ORIGINAL RESEARCH

Computed tomographic features of the proximal petrous facial nerve canal in recurrent Bell's palsy

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Abstract

Objectives: The primary objective was to determine whether the narrowest dimensions of the labyrinthine facial nerve (LFN) canal on the symptomatic side in patients with unilateral recurrent Bell's palsy (BP) differ from those on the contralateral side or in asymptomatic, age- and gender-matched controls on computed tomography (CT). The secondary objectives were to assess the extent of bony covering at the geniculate ganglion and to record inter-observer reliability of the CT measurements.

Methods: The dimensions of the LFN canal at its narrowest point perpendicular to the long axis and the extent of bony covering at the geniculate ganglion were assessed by two radiologists. Statistical analysis was performed using the Wilcoxon signed-rank and Mann-Whitney U tests (LFN canal dimensions) and the Chi-squared test (bony covering at the geniculate ganglion). Inter-observer reliability was evaluated using Intra-Class Correlation (ICC) and Cohen's kappa.

Results: The study included 21 patients with unilateral recurrent BP and 21 asymptomatic controls. There was no significant difference in the narrowest dimensions of the ipsilateral LFN canal when compared to the contralateral side or controls ($P = .43$-$.94$). Similarly, there was no significant difference in the extent of bony covering at the geniculate ganglion when compared to either group ($P = .19$-$.8$). Good inter-observer reliability was observed for LFN measurements (ICC = 0.75-0.88) but not for the bony covering at the geniculate ganglion (Cohen's kappa = 0.53).

Conclusion: The narrowest dimensions of the LFN canal and the extent of bony covering at the geniculate ganglion do not differ in unilateral recurrent BP, casting doubt over their etiological significance.

Level of Evidence: Level IV.

KEYWORDS
Bell's palsy, computed tomography, facial nerve canal, geniculate ganglion

1 | INTRODUCTION

Bell's palsy (BP) is the commonest cause of facial paralysis. While it is currently idiopathic by definition, virally mediated inflammation is widely accepted as the foremost etiological factor.[1,2] It has also been suggested that the anatomy of the facial nerve canal might play an important causative role. In particular, neural inflammation within the narrow portions of the facial nerve canal is presumed to result in neural impingement.[1-4] This is most relevant at the labyrinthine portion and its meatal foramen, where the facial nerve canal is at its narrowest.[2,4] This "compressive theory," however, remains controversial, with contradictory findings reported by a number of radiological studies on labyrinthine facial nerve (LFN) canal dimensions and BP.[5-7] Potential reasons for the discrepancies include the lack of comparison with healthy controls, use of single axial planes for measurement and absence of inter-observer reliability statistics.

In addition to the LFN canal dimensions, other anatomical variations have been proposed as potential predisposing factors for BP, such as shortening of the groove for the greater superficial petrosal nerve (GSPN) and deficient bony covering at the geniculate ganglion. It has been speculated that such anatomical variants may make the facial nerve more vulnerable through closer proximity to the trigeminal nerve and thus easier transmission of virally mediated neural inflammation.[8]

The treatment options for BP include oral corticosteroids, antivirals, combined steroid-anti-viral therapy and surgical decompression. Whilst over 70% of patients spontaneously recover normal facial expression without any specific treatment,[9] the efficacy of oral corticosteroids in improving the chance of complete recovery of facial motor function is well established, especially when commenced within 72 hours of onset.[10,11] The Cochrane review from 2016 concluded that the number needed to treat with corticosteroids to avoid one incomplete recovery was 10.[11] The benefit of antivirals is less certain; there is now growing evidence that antiviral therapy alone does not improve recovery,[12,13] but combined anti-viral and corticosteroid therapy may potentially confer additional benefit compared to corticosteroids alone, in terms of complete recovery and/or reduction in other long-term sequelae such as motor synkinesis and gustatory hyperlacrimation.[13,14]

Decompressive surgery may be considered in some patients with poor prognostic features on electroneurographic and electromyographic testing, or with recurrent BP and incomplete recovery between episodes. Since the first description by Ballance and Dual in 1932,[15] controversies surrounding the role of surgery in BP have never ceased.[7,16,17] Furthermore, the proposed site of decompression, surgical approach and the rationale behind the recommendation have continued to evolve, going from the distal or entire portion of the mastoid segment, the stylomastoid foramen itself, the tympanic segment, then the geniculate ganglion through to the labyrinthine segment.[18,19] Based on the current theory of the LFN canal being the main etiological site, a middle cranial fossa approach is usually advocated.[16,20-24] In this context, it may be hypothesized that patients with unilateral recurrent BP might be more likely to demonstrate predisposing anatomical features, given their greater susceptibility.

Our primary objective was to determine whether the narrowest dimensions of the LFN canal in patients with recurrent unilateral BP differed from those of the contralateral LFN canal or in unaffected age-matched controls, when measured on computed tomography (CT). The secondary objectives included assessment of the degree of osseous covering of the geniculate ganglion and evaluation of inter-observer reliability for the two radiological measurements.

2 | MATERIALS AND METHODS

2.1 | Ethical considerations

The study was reviewed by the local institutional board and approved.

2.2 | Participants

The institutional radiology information system (RIS) was interrogated for temporal bone CT studies associated with digital request cards containing relevant terms ("facial," "palsy," "bell's," "bell," "recurrent," "seventh") between January 2008 and November 2018. Full clinical information was then collected from the Electronic Patient Record (EPR). Patients were included in the study if there were two or more documented episodes of unilateral recurrent BP and if no alternative etiology had been diagnosed on dedicated facial nerve protocol magnetic resonance imaging (MRI).

For the control group, age and gender matched subjects were identified from RIS by obtaining a sample of consecutive temporal bone CT studies carried out over an 18-month period between 2013 and 2015. Exclusion criteria included CT findings of cholesteatoma, significant middle ear soft tissue, evidence of skull base dysplasia or bone metabolic disorders, or a history of facial nerve palsy.

2.3 | CT technique

CT was performed on a variety of systems from Philips Medical Systems (Eindhoven, Netherlands) and Siemens Healthcare (Erlangen, Germany) (Table 1). Single collimation width ranged 0.6-0.8 mm, with an overlap of 0.3-1 mm; kVp ranged 120-140, mA ranged 90-244 and field of view 180-200 mm. Bone convolution algorithm data was available and analyzed in all cases.

2.4 | CT analysis

2.4.1 | LFN canal dimensions

Double oblique reformats of the LFN canal perpendicular to its long axis (Figure 1A-C) were created. Two radiologists (2.5 and 7 years of subspecialty head and neck radiology experience) carried out separate measurements of the LFN canal dimensions and assessments of the

TABLE 1 CT scanning systems

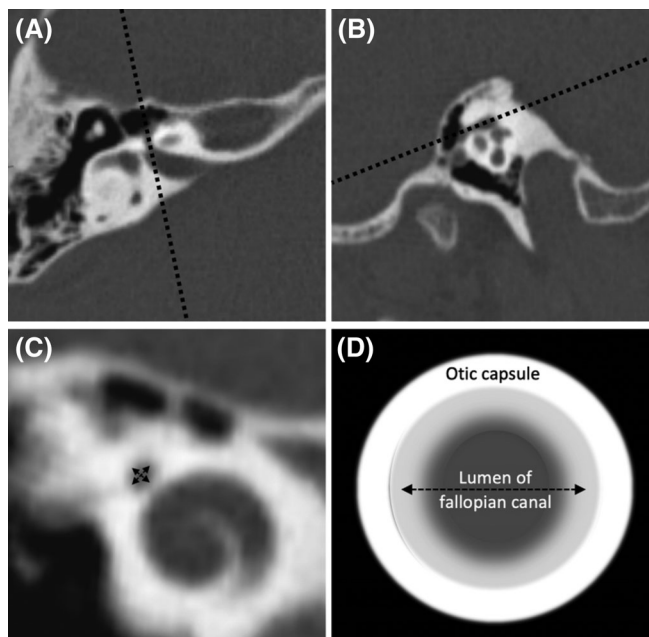| Manufacturer	Philips Philips Medical Systems, Eindhoven, Netherlands	Siemens Siemens Healthcare AG, Erlangen, Germany
Model	MX8000 (n = 6; 14%)	SOMATOM Definition Edge (n = 2; 5%)
	Brilliance 16P (n = 13; 31%)	SOMATOM Force (n = 4; 10%)
	Brilliance 40 (n = 11; 26%)	
	iCT 265 (n = 6; 14%)	

FIGURE 1 Measuring technique using double oblique technique along the line of the facial nerve canal in the axial (A) and sagittal (B) planes to produce a coronal section (C) perpendicular to the axis of the nerve canal (long and short axial dimensions indicated by arrows) (D) Measurements were performed by placing callipers within the intermediate density penumbra, halfway between the low density of the facial canal lumen and high density of the bone of the otic capsule. Measurement was carried out using a set magnification factor
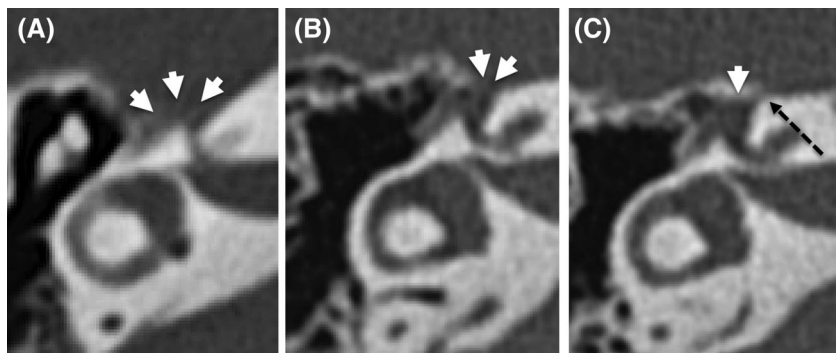
bony covering at the geniculate ganglion. Both radiologists were blinded to the clinical details and analyzed the images independently. Standard bone window settings and agreed magnification settings were employed. The location of the narrowest portion of the LFN was defined visually using all three planes (oblique sagittal, oblique coronal and oblique axial) and its location was recorded. The detailed description of the measurement technique is provided in Figure 1. The mean of the two observers' LFN measurements was used for statistical analysis.

2.4.2 | Bony covering at the geniculate ganglion

In each case, the extent of bony covering was graded as "complete" (the bony canal at the route of the GSPN is clearly seen), "partial" (the bony deficiency at the geniculate ganglion is the same as, or smaller than, the facial nerve canal) or "absent" (the bony deficiency at the geniculate ganglion is wider than the facial nerve canal) (Figure 2). Where there was disagreement between the observers, consensus scores were used.

2.5 | Statistical analysis

Statistical analysis was performed using SPSS Statistics 25.0 (IBM). The Shapiro-Wilkes test was used to assess the normality of data. The measurements of LFN canal dimensions were compared using the Wilcoxon signed-rank test (paired data) and Mann-Whitney U test (unpaired data). For the degree of bony covering of the geniculate ganglion, the Chi squared statistic was used. A P-value of <.05 was deemed statistically significant. Inter-observer reliability was assessed using the intraclass correlation coefficient (ICC)[25] for continuous data (LFN canal dimensions) and Cohen's kappa statistic[26] for categorical data (bony covering at the geniculate ganglion).

3 | RESULTS

3.1 | Participant demographics and characteristics

The initial database search identified 145 cases. Of these, 21 cases subsequently met the inclusion criteria (Figure 3).

FIGURE 2 Double oblique axial reconstructions through the geniculate ganglion demonstrating examples of an absent, A, partial, B, and complete, C, bony coverings. Of note, in the setting of complete bony covering of the geniculate ganglion, C, the canal for the GSPN is clearly delineated
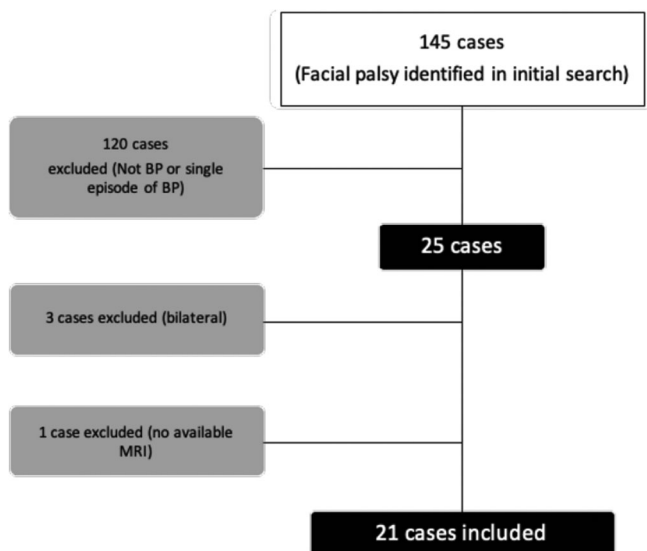
FIGURE 3 Flow diagram illustrating inclusion and exclusion criteria

TABLE 2 Characteristics of recurrent BP episodes

Laterality	Number of BP episodes	Recovery
Left: (n = 11), 52%	2 (n = 11), 52%	Complete: n = 7
	3 (n = 3), 14%	
	4 (n = 2), 19%	Incomplete: n = 11
Right: (n = 10), 48%	5 (n = 2), 10%	
	6 (n = 1), 5%	Unknown: n = 3
	Unknown but documented ≥2 (n = 2), 10%	

The study cohort included 12 females and 9 males, with a mean age of 37.9 ± 14.9 years (range 12.0-67.4 years). An equal number of age- and gender-matched controls were identified, with a mean age of 38.3 ± 14.6 years (range 12.1-65.6 years). Laterality, number of BP episodes and extent of recovery between episodes are summarized in Table 2.

3.2 | LFN canal dimensions

In all cases, the narrowest segment of the LFN canal was at or immediately lateral to the meatal foramen. On the symptomatic sides, long axis measurements through the narrowest portions ranged from 0.75 to 2 mm (median 1.05 mm, interquartile range 0.3 mm) and short axis dimensions ranged from 0.5 to 1.4 mm (median 0.75 mm, interquartile range: 0.1 mm). There was no statistically significant difference in the long and short axis dimensions of the LFN canal between the affected ipsilateral side in patients with unilateral recurrent BP and the asymptomatic contralateral side (P = .43-.67) or matched controls (P = .46-

.94). There was good inter-observer reliability for the LFN canal measurements (ICC = 0.75-0.88) (Table 3).

3.3 | Bony covering at the geniculate ganglion

The bony covering at the geniculate ganglion was either absent or partial in 38.0% of the symptomatic ipsilateral sides compared to 47.6% of asymptomatic contralateral sides and 57.1% of matched control cases. The differences were not statistically significant (P = .80 and P = .19, respectively). There was moderate inter-observer reliability (Cohen's kappa = 0.53). The details are summarized in Table 4.

4 | DISCUSSION

The current study showed no significant difference in the long and short axis dimensions of the LFN canal on the ipsilateral (symptomatic) side in patients with recurrent BP when compared to the contralateral (asymptomatic) side, or age-matched asymptomatic controls. In addition, there was no significant difference in the rates of complete, partial or absent bony covering at the geniculate ganglion.

Although considered idiopathic and a diagnosis of exclusion, a number of factors have been proposed for the potential underlying pathophysiology for BP, including anatomical variants, viral infection, ischemia, inflammation, autoimmunity and environmental conditions.[27,28] These hypotheses can by broadly categorized into two groups; either patients develop recurrent BP due to recurrent episodes of pathological inflammation within an anatomically normal bony canal or they are susceptible to episodes of BP due to a narrow LFN canal. Surgical reports have recorded neural edema and swelling in cases of BP,[2,4] which are supported by findings of increased enhancement (particularly at the geniculate ganglion and along the labyrinthine segment) on MRI.[29] The second, "compressive theory," suggests that swelling of the nerve within the bony canal leads to neural entrapment, increased endoneural pressure and vascular congestion, ischemia, impairment of axoplasmic flow, axonal degeneration and then necrosis.[7,27,30,31]

As for the neural entrapment theory, the most likely location is at the labyrinthine segment of the facial nerve canal and its meatus. The meatal foramen is the narrowest portion of the entire canal and the findings in our study (range of 0.5 mm to 1.4 mm) is in keeping with a mean diameter of 0.68 mm that has been reported in a previous study.[2] In addition, the proximal facial nerve is considered to be proportionally thicker and a dense arachnoid band also encircles the nerve here, which may further contribute to the constriction at this site.[2,32] Surgical decompression of this segment of the facial nerve canal may therefore be considered as a potential treatment option for BP in certain circumstances.[7]

A series of CT based studies, as summarized in Table 5, have argued in favor of this "compressive theory" by demonstrating narrower meatal or mid-labyrinthine segments of the facial nerve canal on the symptomatic

TABLE 3 Statistical evaluation of LFN canal dimensions and inter-observer reliability

observer 1		Median (mm)	IQR	Minimum (mm)	Maximum (mm)	Wilcoxon-signed rank (W)	P-value
Long axis	Symptomatic side	1.05	0.3	0.75	2.0	434.50	.67
	Asymptomatic side	1.05	0.35	0.8	1.1		
Short axis	Symptomatic side	0.75	0.15	0.5	1.4	420.50	.43
	Asymptomatic side	0.75	0.15	0.5	1.4		
observer 2		**Median (mm)**	**IQR**	**Minimum (mm)**	**Maximum (mm)**	**Mann-Whitney U**	**P-value**
Long axis	Symptomatic side	1.05	0.3	0.75	2.0	435.50	.94
	Matched controls	0.95	0.35	0.9	1.8		
Short axis	Symptomatic side	0.75	0.15	0.5	1.4	391.00	.46
	Matched controls	0.7	0.15	0.5	1.2		
Interrater reliability (ICC of the mean measurements)[a]							
Long axis	0.88						
Short axis	0.75						

[a]For ICC, values between 0.75 and 0.88 indicate good inter-observer reliability.[25]

TABLE 4 Bony covering at the geniculate ganglion

	Absent covering	Partial covering	Complete covering	Chi square statistic	P-value
Cases	2 (9.5%)	6 (28.6%)	13 (61.9%)		
Controls (contralateral sides)	3 (14.3%)	7 (33.3%)	11 (52.4%)	0.45	.80
Controls (asymptomatic age-matched)	12 (28.6%)	12 (28.6%)	18 (42.9%)	3.32	.19
Interrater reliability					
Cohen's Kappa	0.53				

Note: For Cohen's Kappa, values between 0.41 and 0.60 indicate moderate inter-observer reliability.[26]

side of patients with BP.[30,33-35] Furthermore, Hervechon et al suggested an association between narrower labyrinthine facial nerve caliber and poorer prognosis with respect to recovery.[36]

However, such findings have been disputed by other groups. For example, a postmortem study by Vianna et al found no significant difference in the mean diameters of the LFN canal between the affected side in BP and the controls.[31] In addition, two CT based studies found no significant difference in the size of the affected facial nerve canals compared to the controls.[37,38]

Given the lack of consensus in the literature and advances in CT technology that enable increasingly detailed study of the intratemporal facial nerve canal,[29,30,33,35,39,40] our study aimed to address the potential methodological limitations of previous CT based studies. In particular, our study utilized multiplanar reconstruction along the course of the LFN canal, which accounts for its obliquity relative to the scan plane. Such reformatting has been adopted by only two groups previously.[33,37] Since the LFN canal is frequently ovoid in cross-section, the area is not adequately described by the unidimensional measures obtained in most studies.[31,35-37] Similar to Eski et al,[39] our study addressed this by acquiring bidimensional measurements, both in the long and short axes.

An additional aspect of the CT analysis was the assessment of the bony covering at the geniculate ganglion. Complete deficiency of

bony covering at this site is found in approximately 15% of the population, which represents a developmental variant.[41] Kim et al used CT to measure the length of the GSPN canal in 20 patients with unilateral BP and found the GSPN canal length to be significantly shorter on the affected side, most notably in patients with recurrent BP. The authors postulated that deficient bony covering and a shorter GSPN canal could lead to greater vulnerability of the geniculate ganglion to inflammation owing to closer proximity to the sphenopalatine ganglion via the GSPN.[8] This finding, however, was not reproduced in our study.

A further area of methodological importance our study aimed to address was inter-observer reliability. There is currently a paucity of data on the agreement parameters of facial nerve canal dimensions, with intra-observer and inter-observer agreement reported in only two studies respectively.[30,37] In the current study, whilst good levels of agreement were demonstrated with respect to the LFN canal measurement, only moderate inter-observer reliability was observed for bony covering at the geniculate ganglion. This was predominantly due to the challenge in differentiating complete from extensive partial dehiscence.

Of note, it should be appreciated that the intention of this study was not to generalize the findings to all cases of BP. The recurrent unilateral BP group was selected since these patients would seem the most likely candidates to demonstrate relevant anatomical risk factors. Since CT is only performed in BP patients who are potential

TABLE 5 CT based studies of intra-temporal facial nerve canal dimensions in patients with BP

	Comparison	Facial nerve segments	Slice thickness/matrix Measurement technique	Other	Outcome
Wadin et al[27] n = 22	Contralateral unaffected	Meatal foramen Mid-labyrinthine GG	1 mm contiguous/NS Single observer On axial sections	Additional imaging of temporal bone specimens	Image quality considered inadequate for evaluation
Kefalidis et al[19] n = 25	Contralateral unaffected	Meatal foramen Mid-labyrinthine	1 mm contiguous/512×512 Single observer On axial sections	Intra-observer agreement	Smaller meatal foramen/mid-labyrinthine diameter on symptomatic side
Murai et al[22] n = 16	Contralateral unaffected	Labyrinthine Tympanic Mastoid	NS/512×512 Single observer MPR oblique perpendicular to canal		Smaller labyrinthine/tympanic smallest cross section on symptomatic side
Rai et al[24] n = 30	Contralateral unaffected	Meatal foramen Mid-labyrinthine	1 mm contiguous/NS Single observer On axial sections	Correlation with facial nerve degeneration	Smaller meatal foramen/mid-labyrinthine diameter on symptomatic side
Eksi et al[26] n = 22	Contralateral unaffected	Labyrinthine Tympanic Mastoid	1 mm contiguous/512×512 Single observer Axial, coronal and sagittal planes Largest, narrowest diameters and segment length		No statistical differences in the diameters of different segments of facial canals between symptomatic and contralateral sides
Celik et al[23] n = 34	Contralateral unaffected	Mid-labyrinthine GG Mid-tympanic Second genu Mid-mastoid Stylomastoid foramen	1 mm contiguous/512×512 Single observer On axial sections (labyrinthine/tympanic) or sagittal sections (mastoid)	Diameter at second genu correlated with House-Brackmann grade	Smaller mid-labyrinthine diameter on symptomatic side
Hervochon et al[25] n = 51	Contralateral unaffected + asymptomatic controls	Meatal foramen Mid-labyrinthine Tympanic above oval window Mid-mastoid	0.6 mm contiguous/NS 2 observers Cross-sectional area and angles using double-oblique, 3D reconstructions perpendicular to the axis of the facial canal	Cross-sectional shape (round vs oval) Inter-observer agreement	Smallest cross-sectional area at meatal foramen No statistically significant differences in facial canal cross-sectional areas or angles for the three groups More oval-shaped labyrinthine segments on symptomatic side

Abbreviations: BMI, body mass index; GG, geniculate ganglion; NS, not stated.

candidates for surgical decompression in our institution, deliberate caution has been exercised in avoiding over-generalization of the study findings.

The authors acknowledge that the study has a number of limitations. Firstly, the small size of the samples limits the statistical power and it is possible that the negative finding was due to a type II error. Secondly, due to the retrospective nature of the study, long-term follow-up data was not available in all cases and the relationship between the LFN dimensions and long-term prognosis could not be established. Thirdly, soft tissue, such as the arachnoid band in the region of the meatal foramen, may determine the space available around the nerve and may play a significant role in the event of edema, but it cannot be evaluated using currently available imaging techniques. Finally, there was only moderate inter-observer reliability for the assessment of the bony covering at the geniculate ganglion, necessitating consensus agreement.

5 | CONCLUSION

This study found no significant difference in the narrowest LFN canal dimensions or the bony covering at the geniculate ganglion in patients with recurrent unilateral BP, compared to the contralateral side and matched control subjects, casting doubt upon their etiological influence in cases of recurrent BP.

CONFLICT OF INTEREST
The authors declare no conflicts of interest.

AUTHOR CONTRIBUTIONS
All authors made substantial contributions to the work: conception (Steve Connor, Rupert Obholzer), design (Steve Connor, Philip Touska), or the acquisition (Philip Touska, Cristina Dudau, Antanas Montvila, Milda Pucetaite), analysis and interpretation of data for the work (Philip Touska, Janki Patel, Irumee Pai, Steve Connor) and preparation of the manuscript (Philip Touska, IP, Steve Connor). All authors contributed to revising the manuscript for important intellectual content. All authors have reviewed and approved the final submitted manuscript, and agree to be accountable for all aspects of the work.

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BIBLIOGRAPHY
1. Shin K-J, Gil Y-C, Lee J-Y, Kim J-N, Song W-C, Koh K-S. Three-dimensional study of the facial canal using microcomputed tomography for improved anatomical comprehension. *Anat Rec*. 2014;297:1808-1816.
2. Fisch U, Esslen E. Total intratemporal exposure of the facial nerve: pathologic findings in Bell's palsy. *Arch Otolaryngol*. 1972;95:335-341.
3. Bodénez C, Bernat I, Willer JC, Barré P, Lamas G, Tankéré F. Facial nerve decompression for idiopathic Bell's palsy: report of 13 cases and literature review. *J Laryngol Otol*. 2010;124:272-278.
4. Yanagihara N, Honda N, Hato N, Murakami S. Edematous swelling of the facial nerve in Bell's palsy. *Acta Otolaryngol*. 2000;120:667-671.
5. Adour KK. Decompression for Bell's palsy: why I don't do it. *Eur Arch Otorhinolaryngol*. 2002;259:40-47.
6. Zandian A, Osiro S, Hudson R, et al. The neurologist's dilemma: a comprehensive clinical review of Bell's palsy, with emphasis on current management trends. *Med Sci Monit*. 2014;20:83-90.
7. Lee SY, Seong J, Kim YH. Clinical implication of facial nerve decompression in complete Bell's palsy: a systematic review and meta-analysis. *Clin Exp Otorhinolaryngol*. 2019;12:348-359.
8. Kim J, Jung GH, Park SY, Ko SH, Lee WS. Anatomical consideration of the temporal bone as a pathogenesis of Bell's palsy. *Med Hypotheses*. 2011;77:705-707.
9. Peitersen E. The natural history of Bell's palsy. *Am J Otol*. 1982;4:107-111.
10. Holland NJ, Bernstein JM. Bell's palsy. *BMJ Clin Evid*. 2014;1204:1-21.
11. Madhok VB, Gagyor I, Daly F, et al. Corticosteroids for Bell's palsy (idiopathic facial paralysis). *Cochrane Database Syst Rev*. 2016;7:CD001942.
12. Turgeon RD, Wilby KJ, Ensom MHH. Antiviral treatment of Bell's palsy based on baseline severity: a systematic review and meta-analysis. *Am J Med*. 2015;128:617-628.
13. Gagyor I, Madhok VB, Daly F, Sullivan F. Antiviral treatment for Bell's palsy (idiopathic facial paralysis). *Cochrane Database Syst Rev*. 2019;9: CD001869.
14. Jalali MM, Soleimani R, Soltanipour S, Jalali SM. Pharmacological treatments of Bell's palsy in adults: a systematic review and network meta-analysis. *Laryngoscope*. 2021. https://doi.org/10.1002/lary.29368.
15. Ballance C, Duel AB. The operative treatment of facial palsy: by the introduction of nerve grafts into the fallopian canal and by other intratemporal methods. *Arch Otolaryngol*. 1932;15:1-70.
16. Casazza GC, Schwartz SR, Gurgel RK. Systematic review of facial nerve outcomes after middle fossa decompression and transmastoid decompression for Bell's palsy with complete facial paralysis. *Otol Neurotol*. 2018;39:1311-1318.
17. Menchetti I, McAllister K, Walker D, Donnan PT. Surgical interventions for the early management of Bell's palsy. *Cochrane Database Syst Rev*. 2021;1:CD007468.
18. Adour KK, Diamond C. Decompression of the facial nerve in Bell's palsy: a historical review. *Otolaryngol Head Neck Surg*. 1982;90:453-460.
19. May M, Schaitkin BM. History of facial nerve surgery. *Facial Plast Surg*. 2000;16:301-307.
20. Zhu Y, Yang Y, Wang D, Dong M. Idiopathic recurrent facial palsy: facial nerve decompression via middle cranial fossa approach. *Am J Otolaryngol*. 2016;37:31-33.
21. Graham MD, Kartush JM. Total facial nerve decompression for recurrent facial paralysis: an update. *Otolaryngol Head Neck Surg*. 1989; 101:442-444.
22. Andresen NS, Sun DQ, Hansen MR. Facial nerve decompression. *Curr Opin Otolaryngol Head Neck Surg*. 2018;26:280-285.
23. Gantz BJ, Gmür A, Fisch U. Intraoperative evoked electromyography in Bell's palsy. *Am J Otolaryngol*. 1982;3:273-278.
24. Gantz BJ, Rubinstein JT, Gidley P, Woodworth GG. Surgical management of Bell's palsy. *Laryngoscope*. 1999;109:1177-1188.
25. Bobak CA, Barr PJ, O'Malley AJ. Estimation of an inter-rater intra-class correlation coefficient that overcomes common assumption violations in the assessment of health measurement scales. *BMC Med Res Methodol*. 2018;18:93.
26. McHugh ML. Interrater reliability: the kappa statistic. *Biochem Med*. 2012;22:276-282.
27. Jain S, Kumar S. Bell's palsy: a need for paradigm shift? *Ann Otol Neurotol*. 2018;1:34-39.
28. Zhang W, Xu L, Luo T, Wu F, Zhao B, Li X. The etiology of Bell's palsy: a review. *J Neurol*. 2020;267:1896-1905.
29. Kim IS, Shin SH, Kim J, Lee WS, Lee HK. Correlation between MRI and operative findings in Bell's palsy and Ramsay Hunt syndrome. *Yonsei Med J*. 2007;48:963-968.
30. Kefalidis G, Riga M, Argyropoulou P, et al. Is the width of the labyrinthine portion of the fallopian tube implicated in the pathophysiology of Bell's palsy? A prospective clinical study using computed tomography. *Laryngoscope*. 2009;120:1203-1207.
31. Vianna M, Adams M, Schachern P, Lazarini PR, Paparella MM, Cureoglu S. Differences in the diameter of facial nerve and facial canal in Bell's palsy: a three-dimensional temporal bone study. *Otol Neurotol*. 2014;35:514-518.
32. Dawidowsky K, Branica S, Batelja L, Dawidowsky B, Kovać-Bilić L, Simunić-Veselić A. Anatomical study of the facial nerve canal in comparison to the site of the lesion in Bell's palsy. *Coll Antropol*. 2011;35:61-65.
33. Murai A, Kariya S, Tamura K, et al. The facial nerve canal in patients with Bell's palsy: an investigation by high-resolution computed tomography with multiplanar reconstruction. *Eur Arch Otorhinolaryngol*. 2013; 270:2035-2038.
34. Celik O, Eskiizmir G, Pabuscu Y, Ulkumen B, Toker GT. The role of facial canal diameter in the pathogenesis and grade of Bell's palsy: a study by high resolution computed tomography. *Braz J Otorhinolaryngol*. 2017;83: 261-268.

35. Rai A, Lal P, Motwani G, Thukral B, Goel S. Significance of facial canal labyrinthine segment diameter in the pathophysiology of Bell's palsy. *Astrocyte*. 2015;2:60-63.

36. Hervochon R, Madelain V, Seiller I, Lahlou G, Nguyen Y, Tankéré F. CT and clinical prognostic factors in Bell's palsy: a study of 56 cases. *Clin Otolaryngol*. 2019;44:861-864.

37. Hervochon R, Seiller I, Trunet S, Nguyen Y, Tankéré F. Morphometric analysis of CT scans of the facial canal in Bell's palsy: a study of 51 patients. *Clin Otolaryngol*. 2019;44:656-659.

38. Eksi G, Akbay E, Bayarogullari H, Cevik C, Yengil E, Ozler GS. The effect of width of facial canal in patients with idiopathic peripheral facial paralysis on the development of paralysis. *Eur Arch Otorhinolaryngol*. 2015;272:2283-2289.

39. Wadin K, Thomander L, Wilbrand H. The labyrinthine portion of the facial canal in patients with Bell's palsy investigated by computed tomography. *Acta Radiol*. 1987;28:25-30.

40. Yang SP, Bach AM, Tuttle RM, Fish SA. Serial neck ultrasound is more likely to identify false-positive abnormalities than clinically significant disease in low-risk papillary thyroid cancer patients. *Endocr Pract*. 2015;21:1372-1379.

41. Isaacson B, Vrabec JT. The radiographic prevalence of geniculate ganglion dehiscence in normal and congenitally thin temporal bones. *Otol Neurotol*. 2007;28:107-110.