maxillofacial surgeon can help the dentist and patient pursue both functional and cosmetic improvement with safe and effective procedures.

Clinical Importance: All dentists should be aware and abreast of advances in all areas of dentistry and have a basic understanding of available procedures that can benefit their patients. Cosmetic oral and maxillofacial surgery can enhance the work of the restorative dentist and improve facial esthetics, as well as enhance the well-being of the patient. Botulinum neurotoxin A (BoNT-A) can be used for treating masseteric hypertrophy. It is also being evaluated for the paralysis or weakening of other masticatory muscles to decrease temporomandibular joint, or TMJ, dislocation. 22 Refs.

Treating severe bruxism with botulinum toxin.

Author: Tan EK and Jankovic J,

Source: J Am Dent Assoc 2000 Feb;131(2):211-216.

Type: Case Report

Abstract Background:

Locally administered botulinum toxin, or BTX, is an effective treatment for various movement disorders. Its usefulness in treating bruxism, however, has not been systematically evaluated.

Subjects and Methods: The authors studied eighteen subjects with severe bruxism and whose mean duration of symptoms was 14.8 +/- 10.0 years (range three-40 years). These subjects audibly ground their teeth and experienced tooth wear and difficulty speaking, swallowing or chewing. Medical or dental procedures had failed to alleviate their symptoms. The authors administered a total of 241 injections of BTX type A, or BTX A, in the subjects' masseter muscles during 123 treatment visits. The mean dose of the BTX A was 61.7 +/-11.1 mouse units, or MU (range 25-100 MU), per side for the masseter muscles.

Results: The mean total duration of response was 19.1 +/- 17.0 weeks (range six-78 weeks), and the mean peak effect on a B-scale of 0 to 4, in which 4 is equal to total abolishment of grinding, was 3.4 +/- 0.9. Only one subject (5.6 percent) reported having experienced dysphagia with BTX A.

Conclusion: The results of this study suggest that BTX administered by skilled practitioners is a safe and effective treatment for people with severe bruxism, particularly those with associated movement disorders. It should be considered only for those patients refractory to conventional therapy. Future placebo-controlled studies may be useful in further evaluating the potential of BTX in the treatment of bruxism. 33 Refs.

Botulinum toxin Type A treatment of cosmetically disturbing masseteric hypertrophy (DUT).

Author: Rijdsdijk BA et al.

Source: Ned Tijdschr Geneesk 1998;142(10):529-32.

Type: CASE REPORT.

Abstract: this Dutch article has an English abstract only.

Two patients, a woman aged twenty-one and a man aged twenty-nine, with asymmetrical swellings of both mandibular angles and a painful, heavy sensation in the masticatory muscles (and in the woman also round the maxillary joint), were diagnosed as having hypertrophy of the masseter muscles. Both had the habit of jaw clenching and tooth grinding. Treatment consisted not of the traditional surgical debulking which also allows correction of overdeveloped osseous mandibular angles, but of injections with botulinum toxin type A. Injection of 40-60 IU (product: Botox) per

muscle was followed by some atrophy; cosmetically satisfactory results were achieved after repetition of the treatment a few months later. Reduction of muscle volume was confirmed by a quantitative volumetric assessment of MRI scans. In the female patient, the pain also abated. 16 Refs.

Bruxism after brain injury: Successful treatment with botulinum toxin-A.

Author: Ivanhoe CB et al.

Source: Arch Phys Med Rehabil 1997 Nov;78(11):1272-3.

Type: Case Report.

Abstract:

Bruxism, the rhythmic grinding of teeth-usually during sleep-is not an infrequent complication of traumatic brain injury. Its prevalence in the general population is 21 %, but its incidence after brain injury is unknown. Untreated, bruxism causes masseter hypertrophy, headache, temporomandibular joint destruction, and total dental wear. The authors report a case of complete resolution of postanoxic bruxism after treatment with botulinum toxin A (BTX-A). The patient was a twenty-eight year old man with no history of bruxism who sustained an anoxic brain injury secondary to cardiac arrest of unknown etiology. On admission to the rehabilitation unit two months after the injury, the patient presented with severe bruxism and heavy dental wear. The patient was injected with a total of 200 units of BTX-A to each masseter and temporalis. There was total resolution of bruxism two days after injection, with no complications. On follow-up three months after injection, the patient remained free of bruxism. The authors proposes that botulinum toxin be considered as a treatment for bruxism secondary to anoxic brain injury. Further studies regarding muscle selection and medication dosage are warranted to elucidate the toxin's efficacy in this condition. 12 Refs.

Treatment of jaw spasm in ALS with botulinum toxin.

Author: O'Connell BK et al.

Source: Neurology 1997 Mar;48(3 Suppl2):A128.

Type: Abstract.

Abstract: Full text of meeting abstract:

OBJECTIVE: To determine the usefulness of botulinum toxin injection in the treatment of severe jaw spasm associated with amyotrophic lateral sclerosis.

BACKGROUND: Amyotrophic lateral sclerosis (ALS) has long been recognized to cause profound changes in motor tone. As it is known to affect both upper and lower motor neurons, the symptoms present at any particular stage of disease depend on the relative involvement of these two neuronal populations. Spasticity in ALS has been traditionally treated with oral agents. The increasing use of botulinum toxin as a treatment for spasticity led us to use it in two ALS patients with jaw spasm.

DESIGN METHODS: The authors recently cared for two patients with ALS who demonstrated different degrees of jaw spasm: one with complete inability to open the mouth, with laceration of the lip by the teeth and complete obstruction of routine oral hygiene, and the other with limited jaw opening associated with pain. Conservative doses of botulinum toxin (12.5U) were initially injected into each masseter muscle of the more severely affected patient, as this muscle showed marked atrophy bilaterally. This was increased to 25U per masseter with addition of 20U in each temporalis muscle when treatment was repeated eight weeks later. In the more mildly affected patient (who still

retained ability to chew and to open his jaws approximately 2 cm) IOU doses were used in each masseter, with 5U given in each temporalis to allow for greater ease of jaw opening without compromising retention of secretions.

RESULTS: In the more severely affected patient, the initial 12.5U dosage resulted in mild relief of pain only; higher dosage provided complete relief of pain and restored the ability to open his mouth easily, permitting adequate oral hygiene and preventing any further laceration by the teeth. In the second patient with reduced dose, ability to open the mouth to eat was improved and pain was completely abolished. No difficulty with secretions was noted in either patient.

CONCLUSION: In the subgroup of ALS patients in whom jaw spasm is a marked and debilitating symptom, botulinum toxin injection is safe and effective for improving the quality of life by reducing the profound change of muscle tone associated with this disease. 0 Refs.

Hypertrophy of the masticatory muscles, bruxism and painful syndrome of the temporomandibular junction. Treatment with botulinum toxin type A.

Author: Chikhani L et al.

Source: Mov Disord 1995 May;10(3):396.

Type: Abstract.

Abstract: Full text of meeting poster presentation.

Patients with hypertrophy of the muscles of mastication, temporalis and masseters complain of chronic pain, tenderness, temporomandibular junction dysfunction difficulties and pain when chewing, culpal wear. They are often grinding their teeth during sleep and some of them feel disfigured. As botulinum toxin (BT) was successfully used in a routine clinic for muscles spasms, this treatment was thought to be efficient in such cases. Fifteen patients with mastication muscles hypertrophy were included. All of them had clinical evaluation, EMG recording and video before and three months after treatment. Patients were treated with BT type A (Dysport) injected in the masseter and/or temporalis muscle. Mean dose on masseter was eighty units. Fourteen out of fifteen patients showed significant improvement which lasted four to six months: pain disappeared, muscles hypertrophy dramatically decreased. Patients stopped grinding. They were satisfied with the cosmetic result. No adverse effect was observed. The balance of the jaw muscles was restored. However further studies are needed to evaluate the benefit on the temporomandibular junction disturbances. According to the authors results BT is a new and efficient treatment for hypertrophy of the masseter and temporal is muscles. 0 Refs.

Successful treatment of bruxism following brain injury with botulinum toxin-A.

Author: Lai Jenny M and Ivanhoe Cindy H.

Source: Arch Phys Med Rehabil 1995 Nov;76(11):1069 Poster 151.

Type: Abstract.

Abstract: Full text of poster.

Bruxism, the rhythmic grinding of teeth usually seen during sleep, is often seen in traumatic brain injury patients. Its prevalence in the general population is 21%, but its incidence after brain injury is unknown. Untreated, bruxism causes masseter hypertrophy, headaches, temporal mandibular joint destructions, and total dental wear. The authors report complete resolution of bruxism treated with botulinum toxin-A. The patient is a twenty-eight year old man with no previous history of bruxism who sustained an anoxic brain injury secondary to cardiac arrest. Two months after the injury, the patient, demonstrating severe bruxism, was admitted to rehabilitation. On examination, there was heavy dental wear. The patient was injected with a total of 200 units of botulinum toxin-A to each masseter and temporalis.

Total resolution of bruxism was seen two days post-injection. No complications were noted after the injections. On follow-up three months after injection, the patient remains free of bruxism. This case illustrates successful treatment of bruxism, and the authors propose that botulinum toxin be considered as a treatment for bruxism secondary to CNS injury. Further studies regarding muscle selection and dosages are warranted to elucidate the toxin's efficacy in this condition. 0 Refs.

Interventional neurology: Botulinum toxin as a potent symptomatic treatment in neurology.

Author: Giladi Net at.

Source: Isr J Med Sci 1994 Nov;30(11):816-9.

Type: Article

Abstract:

Local injections of botulinum toxin is a well-accepted treatment for focal dystonias, hemifacial spasms and strabismus. Its use by skilled neurologists has been reported to be safe and effective. They report their experience with botulinum toxin injections in 108 patients with various central nervous system disorders. Botox was effective in upper face dystonia (86% improvement), spastic dysphonia (92% improvement), platysma muscle spasms and spasmodic torticollis (range of movement 61%, pain and tension 90%). It was also very effective in a few patients with apraxia of eyelid opening, parkinsonian jaw tremor, teeth clenching, palatal myoclonus and adductor leg spasticity. No serious side effects were recorded. Botulinum toxin is a useful symptomatic treatment for many neurological disorders, and one of the leading mode of treatments in the new subspecialty in neurology called "Interventional neurology." 16 Refs.

Treatment approaches to bruxism.

Author: Thompson BA et al.

Source: Am Fam Physician 1994 May 15;49(7):1617-22.

Type: Article.

Abstract:

Bruxism, or the grinding and clenching of teeth, occurs in approximately fifteen percent of children and in as many as 96 percent of adults. The etiology of bruxism is unclear, but the condition has been associated with stress, occlusal disorders, allergies and sleep positioning. Because of its nonspecific pathology, bruxism may be difficult to diagnose. In addition to complaints from sleep partners, signs of teeth grinding include masticatory pain or fatigue, headaches, tooth sensitivity and attrition, oral infection and temporomandibular joint disorders. Signs of bruxism include tooth wear and mobility, as well as tender or hypertrophied masticatory muscles and joints. Children with bruxism are usually managed with observation and reassurance. Adults may be managed with stress reduction therapy, alteration of sleep positioning, drug therapy, biofeedback training, physical therapy and dental evaluation. If significant tooth attrition, mobility or fracture occurs, dental referral is mandatory. Methocarbamol (Robaxin) and injections of botulinum toxin have been anecdotally reported to be useful in the management of bruxism. 25 Refs.

Persistent dystonia possibly induced by flecainide.

Author: Miller LG and Jankovic J.

Source: Mov Disord 1992 Jan;7(1):62-3.

Type: CASE REPORT

A fifty-five year old man developed clicking of his teeth, involuntary biting, chewing and grimacing within three days of initiation of flecainide. The authors suggest flecainide should be added to the list of benzamide derivatives

associated with drug-induced movement disorders. After being unsuccessfully treated with a number of medications, the patient was administered fifty units of botulinum toxin. Within two weeks, his jaw was more relaxed. At the five-month follow-up he was given seventy-five units of botulinum toxin for mild to moderate trismus with bruxism which provided substantial relief for six months. 10 Refs.

Treatment of bruxism with botulinum toxin injections.

Author: Van Zandijcke M and Marchau MMB.

Source: J Neurol Neurosurg Psychiatry 1990;53(6):530.

Type: Letter

Abstract:

Botulinum toxin A successfully treated bruxism, a rhythmic grinding of the teeth, in a thirty-two year old woman. She was admitted in a coma after a car accident. A CT scan showed evidence of brain contusion. The patient was intubated and ventilated. She recovered slowly. After four months, the patient recovered somewhat and developed severe bruxism. This damaged the patient's teeth and annoyed nearby patients. Six months after the accident, 10ng botulinum toxin A were injected into the temporal and masseter muscles. Five days later, bruxism was markedly reduced. Favorable effects lasted for eight weeks. A second injection of 40ng botulinum toxin A was given. The results were similar and persisted twelve weeks later. Botulinum toxin A was safe and effective for the treatment of bruxism. 5 Refs.

Bruxism in cranial-cervical dystonia.

Author: Wooten MP and Jankovic J.

Source: Neurology 1990 Apr;40(4 Suppl):142.

Type: Abstract

To study the association between bruxism and dystonia, the authors reviewed data on 200 patients— 62 males and 138 females- with cranial-cervical dystonia. Thirty-seven patients (18.5%) - 15 males and 22 females - had bruxism. Similar to the other patients with cranial-cervical dystonia, the mean age at onset of dystonia in patients with bruxism was 53.1 years, and the mean duration of dystonia was 4.5 years. Involuntary oromandibular movements (eighteen patients) and blepharospasm (twelve patients) were the most common

Initial symptoms: Eleven patients with bruxism had associated tremor in the upper limbs. Six patients (16.2%) had an eighteen-degree relative with cranial-cervical dystonia, and sixteen (43.2%) had evidence of tardive dystonia. About one-third of bruxism patients had associated dental problems, including TMJ syndrome (24.3%) and tooth wear (8.1%), and eight (21.6%) had oromandibular dental or surgical procedures. While some patients improved with medications, botulinum toxin injections into the masseter, temporalis or both muscles provided the most satisfactory, albeit temporary, relief in most patients. 0 Refs.

ORAL SURGERY:

Management of facial pain with botulinum toxin in a tertiary pain clinic.

Author: Borodic Gary and Acquadro Martin.

Source: Naunyn Schmiedebergs Arch Phannacol 2002;365(Suppl 2):R14.

Type: Abstract.

Full text of abstract from the International Conference 2002 Basic and Therapeutic Aspects of Botulinum and Tetanus Toxins, Hannover, GER, June 8-12, 2002 meeting.

Chronic facial pain is a difficult management problem in a tertiary pain service. The following represents an open label single center trial targeting patients with chronic facial pain who failed conventional management efforts. Each patient failed at least three separate treatment modalities. In this series facial pain was classified as 1. trigeminal neuralgia 2. post-operative wound pain syndromes 3. essential headache- myofascial pain disorders and 4. temporal mandibular joint syndrome. A series of 110 patients with chronic facial pain were injected with botulinum type A toxin ranging from 25-150 LD 50 units given within multiple points over the dermatome involved with the chronic pain. At least two attempts at injection over a one months period was used to determine if botulinum toxin was efficacious in these syndromes. Endpoint was a patient's perception of at least 50% improvement in intensity and frequency of pain. Overall response rate was 73%, with the following response rates for the various syndromes. Trigeminal Neuralgia: (19/27) 70.4%, Post Operative Wound Pain (25/32) 78.1%, Essential Headache (28/36) 77.8%, TMD (618) 75%, Post Dental Extraction ('2J7) 28.6% (p<0.02). Post operative wound pain syndromes included enucleation, orbitectomy for lacrimal cancer, midfacial reconstructive surgery, parotidectomy, posterior craniotomy for the removal of acoustic neuroma, reconstructive facial and oral surgery, traditional and endoscopic sinus surgery, temporal mandibular joint surgery, blowout fracture repair, and cataract surgery. Chronic facial pain following post-dental procedures was associated with a poor result in this subgroup. Discussion of application of botulinum toxin for pain will be discussed in reference to anti-inflammatory properties and effects on peripheral and central neural sensitization. Pertinent bio-effects involving non-musculoskeletal tissues will be demonstrated and discussed. 0 Refs.

Progressive supranuclear palsy: An unusual cause of trismus.

Author: Warwick JP and Popat M.

Source: Anaesthesia 1997;52(12):1236.

Type: LETTER

Abstract:

A sixty-eight old man with a three year history of progressive supranuclear palsy was scheduled for the placement of a percutaneous endoscopic gastrostomy feeding tube. For the preceding six months he had developed worsening masseter spasm producing a complete trismus. Little benefit had been obtained from treatment with dantrolene by mouth and injection of botulinus (sic) toxin into both masseter muscles. Oral surgical assessment had failed to identify any intra-oral sepsis to account for the trismus and even after sedation with midazolam intravenously mouth opening was extremely limited with an interincisor gap of less than 1.5 cm. This complication of progressive supranuclear palsy has not been described before. 0 Refs.

Medical management of masseteric hypertrophy.

Author: Rogers BA and Whear NM.

Source: J Oral Maxillofac Surg 1995;53(4):492.

Type: LETTER.

Abstract:

The authors were interested to read the article on the management of masseteric hypertrophy, J Oral Maxillofac Surg, Nov 1994 (94071554), but were surprised to note that there was no mention of the medical management of this condition using Botulinum toxin type A. This has been reported in the British literature and has proved very successful in their own clinical experience. 2 Refs.

Botulinum toxin for treatment of masseteric hypertrophy.

Author: Hui ACF and Ho WS.

Source: J Neurol 2002 Mar;249(3):345.

Type: Letter.

Abstract: Full text of letter.

The indications for botulinum toxin (BTX-A) have expanded over the past twenty years; the supplement Evidence-based medicine (EBM) in botulinum toxin treatment was a welcome update on the established and emerging use of BTX-A for a number of neurological diseases. One disorder not included in the review by Jost and KoW is masseteric hypertrophy, a condition that can occur in oromandibular dystonia (OMD), particularly in the jaw closing type. BTX-A is injected frequently into the masseters in OMD and in temporomandibular joint disorder with promising results. However, the majority of patients with masseteric hypertrophy presenting with pain and cosmetic disfigurement have no obvious etiology. Current treatment for this group is surgical, with the aim of contouring of the prominent mandibular angle through bony reduction or osteoplasty and supplemental myotomy. Invasive treatment may be complicated by damage to the mandibular branch of the facial nerve, postoperative hemorrhage, infection, scarring and the effects of general anesthesia. The effect of BTX-A has been described in small series with good results persisting for six to twelve months with minimal side-effects. One study reported clinical and ultrasonic improvement with intramuscular injection of 100-300 units of BTX-A into each hypertrophic muscle. BTX-A may be a safer alternative to surgery but as yet there are no placebo-controlled randomized clinical trials for this condition. Errata from J Neurol 2002;249(6):790 - Omitted the co-authors name. The two authors are Dr ACF Hui and Dr WS Ho. 10 Refs.

PTERGYOID:

Recurrent trismus and stridor in an ALS patient: Successful treatment with botulinum toxin.

Author: Winterlwwler MGM et al.

Source: Neurology 2002 Feb 12;58(3):502-3.

Type: Case Report.

The prognosis of patients with ALS with severe bulbar symptoms is worse than for patients without bulbar symptoms. Some of these patients die suddenly and unexpectedly. Upper airway obstruction might be one of the causes. The authors report a patient with ALS who had recurrent trismus, stridor, and dyspnea. She refused non-invasive ventilation and tracheotomy. Anti-spastic treatment with baclofen (20 mg/day) and lorazepam was not effective. After shared decision making, and EMG-guided injection of 6.25 mouse units of BoNTIA (Botox, diluted in 0.5 mL NaCl 0.9%, Allergan Pharmaceuticals, Westport, Ireland) was given intramuscularly into each masseter and lateral pterygoid muscle (total dose 25 mouse units Botox). Three days after the injections, the masseter spasm and recurrent dyspnea stopped. She was now able to close and open her mouth voluntarily. No impairment of bulbar paresis or neurologic status was found. She remained masseter spasm-free and died from progressing ALS six months later at home. 7 Refs.

Bruxism and masticatory myalgias: Use Of botulinum toxin.

Author: Fross RD.

Source: Mov Disord 2000;15(Suppl 2):35.

Type: Abstract.

Abstract: This is the full text of the meeting abstract from the International Conference 1999: Basic and Therapeutic Aspects of Botulinum and Tetanus Toxins in Orlando, FL, Nov 16-18, 1999.

Temporomandibular joint (TMJ) disorders are often associated with bruxism, teeth grinding, and jaw clenching. When chronic, this involuntary jaw muscle contraction leads to masticatory myalgias and associated muscular headaches; teeth become damaged, and TMJs undergo accelerated degenerative changes. One method to stem this cycle of deterioration includes focal chemodenervation of the muscles of mastication using botulinum toxin injections. This paper reports the use of botulinum toxin in eighteen patients with chronic bruxism and masticatory myalgias. Fourteen women and four men had been experiencing chronic temporal and jaw muscle pain and tenderness for at least three years, and all used oral splints. Three patients had had local trigger point injections into jaw muscles, with limited and transient benefit. Five women had had TMJ arthrocenteses at least once. Six patients experienced frequent migraine headaches; eleven experienced temporal headaches in addition to jaw myalgias. Seven were aware of involuntary jaw clenching while awake; most described observer-reported evidence of grinding while asleep. All had palpable tenderness in some muscles of mastication (temporalis, masseter, pterygoid). No involuntary movements were seen during examination. On initial examination, most had some limitation of jaw opening, and masseter or temporalis hypertrophy. Eleven had findings of TMJ tenderness or crepitation. All were treated with intramuscular injections of botulinum toxin A; average dosages were twenty units per temporalis (range 10-50), thirty units per lateral pterygoid (range 20-40), and sixty units per masseter (range 30-80). Injections were bilateral in seventeen patients; only five patients received lateral pterygoid muscle injections routinely. Injections were repeated every three months. Eleven patients have been receiving injections for two years, all others from one to five years. Injections were performed by palpation technique (without EMG guidance). Side effects were minimal, with one intramuscular hematoma, post-injection tenderness in seven patients, and transient weakened chewing strength in thirteen patients (while only one patient changed food texture for several weeks). All patients reported significant reduction in the use of pain medications (eleven were able to discontinue regular analgesic use), with jaw pain reduction from an average pre-injection score of eight (out of ten), to an average post-injection score of two, lasting an average of ten weeks but never returning to pre-injection levels. The patients with migraine headaches reported an average reduction of headache frequency by forty percent. It can be concluded that botulinum toxin injections provide a significant reduction in masticatory myalgias in patients with bruxism. 0 Refs.

Four years experience in application of botulinum toxin A in recurrent dislocations of the TMJ.

Author: Umstadt HE et al.

Source: *Mov Disord* 2000;15(Suppl 2):35-36.

Type: Abstract.

This is the full text of the meeting abstract from the International Conference 1999: Basic and Therapeutic Aspects of Botulinum and Tetanus Toxins in Orlando, FL, Nov 16-18, 1999.

Common characteristics of chronic recurrent dislocations or luxations of the TMJ are functional restriction at the long term. In this study, the authors tried to treat causally without surgical invasion as well as little functional loss. Twelve patients with recurrent painful dislocations but regular joint morphology have been treated in the last four years under the supposition that muscular dyscoordination is causally. Morphometric studies about the lateral pterygoid muscle have been done in MRI before treatment and after restitution of the mandibular movement at the end of the effect of the toxin. They paralyzed both Mm. pterygoidei lat. using 100 IU Botulinum toxin A (Dysport, IPSEN Pharma). The correct position of the syringe was detected with EMG (Dantec Corp.) during voluntary motion by the patients. One week and eight weeks later, they were interviewed about frequency of dislocations, and a functional status was determined by clinical examination. No serious side effects were detected after EMG-controlled application of Botulinum toxin A. Maximal mouth opening was reduced under full action of the toxin to a mean of 32mm; it resolved after fourteen weeks (mean) to normal opening. Only in one case dislocations were persisting but in a lower frequency. In all other cases, no dislocations have been noticed any more during time of action of the toxin. In eight of twelve cases persistent decrease of dislocations was noticed even when the toxic effect wore off, what seems to conclude a

change in neuromuscular relations. When the effect of Botulinum toxin disappeared, MRI controls showed smaller volume of the Mm. pterygoidei lat. They repeated the injections after renewed appearance of dislocations in eight cases. Application of Botulinum toxin into the Mm. pterygoidei lat. seems to be a noninvasive and repeatable possibility to treat recurrent dislocations of the TMJ without negative side effects. They started a prospective multicenter study (twelve centers) in Germany to evaluate the effect of the toxin in cases of dislocations in a greater number of patients. 0 Refs.

Hallervorden-Spatz Syndrome

Author: Wittstock Met al.

Source: Mov Disord 2000;15(Suppl 2):36.

Type: Abstract

Abstract: This is the full text of the meeting abstract &om the International Conference 1999: Basic and Therapeutic Aspects of Botulinum and Tetanus Toxins in Orlando, FL, Nov 16-18, 1999.

Hallervorden-Spatz syndrome (HSS) is a rare condition characterized by early onset, progressive dystonia, rigidity or choreoathetosis, sometimes mild dementia and typical MRI findings. It predominantly affects the jaw muscles. Various treatment strategies have been tried, but are usually ineffective. The authors are reporting a 20-year-old patient with a 4-year history of severe progressive jaw opening dystonia with mandibular joint subluxations, limb choreoathetosis, 1Tontal lobe dysfunction and mild dementia. After various medications failed, botulinum toxin type A (BT) (Dysport, Ipsen Ltd., 500 MU in 2.5 ml 0.9% NaClH₂O) was injected into the pterygoid muscles (120 MU each side) using a transcutaneous approach through the incisura mandibulae under maximal jaw opening and into the mylohyoid muscles (80 MU each side). After 2 weeks, the frequency and severity of the jaw opening movements were reduced. Jaw protrusion and subluxation had stopped. Lateral control of the jaw, jaw closure, tongue movements and parotid function were preserved. After 3 months, the symptomatology began to reappear and BT therapy was repeated as before. BT injections into the pterygoid and mylohyoid muscles are safe and effective to control jaw opening dystonia, the key feature of HSS. They may also be used for jaw opening dystonia of other aetiologies. 0 Refs.

[Botulinum toxin treatment of neurogenic dislocation of the temporomandibular joint]. Botulinumtoxinbehandlung der neurogenen Kiefergelenkluxation (GER).

Author: Daelen B et ai.

Source: Mund Kiefer Gesichtschir 1998 May;2(Suppl 1):S125-9.

Type: Article

Abstract: This German article has an English abstract only.

Botulinum toxin leads to paresis of the skeletal muscle lasting 2-4 months via an inhibition of acetylcholine release at the neuromuscular junction. Since 1995, botulinum toxin injections have been used in the treatment of reCUITent dislocation of the temporomandibular joint (TMJ). The chemical denervation of the external pterygoid muscle restricts the angle of mouth opening, thus helping to prevent dislocation. TMJ dislocations that occur as a result of increased tone in the protracted masticatory muscles were recently termed as neurogenic dislocations of the TMJ. The authors conducted a clinical study to investigate the efficacy of botulinum toxin injections into the external pterygoid muscle in five patients with reoccurring neurogenic dislocations of the TMJ. In the 3 months prior to the first treatment, the patients had suffered a total of 19 dislocations. In the 3-month period following the initial treatment, only one woman experienced a dislocation. They performed the treatment a total of 25 times. Five dislocations occurred during the 6- to 36-month observation period. In the meantime, two patients remain reoccurrence-free 1 year after receiving treatment. All the patients had a restricted ability to open their mouths as a side effect of the weakening of the external pterygoid

muscle that was completely reversible over the course of 3-4 months. All other side effects were equally well-tolerated by the patients and fully reversible after 3 weeks at the most. In the two patients who remain reoccurrence free without any further treatment, the increased tone of the muscles serving the jaw normalized spontaneously over the course of the underlying neurological disease. The results show that, in the treatment of reoccurring neurogenic dislocations of the TMJ, botulinum toxin injections represent a therapeutic alternative that has few side effects. 27 Refs.

Treatment of recurrent dislocation of the temporomandibular joint with type A botulinum toxin.

Author: Daelen B et al.

Source: Int J Oral Maxillofac Surg 1997 Dec;26(6):458-60.

Type: CASE REPORT

Abstract:

A case is reported of a 56-year-old woman who suffered from reoccurring dislocations of the temporomandibular joint (TMJ) secondary to an exacerbated tetraspastic syndrome of multiple sclerosis. Following chemical denervation of the masseter and pterygoid muscles with injections of type A botulinum toxin, no further dislocations occurred for periods of up to four months. The treatment has been repeated five times. Some of the indications and possible adverse reactions to this therapy are discussed and comparisons made with other, conventional methods for managing recurrent dislocation of the TMJ. 7 Refs.

Tardive jaw tremor.

Author: Ebersbach G et al.

Source: Mov Disord 1997;12(3):460-2.

Type: CASE REPORT

Abstract: The authors describe the case of a patient in whom tardive tremor was confined to the jaw, producing constant and rhythmic clapping to the jaw, producing constant and rhythmic clapping of his teeth. The tremor involved all jaw-closing muscles and was characterized by a frequency of 2.5 Hz. There was complete suppression of tremor during voluntary jaw movement. The involuntary movements responded to combined treatment with tetrabenazine and tiapride. Postural tremor of the upper limbs (6 Hz) was the only additional movement disorder observed in the patient. Tardive jaw tremor has not been described in humans before. Neurophysiological studies of brain-stem responses included assessment of blink and masseter reflexes, which were normal. Magnetic resonance imaging of the brain also gave normal results. The first treatment tried was two sessions of local injection of botulinum toxin type A (Dysport). In the first session 140 U was injected into each masseter muscle and 40 U in each temporalis muscle, without benefit. In the second session, 2 months later, 280 U was injected on both sides into the masseter muscles, 120 U into each temporalis muscle, and 160 U (EMG guided) into each medial pterygoid muscle. Two weeks later atrophy of the injected muscles and mild paresis of jaw closure was observed, but the decrease in tremor severity was only minimal. The patient was subsequently started on tetrabenazine. When tetrabenazine had no effect at 200 mg per day, tiapride (300 mg per day) was added, and the tremor ceased completely. 14 Refs.

Medical treatment temporomandibular joint botulinum toxin A.

Author: Moore AP and Wood GD.

Source: Br Den J 1997 Dec 13-27;183(11-12):415-7.

Type: CASE REPORT

Abstract:

This paper describes a new technique for prophylactic treatment of recurrent mandibular dislocation using injection of botulinum toxin A (BtA) into the lateral pterygoid muscles. BtA temporarily weakens muscles by blocking acetylcholine release, and thus operates through a principle different from established treatments such as joint sclerosant therapy, eminectomy or Dautry's procedure. The patient suffered recurrent mandibular dislocations caused by tardive dystonia. The authors injected 75 mu BtA percutaneously into each lateral pterygoid muscle under electromyographic guidance. No further dislocation occurred over the subsequent 10 months, and follow-up continues. there were no immediate or delayed side effects. More experience is required before this becomes an established treatment. BtA is usually given in outpatients, and is less invasive or destructive than previous options. It may not be suitable if dislocation is due to lax ligaments or weak muscles. Operators must be aware that other BtA preparations require a different dose. 13 Refs.

Experience with long-term botulinum toxin treatment of oromandibular dystonia, guided by quantitative EMG.

Author: Erdal J et al.

Source: Mov Disord 1996;11(Suppl- Abstract P790):210.

Type: Abstract

Abstract:

Full text of meeting abstract: In a retrospective study the authors evaluated 13 consecutive patients (9 women) with 23 botulinum toxin (BT) treatment sessions for oromandibular dystonia. Mean age at first treatment was 53.3 years (range 26.9-67.0) and mean symptom duration 6.9 years (0.4-21.4). Symptoms were often varied but were primarily (no. of patients): jaw tension (4), jaw closure (2), jaw opening (3), difficulties swallowing (1), pharyngeal myoclonia (2) and cricopharyngeal spasms (1). Eight patients had other focal dystonias and two had generalized dystonia.

Pharmacotherapy was unsatisfactory in all patients. Several patients were severely disabled before BT treatment; pre- and post-treatment video recordings of a woman initially wearing a suspensory device to prevent jaw luxation will be shown. BT treatment was undertaken in collaboration between a neurologist, an otolaryngologist and a neurophysiologist. Clinically dystonic muscles were examined with turns-amplitude analysis of the intramuscular EMG and treated according to activity at rest, usually when turns >100 . Patients received a total of ninety-eight treatment sessions (mean 7.5 pr. patient range 3-23) with a total of 244 BT treated muscles (mean 2.5 pr. session, range 1-6). The most frequently treated muscles were (mean U Botox; no. of treatments): thyrohyoid (16 U; 41), masseter (23 U; 38) internal pterygoid (16 U; 37), geniohyoid (18 U; 32), external pterygoid (17 U; 31), digastric (21 U; 17), mylohyoid (16 U; 14), cricopharyngeal (10 U; 8), and sternothyroid (16 U; 7). Subjective effect was reported after 66/98 treatment sessions (67%) by 11/13 patients (85%). Two patients, one with jaw tension and one with jaw opening dystonia did not improve after 3 visits each and stopped BT treatment. One patient experienced secondary loss of effect and stopped BT treatment after 9 visits; BT antibodies were shown by in vivo assay. Six patients (46X) reported a total of 11 adverse events, of which dysphagia (one severe case) was by far the most common. The authors found that quantitative EMG is very useful for the selection of muscles to be treated and to determine the treatment dose. This study supports that BT is a safe and effective therapy for oromandibular dystonia. 0 Refs.

Treatment of oromandibular dystonia.

Author: Brookes GB and Harcourt JP.

Source: Mov Disord 1995 May;10(3):396.

Type: Abstract

Abstract:

Full text of meeting poster presentation: Oromandibular dystonia (OMD) is a disorder that affects the masticatory, lower facial and tongue muscles producing spasms and jaw deviation. These symptoms may be present alone, or in association with other focal dystonic movements, as in Meige's syndrome, or part of a more generalized dystonia. The efficacy of drug therapy is very limited. Previous studies have shown increased benefit with local botulinum toxin injections. Unfortunately good symptomatic improvement is only achieved in about 45% of patients, with a moderate response in a further 15%. During the last five years thirty-six patients with OMD have been assessed at the Neurolaryngology Clinic and the majority treated with botulinum toxin. There were twenty-eight females and eight males; six were suffering from Meige's syndrome. The spectrum of muscles injected in order of frequency were masseter, lateral pterygoid, temporalis, medial pterygoid, orbicularis oris, platysma, mylohyoid, genioglossus and hyoglossus. As in previous studies excellent results have been obtained in some, though often required successive injections into various muscle groups. In others the results have been disappointing. A retrospective review has therefore been carried out to determine the reasons for treatment failure. This has been due to the authors' inability to identify the primary muscle affected or to recognize concurrent involvement of several different muscle groups. Local muscle discomfort and palpable spasms were used early on to select treatment sites for botulinum toxin. Latterly the authors conducted comprehensive multiple EMG recordings both prior to initial treatment, and before succeeding injections. This current management strategy is significantly improving the results. 0 Refs.

Strategies to improve the response to BTX therapy in cranial dystonia.

Author: Ceballos-Baumann AD.

Source: Mov Disord 1995 May;10(3):368.

Type: Abstract

Abstract:

Full text of meeting report abstract: Blepharospasm: Presently there are two trends for choosing periorbital injection sites. One strategy involves multiple sites including injections close to the palpebral fissure and the other utilizes a fixed scheme with three or four injections directed medially and laterally into the lower and upper lid at the junction between the preseptal and orbital parts of the orbicularis oculi muscle. Controlled studies comparing both strategies have not been performed. However the difference in the incidence of side effects, especially ptosis, could be related to the different injection technique. Identification of pretarsal dystonia also described as akinesia or apraxia of eye opening, levator inhibition, atypical blepharospasm improves outcome and determines a different injection strategy with tarsal injections. Jaw closing dystonia: Straightforward injections into the masseter muscles may be sufficient in some patients, while in others injections into the temporalis and medial pterygoideal muscles improve the outcome. EMG guidance may be useful. Jaw opening dystonia: injections into the submental muscles carry a significant risk of severe dysphagia and improvement is marginal. Percutaneous injections into the lateral pterygoideal muscles may be functionally superior. Adductor spasmodic dysphonia: Treatment failures after percutaneous EMG guided laryngeal injections may benefit from a transoral approach which depending on the expertise of the injector allows a more precise placement of the injections into the vocal cords. Supported by BMFT OIKL92018. 0 Refs.

Hypertrophic branchial myopathy treated with botulinum toxin type A

Author: Doyle M and Jabbari B.

Source: Neurology 1994 Sep;44(9):1765-6.

Type: Case Report

Abstract:

Hypertrophic branchial myopathy (HBM) is a benign condition in which the muscles of mastication, usually the masseters but also the temporalis and pterygoid muscles, become insidiously enlarged. The authors report a case of HBM which they treated with botulinum toxin type A injections. Within one week of injection the patient reported improvement in symptoms, including a reduction in pain on mastication and alleviation of the chronic daily headache that had plagued her since onset of her symptoms. Two weeks after the initial (unilateral) injection, a decrease in the bulk of the treated temporalis muscle was evident. The patient returned after four months due to recurrence of local pain and tenderness. Each temporalis muscle was injected with fifteen units botulinum toxin and after one week, the patient reported marked reduction of local pain and improvement in jaw motility. Three months after these injections the patient was without symptoms. The authors note that the remarkable response of the patient to treatment with botulinum toxin introduces a new and effective treatment for some patients with focal painful, hypertrophic myopathies. 7 Refs.

Botulinum toxin A in the management disorders of the masticatory muscle system.

Author: McKellar G et al.

Source: Mov Disord 1994;9(Suppl1):87.

Type: Abstract

Abstract:

During the past three years in an open study the authors have used Botulinum toxin A in the management of oromandibular dystonia, the reduction of mandibular condylar fractures and intractable facial pain due to masticatory muscle spasm. Sixty-five patients have been comprehensively studied who comprised twenty-six males and thirty-nine females (age range 10-71 years, median age 40). The groups were: oromandibular dystonia, n=16, condylar fractures, n=20 and masticatory muscle spasm, n=29. Botulinum toxin A (Allergan 50 mouse units/ml) was injected directly into the muscle belly, under EMG control and the dose for each muscle was 15-20 mouse units. Dystonia patients showed a marked improvement. The (jaw) opening dystonias were best controlled by injecting anterior temporalis and lateral pterygoid muscles, while closing dystonias responded to partial paralysis of the masticatory elevatory muscles. The management of mandibular condylar fractures by paralyzing the lateral pterygoid muscle facilitated conservative and operational procedures, relieved pain in obtaining normal occlusion and stabilized the condylar position during union. The facial pain group have experienced relief of their symptoms from fifteen to thirty-six weeks and repeated injections have produced similar results. The use of Botulinum Toxin A has been found to be effective in the management of these disorders. 0 Refs.

Botulinum toxin in the management paradoxical activity of jaw muscles.

Author: Naumann Met at.

Source: Mov Disord 1994;9(Suppl1):148.

Type: Abstract

Abstract:

Full text of congress abstract: Paradoxical activity of jaw muscles (PAJM) during voluntary jaw opening results in the inability to open the mouth. Instead of being inhibited, jaw closing muscles (i.e. masseter temporal, and medial pterygoid muscles) are activated when the mouth is intended to be opened, thus antagonizing the initiated movement. This rare phenomenon has been described following vascular or traumatic lesions of the brainstem and so far has been difficult to manage. We report on a sixty-two year old patient who developed paradoxical activity of jaw opening muscles following basilar artery thrombosis with a mainly right sided brainstem infarction. Four months later he became progressively unable to eat due to immobilization of the jaws. Electromyographical examination revealed

coinnervation of antagonists (masseter and digastricus muscles) on the right side on attempt to open the mouth, while the innervation pattern of these muscles was regular on the left side. Mouth closure was normal. Paralysis of the right masseter and temporal muscles after local injection of Botulinum toxin restored his ability to open the mouth and accept food. Thus, treatment with Botulinum toxin is recommended as a simple, safe and effective method in cases of PAJM. 0 Refs.

Botulinum toxin treatment of hemimasticatory spasm in Parry Romberg syndrome.

Author: Trosch RM and Rontal M.

Source: Mov Ulsord 1~4;~(:suppl1):60.

Type: Abstract

Abstract:

The authors describe the pathology of progressive hemifacial atrophy (Parry Romberg syndrome), a common cause of painful hemimasticatory spasm (HMS) and describe one case. They report a forty-one year old physician with Parry Romberg syndrome manifesting right facial hemiatrophy, hyperpigmented linear localized scleroderma, hemimasticatory muscle hypertrophy and HMS. Painful, paroxysmal bruxism was triggered by speaking, eating or with forceful jaw closure and persisted in sleep. Absent were seizures, limb hemiatrophy, ophthalmic or other cranial nerve signs. Monopolar EMG recordings revealed brief, repetitive, irregular bursts of motor units discharges in the right masseter, temporalis and lateral pterygoid muscles. Facial and left-sided masticatory muscles were silent except during voluntary contractions. Prior trials of carbamazepine, phenytoin and diazepam failed. EMG guided botulinum toxin injection (masseter-50 U. temporalis-25U and lateral pterygoid 20U) provided relief from HMS lasting four months. Bruxism could no longer be triggered and ease of jaw opening markedly improved. No adverse effects were noted with the first or subsequent injections nor has efficacy diminished. Botulinum toxin injection appears to be a safe and effective treatment of HMS associated with Parry Romberg syndrome. (A videotaped demonstration was originally provided with the abstract, but is not available.) 0 Refs.