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REVIEW ARTICLE

Geniculate neuralgia: a systematic review

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Abstract

Objective: To systematically summarise the peer-reviewed literature relating to the aetiology, clinical presentation, investigation and treatment of geniculate neuralgia.

Data sources: Articles published in English between 1932 and 2012, identified using Medline, Embase and Cochrane databases.

Methods: The search terms 'geniculate neuralgia', 'nervus intermedius neuralgia', 'facial pain', 'otalgia' and 'neuralgia' were used to identify relevant papers.

Results: Fewer than 150 reported cases were published in English between 1932 and 2012. The aetiology of the condition remains unknown, and clinical presentation varies. Non-neuralgic causes of otalgia should always be excluded by a thorough clinical examination, audiological assessment and radiological investigations before making a diagnosis of geniculate neuralgia. Conservative medical treatment is always the first-line therapy. Surgical treatment should be offered if medical treatment fails. The two commonest surgical options are transection of the nervus intermedius, and microvascular decompression of the nerve at the nerve root entry zone of the brainstem. However, extracranial intratemporal division of the cutaneous branches of the facial nerve may offer a safer and similarly effective treatment.

Conclusion: The response to medical treatment for this condition varies between individuals. The long-term outcomes of surgery remain unknown because of limited data.

Key words: Geniculate Neuralgia; Nervus Intermedius Neuralgia; Neuralgia; Facial Neuralgia; Neuropathic Pain; Otalgia

Introduction

Geniculate neuralgia, also known as nervus intermedius neuralgia, is an extremely uncommon form of cranial nerve or facial neuralgia that is believed to originate from the nervus intermedius. According to the International Headache Society, it is defined as intermittent episodes of pain located deep in the ear that last for seconds or minutes and are often triggered by sensory or mechanical stimuli at the posterior wall of the auditory canal without any pathology.[1] Because of its extreme rarity, precise data regarding its prevalence are not available.

This study aimed to systematically review the existing literature related to the management of geniculate neuralgia.

Methods

A systematic search of articles published in English from 1932 to 2012 was carried out using PubMed, Embase, the Cumulative Index to Nursing and Allied Health Literature, and the Cochrane library. The search terms were: 'geniculate neuralgia', 'nervus intermedius neuralgia', 'facial pain', 'otalgia' and 'neuralgia'. All original articles related to geniculate neuralgia, including reviews, observational studies, case series and case reports, were included.

Two researchers examined the literature and excluded those papers not related to geniculate neuralgia. Reports referring to a treatment relevant, although not directly pertaining to, geniculate neuralgia were not excluded. Papers were then assessed with regard to the level of evidence, according to the Oxford Centre of Evidence-Based Medicine.

A total of 143 papers relating to the treatment of geniculate neuralgia were identified from the preliminary search. Of these, 125 were excluded because they were not directly relevant to the management of geniculate neuralgia. Therefore, 18 papers were included in the review.[2–19] All reports directly related to geniculate neuralgia were classified as level 4 evidence, consisting of single case reports or small case series. There were two level 1 systematic reviews, which discussed

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the medical therapy of neuralgia.[20–27] In total, there have been fewer than 150 cases of geniculate neuralgia reported to date.

Functional anatomy

The nervus intermedius, also named the intermediary nerve or Wrisberg's nerve, was identified by Professor Heinrich August Wrisberg from the University of Gottingen, Germany, in 1777.[28] It travels alongside the motor component of the facial nerve and is considered to be part of this nerve. It contains sensory and parasympathetic fibres derived from three distinct nuclei: (1) parasympathetic secretory fibres from the superior salivary nucleus; (2) special sensory fibres from the gustatory superior pole of the solitary nucleus; and (3) cutaneous sensory fibres from the dorsal part of the trigeminal tract.

The parasympathetic efferent fibres pass to the geniculate ganglion, with or without synapses at the ganglion, before innervating the sublingual and submandibular salivary glands, as well as the main and ancillary lacrimal glands, via the superior petrosal nerve. The special sensory afferent fibres bring impulses from the taste receptors located in the anterior two-thirds of the tongue, the floor of the mouth and part of the palate, via the chorda tympani, to the solitary nucleus. The cutaneous somatic afferent fibres bring impulses from the sensory receptors located: in the concha of the auricle, behind the ear, in part of the posterior wall of the external auditory canal and in the outer layer of the tympanic membrane.[29]

The nerve leaves the brainstem as a number of roots. According to Rhoton *et al*.[29] and Oh *et al*.[30] the number of roots varies from one to five and the number of the rootlets per root also varies from one to five. Rhoton *et al*.[29] divided the course of the nervus intermedius before it reaches the geniculate ganglion into three segments: the first segment adheres to the vestibulocochlear nerve at the nerve root; the second segment runs separately between the VIIth and VIIIth cranial nerves; and the third segment joins the motor root of the facial nerve within the internal acoustic meatus. These anatomical characteristics are important when transection of the nervus intermedius is being considered as a means of treatment for geniculate neuralgia. A detailed discussion of the anatomy of the chorda tympani and greater superficial petrosal nerves is beyond the remit of this paper.

The detailed anatomy of the branches of the nervus intermedius that provide cutaneous innervation is, however, important to the treatment of geniculate neuralgia. According to Eshraghi *et al*.[31] the sensory auricular branch of the facial nerve arises from the vertical segment of the facial nerve, between the second genu and the chorda tympani nerve origin. This auricular sensory nerve usually arcs laterally and courses inferiorly to supply the posterior and inferior external ear canal at the region of the osseous–cartilaginous junction, as well as the inferior portions of the pinna. Understanding the anatomy of the sensory auricular branches of the facial nerve is helpful when considering some of the surgical treatments for geniculate neuralgia.

Aetiology

In some cases, the aetiology may be vascular compression at the root entry zone of the VIIth and VIIIth cranial nerves. The anterior inferior cerebellar artery, the posterior inferior cerebellar artery and the branches of the vertebral arteries are the vascular structures likely to exert pressure upon this nerve complex. In cases where vascular compression does not exist, the aetiology of the pain remains unknown.

Clinical presentation

The clinical presentation of geniculate neuralgia is varied, although it classically manifests as acute paroxysmal stabbing pain centred within the ear. It may be localised in the auditory canal, pinna, retroauricular region or even the soft palate, and may sometimes radiate to the temporal region or the angle of the mandible. The pain may be triggered by sensory or mechanical stimuli, such as touching the posterior wall of the auditory canal. The pain may be associated with disorders of lacrimation, gustatory sensation and salivation.

Diagnosis and differential diagnosis

In order to diagnose geniculate neuralgia, all other possible non-neuralgic causes of otalgia must be eliminated. These include: otitis externa or media; malignancy of the pinna, external auditory canal, temporal bone or nasopharynx; lesions of dental origin; temporomandibular joint diseases; vascular lesions; referred pain from nasopharyngeal and laryngeal lesions; intracranial lesions in the cerebellopontine angle; and rare syndromes such as Eagle's syndrome. Therefore, thorough neurological, otological, dental and other ENT examinations must be performed.

It is also important to differentiate geniculate neuralgia from other forms of neuralgia. The sensation of the ear is supplied by the Vth, VIIth, IXth and Xth cranial nerves, and the IInd and IIIrd cervical nerves, and it is not uncommon to see an overlap of other facial neuralgias. The two commonest differential diagnoses of geniculate neuralgia are trigeminal neuralgia and glossopharyngeal neuralgia. Characteristics of the pain in both are similar to those of geniculate neuralgia. Only the location and distribution of the pain enable differentiation between them. Trigeminal neuralgia is by far the most frequent facial neuralgia, with the pain typically located in the maxillary (V_2) and mandibular (V_3) nerve distributions. The pain can occur spontaneously or can be precipitated by sensory stimulation of certain areas in the face, usually coinciding with pain localisation. Touching or washing the face, shaving, brushing the teeth, and chewing are all considered to be typical triggers. An underlying cause may be

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identified in 15 per cent of patients presenting with trigeminal neuralgia, with the commonest being compression of the trigeminal nerve root by an aberrant loop of a blood vessel, usually within a few millimetres of the root entry point into the pons.

Glossopharyngeal neuralgia is characterised by paroxysmal neuralgia pain attacks localised in the throat near the base of tongue, soft palate and tonsillar fossa. It can radiate to the angle of the mandible and rarely into the external auditory canal or the neck. The pain is often triggered by swallowing, chewing, talking, yawning, laughing or coughing. Most cases are idiopathic.[32] Sequential local nerve blockade using local anaesthesia may help to identify the nerve origin.[31,33]

The most important investigation when assessing a patient with geniculate neuralgia is fine-cut magnetic resonance imaging of the cerebellopontine angle. This may identify a vascular loop compressing the nervus intermedius, although it is not 100 per cent sensitive. However, it may have poor specificity because a significant proportion of asymptomatic individuals also have a vascular loop compressing the nerve.[8,34–36] Other investigations that may help to exclude other causes of otalgia include pure tone audiometry, auditory evoked brainstem responses, vestibular function tests and fine-cut computed tomography.

Treatment

The first-line treatment for geniculate neuralgia is medical therapy, with surgery held in reserve for those patients who do not respond to medication. No studies comparing different treatment modalities have been reported.

Medical therapy

Medical therapy is routinely used in the treatment of neuralgia, with varying degrees of success. A large number of papers have been published on this subject.[29–36] There are, however, no reports specifically related to the medical therapy of geniculate neuralgia. A full review of the literature related to the medical therapy of neuralgia in general is beyond the scope of this review, but a brief summary is outlined below.

Commonly used medications include anticonvulsants such as carbamazepine, gabapentin and lamotrigine, and tricyclic antidepressants such as amitriptyline.

Carbamazepine is the medication most commonly prescribed for the treatment of neuralgia. It often gives a good initial response.[20] Overall, about 70 per cent of patients achieve some relief of chronic neuropathic pain.[21] Carbamazepine stabilises the inactivate state of synaptic voltage-gated sodium channels, thus making fewer channels available for subsequent opening. This blocks synaptic transmission and reduces the activation of afferent pain fibres. The usual maintenance dosage is in the range of 200–800 mg per day, depending on the individual's response and tolerability.[22] Common side effects include drowsiness, diplopia, ataxia and hyponatraemia. Uncommon but serious adverse effects include allergic rash, myelosuppression, hepatotoxicity, lymphadenopathy, systemic lupus erythematosus, Steven–Johnson syndrome and aplastic anaemia.[23]

Gabapentin acts on the voltage-gated calcium channels of cortical neurones and reduces axonal excitability. The effective maintenance dosage is in the range of 900–3600 mg per day, depending on the individual's response and tolerability.[20] It provides a high level of pain relief in about a third of patients with severe neuropathic pain.[24] Common side effects are dizziness, coordination problems, fatigue and nystagmus.

Lamotrigine blocks presynaptic sodium channels and thus inhibits the release of glutamate and aspartate.[25] The usual dosage is 200–400 mg per day, depending on the individual's response and tolerability. An article in the *Cochrane Database of Systematic Reviews* of 2011 found that lamotrigine lacks efficacy against chronic neuropathic pain.[21] However, it is still used occasionally. Common side effects are ataxia, constipation, vomiting and rashes.

Amitriptyline blocks serotonin and noradrenaline reuptake in the central nervous system, hence increasing the activity of the descending inhibitory pain pathways.[26] The dose is generally between 25 and 125 mg per day, depending on the individual's response and tolerability. Whilst it has historically been the first-line treatment for neuropathic pain, an article in the *Cochrane Database of Systematic Reviews* of 2012 indicates that only a minority of patients achieve satisfactory pain relief.[27] Common side effects are anticholinergic effects, cardiovascular effects, drowsiness, ataxia and tremor.

As these drugs have differing sites of action, combinations of drugs from different groups may benefit patients who are resistant to standard regimens of single drug therapy. It is important to note, however, that medical treatments may eventually fail because of intolerability to the adverse effects and decreasing drug effectiveness after prolonged use.

An alternative to medical therapy is regional nerve blockade performed using local anaesthesia. A number of investigations into the use of regional nerve blockade in treating neuralgic pain in general have been reported, although none specifically refer to geniculate neuralgia. A systematic review was recently carried out by Vlassakov *et al.*[37] These authors found that although the quality of the evidence for the therapeutic efficacy of this technique was poor, there were strong indications that it was effective even beyond the period of conduction blockade.

Surgery

Surgery may be considered when medical therapy or local nerve blockade fail to produce adequate analgesia. A number of surgical procedures have been

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described for the management of patients with geniculate neuralgia. The most common surgical procedures are transection of the nervus intermedius, intracranially and/or at the geniculate ganglion, and microvascular decompression of the nervus intermedius at its root entry zone to the brainstem if compression by a vascular loop is present. The quality of the evidence base for these two procedures is poor. Nevertheless, the literature suggests that they are effective in patients for whom medical therapy has failed. It is unclear whether nerve division or microvascular decompression alone is effective. The relevant papers are summarised below.[2,3]

Pulec[2,3] described the surgical procedure for excision of the nervus intermedius and the geniculate ganglion via a middle cranial fossa approach. Using this approach, he exposed the motor component of the facial nerve, geniculate ganglion, greater superficial petrosal nerve and nervus intermedius from the cochleariformis process to the cerebellopontine angle. As the second segment of the nervus intermedius runs between the superior vestibular nerve and the facial nerve at the internal auditory canal, the internal auditory canal can be opened and a 5 mm segment of the nervus intermedius can be excised (before it joins the facial nerve). The geniculate ganglion can then be excised from the internal genu of the facial nerve, together with a 5 mm segment of the greater superficial petrosal nerve. Pulac suggested that 30 per cent of the anterior portion of the internal genu (the motor portion of the facial nerve) can be excised along with the ganglion without causing permanent facial paralysis. He treated 64 patients with this surgical technique between 1966 and 1996. He reported that all of the patients experienced excellent relief of ear pain, except for one with Lyme disease. His surgical procedures did not have complications of hearing loss, cerebrospinal fluid leak or permanent facial paralysis. However, all patients had permanent non-lacrimating ipsilateral eyes after the surgery.[3]

Rupa *et al*.[4] published a review of their experience in the surgical management of primary otalgia. They performed multiple nerve transections and microvascular decompression via a middle cranial fossa approach, a posterior cranial fossa approach or a combined approach in 18 patients. Based on clinical diagnosis, the nerves were sectioned either singly or in combination as follows: nervus intermedius (14 patients), geniculate ganglion (10 patients), IXth nerve (14 patients), Xth nerve (11 patients), tympanic nerve (4 patients) and chorda tympanic nerve (1 patient). Nine patients had microvascular decompression of the involved nerves. The authors reported an overall success rate in providing pain relief in 72.2 per cent of cases and post-operative complications in 33.3 per cent. Complications included cerebrospinal fluid leak (one patient), aseptic meningitis (one patient), sensorineural hearing loss (two patients), vertigo (one patient) and transient facial paresis (one patient).

Lovely and Jannetta[5] performed retromastoid craniectomies with microvascular decompression of the Vth, IXth and Xth cranial nerves with sectioning of the nervus intermedius. Their study comprised 14 patients with geniculate neuralgia who had failed conservative treatment. The authors reported long-term relief of ear pain in 90 per cent of patients. Surgical complications included transient facial paresis (one patient), facial numbness (one patient), paresis of the IXth and Xth cranial nerves (one patient), chemical meningitis (one patient), cerebrospinal fluid leak (two patients) and wound infection (one patient).

Sachs[6] reported a case series of four patients with geniculate neuralgia who had undergone nervus intermedius transection and were pain free after surgery. However, the patients did experience some degree of decreased lacrimation and taste sensation.

Tubbs *et al*.[7] reported the case of a patient with geniculate neuralgia in whom medical treatment failed. Magnetic resonance imaging revealed the posterior inferior cerebellar artery loop impinging on the root entry zone of the nerve intermedius–vestibulocochlear nerve complex. The patient then underwent a left-sided retromastoid craniotomy with transection of the nervus intermedius, and microvascular decompression of the lower cranial nerve at the level of the brainstem using a polytetrafluoroethylene implant. He remained pain free after surgery without complication.

Saers *et al*.[8] reported another patient with geniculate neuralgia for whom medical treatment was unsuccessful. Magnetic resonance imaging showed compression of the nervus intermedius by the anterior inferior cerebellar artery. Arachnoidal connective tissue tethering the vessel to the nervus intermedius was removed intra-operatively. Microvascular decompression of the compressed nervus intermedius was performed with Gelita-Spon® and Tissucol Duo 500® haemostatic materials. This patient remained pain free after surgery, without complication.

Sakas *et al*.,[9] Bellotti *et al*.,[10] Younes *et al*.[11] and Ozer *et al*.[12] reported similar cases of geniculate neuralgia with suspected compression of nervus intermedius by the anterior inferior cerebellar artery. All patients underwent microvascular decompression of the compressed nervus intermedius, with long-term resolution of pain.

An alternative surgical strategy described by Ulubil *et al*.[33] is division of the sensory auricular branch of the facial nerve. According to Eshraghi *et al*.[31] this can be done using a standard postauricular cortical mastoidectomy. These reports described three cases in which the vertical segment of the facial nerve was widely skeletonised and the chorda tympani was identified and preserved. The sensory branches of the facial nerve, as it passed towards the posterior canal wall, were identified and transected, with a length of at least 5 mm of the nerve being removed. This was followed by elevation of the posterior and inferior external ear canal skin in order to divide any sensory nerves

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passing through the bone to the canal skin. All three patients remained symptom free for up to a year after surgery and none experienced complications. There are no other reports of this technique in the literature, but the extracranial nature of this type of surgery reduces the potential morbidity of treatment compared to intracranial procedures, although there is a small risk of facial weakness.

Conclusion

Geniculate neuralgia is a rare form of cranial nerve neuralgia originating from the nervus intermedius that presents considerable diagnostic and therapeutic challenges. Non-neuralgic causes of otalgia should be excluded before making a diagnosis through clinical examination, audiological assessment and radiological investigations. The latter may help to identify a vascular loop that may be compressing the nervus intermedius. Medical treatment is always the first-line therapy. Combinations of different groups of drug may be of value to patients resistant to the standard regimens of single drug therapy. Regional nerve blockade may also be helpful and could be effective well after the initial conduction blockade has passed. If medical treatment fails, then surgery may be considered. Systematic regional nerve blockade may help to identify the nerve or nerves responsible for the neuralgia. The two most widely described surgical options are: transection of the nervus intermedius and the geniculate ganglion; and microvascular decompression of the nerve at its root entry zone, if vascular compression exists. The literature suggests that these procedures are effective, but the evidence base is poor and it remains unclear whether these two procedures are effective when performed individually. The influence of the division of other cranial nerves, which is often performed simultaneously, also makes interpretation of the current literature difficult. Extracranial intratemporal division of the cutaneous branches of the facial nerve may offer a safer treatment with similar effectiveness.

Based on our analysis of the current literature, when faced with failed medical therapy, we recommend an initial intratemporal division of the cutaneous branches of the facial nerve. If this is unsuccessful, resection of the nervus intermedius is advised; simultaneous microvascular decompression should be performed if there is also vascular compression.

Because of small sample sizes, relatively short follow up and poor study design, further research is required to determine the effectiveness of the intratemporal approach and to assess whether microvascular decompression alone or nervus intermedius division alone are effective therapies.

References

1 Headache Classification Subcommittee of the International Headache Society. The International Classification of Headache Disorders: 2nd edition. *Cephalagia* 2004;**24**:9–160
2 Pulec JL. Geniculate neuralgia: diagnosis and surgical management. *Laryngoscope* 1976;**86**:955–64
3 Pulec JL. Geniculate neuralgia: long-term results of surgical treatment. *Ear Nose Throat J* 2002;**81**:30–3
4 Rupa V, Saunders RL, Weider DJ. Geniculate neuralgia: the surgical management of primary otalgia. *J Neurosurg* 1991;**75**: 505–11
5 Lovely TJ, Jannetta PJ. Surgical management of geniculate neuralgia. *Am J Otol* 1997;**18**:512–17
6 Sachs E Jr. The role of the nervus intermedius in facial neuralgia. Report of four cases with observations on the pathways for taste, lacrimation, and pain in the face. *J Neurosurg* 1968;**28**:54–60
7 Tubbs RS, Mosier KM, Cohen-Gadol AA. Geniculate neuralgia: clinical, radiologic, and intraoperative correlates. *World Neurosurg* 2013;**80**:e353–7
8 Saers SJ, Han KS, De Ru JA. Microvascular decompression may be an effective treatment for nervus intermedius neuralgia. *J Laryngol Otol* 2011;**125**:520–2
9 Sakas DE, Panourias IG, Stranjalis G, Stefanatou MP, Maratheftis N, Bontozoglou N. Paroxysmal otalgia due to compression of the intermediate nerve: a distinct syndrome of neurovascular conflict confirmed by neuroimaging. Case report. *J Neurosurg* 2007;**107**:1228–30
10 Bellotti C, Medina M, Oliveri G, Ettorre F, Barrale S, Sturiale C *et al*. Neuralgia of the intermediate nerve combined with trigeminal neuralgia: case report. *Acta Neurochir (Wien)* 1988;**91**: 142–3
11 Younes WM, Capelle HH, Krauss JK. Microvascular decompression of the anterior inferior cerebellar artery for intermediate nerve neuralgia. *Stereotact Funct Neurosurg* 2010;**88**:193–5
12 Ozer FD, Duransoy YK, Camlar M. Atypic geniculate neuralgia: atypic anatomic correlation of cranial nerve roots and AICA. *Acta Neurochir (Wien)* 2009;**151**:1003–4
13 Furuya Y, Ryu H, Uemura K, Sugiyama K, Isoda H, Hasegawa S *et al*. MRI of intracranial neurovascular compression. *J Comput Assist Tomogr* 1992;**16**:503–5
14 Cooper DW, Cavicke DC. Primary juvenile otalgia. Report of a case with surgical cure. *J Neurosurg* 1963;**20**:895–8
15 Furlow LT. Tic douloureux of the nervus intermedius (so-called idiopathic geniculate neuralgia). *JAMA* 1942;**119**:255–9
16 Hunt JR. Geniculate neuralgia (neuralgia of the nervus facialis). A further contribution to the sensory system of the facial nerve and its neuralgic conditions. *Arch Neurol Psychiat* 1937;**37**: 253–85
17 Quaknine GE, Robert F, Molina-Negro P, Hardy J. Geniculate neuralgia and audio-vestibular disturbance due to compression of the intermediate and eighth nerves by the postero-inferior cerebellar artery. *Surg Neurol* 1980;**13**:147–50
18 Wilson AA. Geniculate neuralgia. Report of a case relieved by intracranial section of the nerve of Wrisberg. *J Neurosurg* 1950;**7**:473–81
19 Yeh HS, Tew JM Jr. Tic convulsif, the combination of geniculate neuralgia and hemifacial spasm relieved by vascular decompression. *Neurology* 1984;**34**:682–4
20 McQuay HJ, Carroll D, Jadad AR, Wiffen P, Moore A. Anticonvulsant drugs for management of pain: a systemic review. *BMJ* 1995;**311**:1047–52
21 Wiffen PJ, Derry S, Moore RA, McQuay HJ. Carbamazepine for acute and chronic pain in adults. *Cochrane Database Syst Rev* 2011;(**1**):CD005451
22 Centre for Clinical Practice at NICE. *Neuropathic Pain. The Pharmacological Management of Neuropathic Pain in Adults in Non-specialist Settings. NICE Clinical Guideline 96*. Centre for Clinical Practice at NICE (UK). London: National Institute for Health and Clinical Excellence (UK), 2010
23 Moore RA, Wiffen PJ, Derry S, McQuay HJ. Gabapentin for chronic neuropathic pain and fibromyalgia in adults. *Cochrane Database Syst Rev* 2011;(**16**):CD007938
24 Allen S. Pharmacotherapy of neuropathic pain. *Contin Educ Anaesth Crit Care Pain* 2005;**5**:134–7
25 Wiffen PJ, Derry S, Moore RA. Lamotrigine for acute and chronic pain. *Cochrane Database Syst Rev* 2011;(**16**): CD006044
26 Dworkin RH, Backonja M, Rowbotham MC, Allen RR, Argoff CR, Bennett GJ *et al*. Advances in neuropathic pain: diagnosis, mechanisms and treatment recommendations. *Arch Neurol* 2003;**60**:1524–34
27 Moore RA, Derry S, Aldington D, Cole P, Wiffen PJ. Amitriptyline for neuropathic pain and fibromyalgia in adults. *Cochrane Database Syst Rev* 2012;(**12**):CD008242

https://doi.org/10.1017/S0022215114000802 Published online by Cambridge University Press

28 Alferi A, Strauss C, Prell J, Peschke E. History of the nervus intermedius of Wrisberg. *Ann Anat* 2010;**192**:139–44
29 Rhoton AL Jr, Kobayashi S, Hollinshead WH. Nervus intermedius. *J Neurosurg* 1968;**29**:609–18
30 Oh CS, Chung IH, Lee KS, Tanaka S. Morphological study on the rootlets comprising the root of the intermediate nerve. *Anat Sci Int* 2003;**78**:111–13
31 Eshraghi AA, Buchman CA, Telischi FF. Sensory auricular branch of the facial nerve. *Otol Neurotol* 2002;**23**:393–6
32 Siccoli MM, Bassetti CL, Sandor PS. Facial pain: clinical differential diagnosis. *Lancet Neurol* 2006;**5**:257–67
33 Ulubil SA, Eshraghi AA, Telischi FF. Sectioning the sensory auricular branch of the facial nerve to treat recalcitrant otalgia. *Otol Neurotol* 2009;**30**:522–4
34 Yap L, Pothula VB, Lesser T. Microvascular decompression of the cochleovestibular nerve. *Eur Arch Otorhinolaryngol* 2008; **265**:861–9
35 Adler CH, Bernardi B, Bosley TM, Savino PJ, Sergott RC, Zimmerman RA. Hemifacial spasm: evaluation by magnetic resonance imaging and magnetic resonance tomographic angiography. *Ann Neurol* 1992;**32**:502–6
36 Bernardi B, Adler C, Savino PJ, Zimmerman RA. Magnetic resonance tomographic angiography in the investigation of hemifacial spasm. *Neuroradiology* 1993;**35**:606–11
37 Vlassakov KV, Narang S, Kissin I. Local anesthetic blockade of peripheral nerves for treatment of neuralgias: systematic analysis. *Anesth Analg* 2011;**112**:148–93

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