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Anaesthesia of the inferior alveolar and lingual nerves following subcondylar fractures of the mandible



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ABSTRACT

A retrospective chart review of 387 patients with condylar and subcondylar fractures revealed 2 cases of inferior alveolar nerve (IAN) and lingual nerve (LN) anaesthesia following the subcondylar fracture. Only 5 cases have been reported previously. The mechanism of action remains unknown but a review of the literature and an analysis of 120 dry human skulls supported the hypothesis that compression of the mandibular nerve at a high level, close to the foramen ovale, could cause anaesthesia. This complication is rare, because it requires compression at a particular angle. The antero-median angulation of the condyle must be close to the foramen ovale, and the fracture must be a unilaterally displaced fracture. The presence of an enlarged lateral pterygoid plate appeared to enhance the risk of compression. The IAN and LN anaesthesia could be resolved after open reduction of the fracture and IAN and LN anaesthesia constitute a strict indication for an early open fracture reduction.

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1. Introduction

Condylar and subcondylar fractures are the most common of all mandibular fractures (Chrcanovic et al., 2012). Despite the fact that the mandibular nerve exits the foramen ovale in the proximity of the condyle, condylar fractures are rarely associated with nerve disturbances although some cases have been reported (Fourestier et al., 1966; Laws, 1967; Zielinski, 1969; Schmidseder and Scheunemann, 1977; Goga et al., 1990; Griffiths and Townend, 1999; Renzi et al., 2004). The purpose of this article was to investigate the causes of nerve disturbances associated with condylar and subcondylar fractures. We investigated clinical cases with MRI images, measured 120 human skulls, and reviewed previous studies to inform discussions on the incidence and pathogenesis of this unusual complication, and provide a basis for advising treatment. Lesions of the facial nerve have been reviewed previously (Weinberg et al., 1995); thus, they will not be discussed here.

2. Materials and methods

We retrospectively reviewed the available maxillo-facial trauma charts for patients treated in 1989–2011. All charts with registered condylar and/or subcondylar fractures were further reviewed. Of these, cases were selected based on the following inclusion criteria: displaced or dislocated fractures, isolated fractures, or fractures that occurred in combination with other mandibular or other maxillo-facial fractures.

We also performed a literature search in Pubmed and Scopus for publications in English, French, or German that focused primarily on sensory nerve deficit after condylar and/or subcondylar fractures. The resulting articles were analyzed for data on the occurrence of sensory nerve deficits, the occurrence of pain in a sensory area of the mandibular nerve, the therapy applied, the possible mechanisms that could explain the connection between symptoms, and the type of fracture. Available panoramic radiographs and CT scans were compared for similarities to those of the selected clinical patients.

In addition we analyzed 120 dry, adult Indian human skulls of unknown sex and origin from 240 sites. We performed morphometric measurements of these skulls to provide a basis for postulating the pathophysiology of this condition and its extreme rarity of occurrence. We performed the following measurements with a calliper: from the deepest point in the glenoid fossa to the lateral

Abbreviations: ATN, auriculotemporal nerve; IAN, inferior alveolar nerve; LN, lingual nerve; FN, facial nerve; TMJ, temporomandibular joint.

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side of the foramen ovale; and from the deepest point in the glenoid fossa to the lateral rim at the base of the lateral pterygoid plate. Both were measured on the right and left sides (fo_left/right and pt_left/right, respectively). We noted the incidence of skulls with very large lateral pterygoid plates, compared to the findings of Krmpotic-Nemanic (Krmpotic-Nemanic et al., 1999). In this type of skull, the mandibular nerve, before branching into the IAN and LN, rested medially on the bony pterygoid plate, which exposed the nerve to the risk of compression onto a bony plate. We established a clinical correlate by examining cadaver heads, dissected at the department of Anatomy of Hasselt University.

This retrospective study was approved by the Institutional Review Board, and was conducted in compliance with the Helsinki Declaration guidelines.

The panoramic radiographs in Cases 1 and 2 were acquired with an Orthophos XGPlus (Sirona, Bensheim, Germany). The exposure parameters for panoramic radiographs were 66 kV, 10 mA, and 5 s. The cone-beam computed tomography (CBCT) data in Cases 1 and 2 were acquired with the Galileos CBCT (Sirona, Bensheim, Germany). The scan parameters for a 3-dimensional volumetric cone-beam scan from the Galileos were 85 kV, 7 mA, and 14 s. The Galileos device featured a fixed field of view of 15 cm, which resulted in a scan volume of $15 \times 15 \times 15$ cm with a resolution of 300 µm. The CT scans in Cases 1 and 2 were acquired with a Siemens Somatom Definition, 64-slice.

2.1. Statistical analysis

All statistical analyses were performed using SAS software (Version 9.2). Patient data were tested for normality according to Kolmogorov–Smirnov. After dichotomization of the variable indicating age, based on its median value (26 years), a Chi-square test was implemented.

Concerning the skull study, *T*-tests were conducted to determine differences between the 2 sides of each skull.

3. Results

The retrospective chart review identified 3 patients with registered sensory nerve deficits out of 387 charts that met the inclusion criteria. The age distribution of patients with condylar and/or subcondylar fractures (Fig. 1) was tested with the Kolmogorov– Smirnov Goodness-of-Fit Tests for Normal Distribution and



Fig. 1. Histogram of the age distribution of patients with condylar and/or subcondylar fractures.

yielded a *p*-value <0.01 pointing to a strong indication of the violation of the normality assumption.

The mean age was 30.9 years (standard deviation: 18.3 years). The patients included 262 males (67.7%) and 125 females (32.3%). The (sub)-condylar fractures included 320 unilateral (82.7%) and 67 bilateral (17.3%). In total, 454 (sub)-condylar fractures had been diagnosed. The mean age for unilateral fractures (30.6 years) was not significantly different from the mean age for bilateral fractures (31.8 years). Statistically it cannot be claimed that there are more bilateral fractures at a young age. The Pearson correlation coefficient was equal to 0.026 with a *p*-value equal to 0.614. The Chi-square test showed no correlation between age and fracture type, since the *p*-value was equal to 0.614. The same finding holds for the correlation between the gender of the patients and their type of fracture. The *p*-value was equal to 0.697 implying no statistically significant difference.

Case 1 exhibited deficits in an inferior alveolar nerve (IAN), a lingual nerve (LN), a long buccal nerve, and a motor branch to the medial pterygoid muscle. Case 2 exhibited deficits in an IAN and LN. The cases of IAN and LN are further described in detail. Case 3 exhibited Frey's syndrome, which occurred 15 years after the original fracture.

3.1. Case 1

On 5 August 2010, a white male, age 33 years, was treated in the emergency room after sustaining a fist blow to the right face and jaw during a soccer game. The panoramic radiograph (Fig. 2) showed a subcondylar fracture with some anterior displacement of the condyle, but no substantial loss of vertical height. A CBCT scan was also performed, and it revealed a subcondylar fracture with a medial displacement at an angle of 117°. The condyle was very close to the skull base. The dysocclusion was treated with closed reduction and maxillomandibular fixation (MMF), which was placed under general anaesthesia the next day (6 August 2010). Shortly after the closed maxillomandibular fixation, the patient began to notice a tingling sensation over the right half of the tongue. This worsened over the next days, until the patient noticed full hypoaesthesia of the right half of the tongue and lower lip. In addition, mouth opening was limited, and anaesthesia occurred over the right cheek. The patient also reported difficulties chewing on the affected side, due to reduced chewing force. Because the hypoesthesia did not resolve spontaneously, a CT scan was performed 6 weeks after the accident, which revealed that the displaced condyle had remained in close vicinity to the foramen ovale (Figs. 3 and 4). No spontaneous recovery was noticed in the IAN, LN, or long buccal nerve anaesthesia in the weeks following the closed reduction. An extra-oral approach to the fracture was performed



Fig. 2. Case 1. Panoramic radiograph of a subcondylar fracture at the right side. Image was acquired shortly after the trauma (R: right side).



Fig. 3. Case 1. CT-scan, frontal view, before the open reduction of the subcondylar fracture. The condylar head remains very near to the foramen ovale.



Fig. 4. Case 1: Fusion of 2 CT-sections: the displaced condyle (Co) (upper arrow) is in close vicinity to the foramen ovale (FO) (lower arrow).

but adequate reduction of the fracture was not possible. Clinically, a modest improvement of the lingual (LN) and labial hypoaesthesia (IAN) was noticed over the next weeks. In the following months. the IAN had fully recovered, but hypoaesthesia of the tongue remained. At the latest examination, on 15 January 2012, both the IAN and the LN had fully recovered. The mouth could be opened to 36 mm. The long buccal nerve anaesthesia and the pain incurred at the start of chewing and drinking remained permanent. The pain on chewing spontaneously disappeared after a few bites, but at the end of the meal, the patient reported "fatigue" in the right chewing muscles. Currently, the patient has reported continued diminished force in the jaw muscles of the affected side, and the healthy side is favoured for chewing meals. Ectromyography of the masticatory muscles was performed on 9 December 2011. This revealed a permanent, partial deficit in the medial pterygoid branch of the right mandibular nerve. This explained the motor hypofunction of the right medial pterygoid muscle.

3.2. Case 2

On 22 September 2011, a 16-year-old white male was hospitalized after an accident with an ATV (All-terrain Vehicle). The mandible had shifted to the right side with an open bite at the left side and the IAN and right LN had complete anaesthesia. The anaesthesia appeared immediately after the accident. The patient history revealed no previous TMJ complaints before the accident.

A panoramic radiograph (Fig. 5) revealed a subcondylar fracture, with little anterior displacement. A 3D CBCT scan clearly showed a 90° medial angulation of the condylar fragment. Fig. 6 shows the medial displacement and the very narrow passage for the nerve between the condylar head and the enlarged lateral pterygoid plate. The axial slides showed that the right lateral pterygoid plate was enlarged, but the left lateral pterygoid plate was within the normal size range (Fig. 7). There was only a 2.4 mm space between



Fig. 5. Case 2. Panoramic radiograph of the subcondylar fracture. Image was acquired shortly after the trauma.



Fig. 6. Case 2. CBCT scan of a subcondylar fracture. Frontal section, right subcondylar area. The vertical white arrow points to the foramen ovale (FO); the horizontal white arrow points to the enlarged lateral pterygoid plate (p). The lower vertical arrow points to the condyle (Co).

the tip of the fractured condylar head and the enlarged pterygoid plate, which left a very narrow passage for the mandibular nerve. Magnetic resonance imaging (MRI) revealed that the mandibular condyle was at a 90° angle, the mandibular nerve was under pressure and displaced, and an enhanced contrast captation appeared slightly posterior to the right foramen ovale (Fig. 8).

Due to the nerve deficit, open reduction and internal fixation was performed the next day. A perfect reduction and stabilization was achieved with one 4-hole osteosynthesis plate (Leibinger, Stryker, Germany) and four 7 mm screws. The day after the surgical procedure, the patient reported full sensory recovery. The remaining course was uneventful.

3.3. Case 3

This patient was examined in 2011, and diagnosed with Frey's syndrome on the right side. The chart on file showed that this patient had sustained a subcondylar fracture on the right side in September 1995. Because a dysocclusion had persisted, this was corrected a few months later, with a bilateral sagittal split osteotomy. Frey's syndrome was treated with 2% scopolamine ointment.

3.4. Morphometric measurements

The results from morphometric measurements of Cases 1 and 2 are summarized in Table 1.



Fig. 7. Case 2. CBCT scan of a subcondylar fracture. Axial slice. The white arrows clearly show the difference in length between the right and left pterygoid plates (p); the enlarged right plate is at the fracture site.



Fig. 8. Case 2. MRI scan of a subcondylar fracture. Frontal slice.

Table 1

Morphometric measurements for Case 1 and Case 2, deducted from the CBCT images.

CBCT morphometric measurements	Case 1	Case 2
Diameter of foramen ovale (width, length) (mm) Length of broken condylar process (mm) Closest distance between tip of condyle and any media bony ctructure in a vial slides of CBCT	8.38 * 5.06 23.75 mm 4 mm	7.15 * 3.18 20.54 mm 2.06 mm
(mm) Enlarged lateral pterygoid plate in 3D CBCT (yes/no) cfr. Skulls that we analyzed	No	Yes (R side only)

We created a reference set of measurements by examining 120 dry skulls. We measured the distance to the lateral edge of the foramen ovale to determine the minimum distance that the broken fragment must traverse to reach the lateral side of the foramen ovale in cases of very high local compression.

The following skull base morphometric measurements were performed:

- fo_left: deepest point of the midfossa (MF) to the lateral edge of the left foramen ovale
- pt_left: deepest point of the MF to the rim of the base of the left lateral pterygoid plate

- fo_right : deepest point of the MF to the lateral edge of the right foramen ovale
- pt_right : deepest point of the MF to the rim of the base of the right lateral pterygoid plate

The skull base morphometric measurements were analyzed with descriptive statistics (Table 2). Normality was rejected for all 4 variables (fo_left, pt_left, fo_right, and pt_right).

The mean values for the pairs of right and left measurements were 18.84 and 18.28 mm for the foramen ovale and 26.72 and 26.39 mm for the pterygoid plate. Although the differences between sides (0.56 and 0.33 mm, respectively) were small, they were highly significant (p = 0.0003 and p = 0.0280, respectively). The Agreement Plot shows the distribution of skulls with unequal measurements between sides (Fig. 9, Fig. 10).

More specifically, the agreement plot for the distance from the midfossa to the foramen ovale (Fig. 9) showed that more skulls had a larger measurement on the left side than the right side. In contrast, the agreement plot for the distance from the midfossa to the pterygoid plate (Fig. 10) showed that the measurements were more uniform on the 2 sides.

We also analyzed the extension of the lateral plate of the pterygoid bone according to the coordinates described by Krmpotic-Nemanic (Krmpotic-Nemanic et al., 1999). An extremely large lateral lamina of the pterygoid process would be expected to force the LN and the IAN into a long, curved course that followed the shape of the enlarged lamina. This anatomical variation constitutes a risk for compression against this enlarged plate at a level below the foramen ovale and below the lateral rim, at the base of the lateral pterygoid plate. We found this extremely large plate was present in 18 out of 240 sides (7.50%). However, only one skull displayed a bilateral occurrence.

Fig. 11 shows an example of a short pterygoid plate. In contrast, Fig. 12 shows a very large pterygoid plate. Fig. 13 shows how the position of foramen ovale relative to the enlarged lateral pterygoid plate presents a bony wall at the medial side of the exiting mandibular nerve. This close proximity presents a high risk for nerve damage in case of a compression injury close to the foramen ovale.

The drawings in Fig. 14(a,b) illustrate potential mechanisms of compression, where the nerve is pressed onto an enlarged pterygoid plate in the case of a subcondylar fracture.

A dissection specimen of the IAN and LN (Fig. 15) showed that the distant position of the medial pterygoid muscle precluded a potential role in the compression of the IAN or LN after a subcondylar fracture. For the medial pterygoid muscle to play a role, the displacement would have to be very anterior and inferior, which was not observed in the cases described in the literature or in the charts reviewed in this study.

4. Discussion

Dislocated mandibular fractures between the mandibular and mental foramina were found to have a high incidence of damaging

Table 2

Descriptive statistics on skull base measurements for 120 dry skulls; cadaver study.

Variable	Label	Ν	Mean (mm)	Median (mm)	Variance	Min (mm)	Max (mm)
fo_left	fo_left	120	18.84	18.5	5.193	15	25
pt_left	pt_left	120	26.72	27	6.588	20	40
fo_right	fo_right	120	18.28	18	4.927	15	25
pt_right	pt_right	120	26.39	26	7.568	20	40

Abbreviations: fo_left, fo_right: from the midfossa to the foramen ovale, right and left sides, respectively; pt_left, pt_right: from the midfossa to the lateral pterygoid plate, right and left sides, respectively.



Fig. 9. *T*-test results. Agreement plot compares the left and right measurements of the fo_ variable for each skull.



Fig. 10. *T*-test results. Agreement plot compares the left and right measurements of the pt_ variable for each skull.

the mandibular nerve, and they were associated with slow recovery rates (Renzi et al., 2004). This was expected, because the displacement can disrupt the bony canal that carries the IAN. However, Renzi et al. (2004) did not study condylar fractures of the mandible.

Condylar fractures can be dislocated or displaced. In the dislocated condylar fracture, the condylar fragment is disconnected from the mandibular ramus, and it hangs loose, separated from the mandible. In the displaced condylar fracture, a radiographic



Fig. 11. Dry human skull study; skull base. An example of a short pterygoid plate (black arrow points to the lateral pterygoid plate); the foramen ovale is indicated with a blue star, and the deepest point of the middle of the glenoidal fossa is indicated with the red dot.



Fig. 12. Dry human skull study; skull base. An example of a very large pterygoid plate (double sided arrow). The black arrow points to the end of the pterygoid plate. The red arrow points to the rim of the base of the pterygoid plate. The blue star indicates the foramen ovale, and the red dot indicates the deepest point in the middle the glenoid fossa.



Fig. 13. Dry human skull study; skull base; lateral view of the proximity between the foramen ovale and the enlarged lateral pterygoid plate. This proximity presents the exiting mandibular nerve (blue string) with a bony wall very close to the medial side.

connection remains between the ascending ramus and the condylar or subcondylar segment, but the fractured segment is angulated towards the medial, lateral, anterior, or posterior direction. These definitions highlight an important distinction in the 2 types of condylar fractures. Nevertheless, there is no uniform use of the



Fig. 14. Illustrations of a potential mechanism for nerve compression. The mandibular nerve (yellow chord) is pressed by the condylar head (rounded structure) against an enlarged lateral pterygoid plate. (a) posterior view; (b) lateral view shows the position of the compressed nerve in white outlines under the condylar head.

terms 'displacement' and 'dislocation' throughout the literature of different case-reports and corresponding radiographs (Brandt and Haug, 2003).

Both dislocated and displaced condylar fractures have been associated with deficits of the mandibular nerve (Table 3). Most reports are anecdotal case reports. Only one report (Schmidseder and Scheunemann, 1977) provided a prospective study of 237 subcondylar fractures of the mandible. They described "ad latem" condylar dislocations as a displacement of the condyle to the lateral side, and it could be anterior, posterior, medial, and lateral; they described "ad axim" condylar dislocations in terms of the displaced condylar fracture defined above. In the group of dislocated subcondylar fractures, they found 1 chorda tympani deficit, 1 facial nerve deficit, and 3 auriculotemporal nerve (ATN) deficits, and 2 of



Fig. 15. Cadaver dissection specimen displays the IAN and LN. This shows that the medial pterygoid muscle is located at a distance too far from the IAN or LN to play a role in compression after a subcondylar fracture. The blue star indicates the medial pterygoid muscle. The yellow arrow extends from the condyle to the IAN and LN, below the foramen ovale.

the latter 3 developed Frey's syndrome. In the group of displaced subcondylar fractures, they found 2 ATN deficits and 1 long buccal nerve deficit, but the latter only occurred after a symptom free interval. A total of 8 nerve deficits were found in 237 subcondylar fractures; an incidence of 3.38%. No deficit of the IAN or LN was found in that prospective study.

Our retrospective chart review showed a nerve deficit incidence of 0.775% (3/387), far lower than that reported by Schmidseder and Scheunemann (Schmidseder and Scheunemann, 1977).

Displaced condylar fractures also have been reported to cause deficits to the mandibular nerve.

Other reports have included lesions of the ATN, the IAN, the LN, and the buccal nerve. Our report included a deficit of the motor branch to the medial pterygoid muscle.

Sensory lesions secondary to displacement or dislocation of (sub)-condylar fractures typically displayed the following characteristics:

- 1. a symptom-free interval, up to about 1 week, occurring between the trauma and the occurrence of the nerve impairment symptoms
- 2. a variable interval occurring between the condylar fracture and Frey's syndrome, from 2 to 3 months up to 2 years, in the case of a disrupted ATN
- 3. IAN and/or LN deficits were only associated with unilateral subcondylar fractures with a condylar fragment displacement; they were never found in unilateral or bilateral condylar fractures with dislocations (as defined above)
- 4. a closed reduction of the fracture did not improve the symptoms
- 5. open reduction with a correctly positioned condyle with rigid fixation or a condylectomy typically improved the symptoms

Table 3

Nerve disturbances that have been reported in the literature secondary to dislocated or displaced condylar and/or subcondylar fractures of the mandible.

Author	Year	Chorda	Auriculo-temporal	Medial pterygoid	Long buccal	Inferior alveolar	Lingual	DL/	U/B
		tympam	lielve	lierve	lielve	lierve	lieive	DP	
Schmidseder and	1977	+						DL	NS
Scheunemann									
Schmidseder and	1977		+					DL	NS
Scheunemann									
Schmidseder and	1977		+					DL	NS
Scheunemann									
Schmidseder and	1977		+					DL	NS
Scheunemann									
Schmidseder and	1977		+					DP	NS
Scheunemann									
Schmidseder and	1977		+					DP	NS
Scheunemann									-
Laws	1967		+					DL	В
Laws	1967		+					NS	U
Martis and Athanassiades	1969		+					DP	B (condyle/body)
Zoller et al.	1993		+					DP	B (condyle/
	1007							NG	sympnysis)
Goodman and Heights	1987		+					INS NG	В
Mellor and Shaw	1996		+					INS DI	B D (auto a state st
Sverzut et al.	2004		+					DL	B (SUDCONDYIAT/
Channe	1074							DI	Dody)
Storrs Criffithe and Town and	1974		+		+			DL	В
Zielinelti	1999					+	+	DP	U
Champushau	1969					+		DP	U
Chernyshev	2004		+			+	+	DP	U
Goga et al.	1990					+	+	DP	U
Laws	1967		+		+	+		DP	U
Laws	1967		+		+	+	+	DP	U
Laws Cohmideeden end	1907					+		DP	U
Scheupemann	1977				+			DP	U
Scheuhemann	1062							NC	D
Egyeul	1903				+			IND	D

DL, Dislocation; DP, Displacement; NS, not stated; U, unilateral; B, Bilateral including mandibular fractures.

significantly within 24 h (Egyedi, 1963; Schmidseder and Scheunemann, 1977; Goga et al., 1990; Chernyshev, 2004)

The data indicated that the ATN was more often at risk than the IAN or LN (Table 3).

Several different theories have been proposed to explain IAN and/or LN deficits, including compression, elongation, haematoma, oedema, and fracture of the base of the skull. The notion that haematoma and oedema play a role can explain the symptom free interval. Although the participation of haematoma and oedema cannot be ruled out, we believe they represent a minor source of pressure. In our cases, the MRI analysis ruled out any vascular damage to the maxillary artery that would cause life-threatening haemorrhage (Bozkurt et al., 2009).

Goubran (1978) speculated that the paraesthesia cases described by Laws (1967) were not due to extracranial damage to the trigeminal nerve, but to a transverse fracture of the base of the skull (Goubran, 1978). A violent upward displacement of the condylar process into the mandibular fossa has the potential to cause a fracture of the middle cranial fossa (Rowe and Killey, 1970). Goubran hypothesized that, when the fracture line radiated medially to involve the foramen ovale, it might give rise to neuropraxia of the third division of the trigeminal nerve (Goubran, 1978). Indeed, Tekeli described lingual nerve paraesthesia after a skull base fracture that included a fracture through the wall of the foramen ovale (Tekeli et al., 2008). After 3 weeks, that patient recovered completely from the paraesthesia. In addition, Lanigan could not exclude a fracture at the skull base as a possible mechanism of sensory nerve deficit after (sub) condylar fractures (Lanigan, 2003). Stranjalis reported a case of post-traumatic, transient, bilateral, trigeminal sensory and motor neuropathy after a skull base fracture that involved fractures through both foramina ovale, visible on the CT scans (Stranjalis and Sakas, 2002). That patient did not fully recover from the sensory deficits, but did fully recover from the motor nerve deficits.

A fracture at the skull base involving the foramen ovale would imply an indirect crushing injury of the trigeminal nerve in the foramen ovale. This would certainly explain the involvement of a motor branch, but this mechanism would not explain the immediate recovery of sensation after repositioning or after removal of the condylar process. In our opinion, the scenario described in Goubran's hypothesis (a fracture of the middle cranial fossa that includes the wall of the foramen ovale following a subcondylar fracture) would be highly unlikely in the cases reviewed here, based on following observations:

- the sensory deficits fully and immediately recovered after open reduction of the condylar fracture
- the CT and MRI scans showed no sign of any fracture at the foramen ovale or at the skull base
- variable symptomatologies were observed with separate involvement of the IAN and the LN
- the symptom-free interval we observed was not present in reported cases of fractures at the wall of the foramen ovale (Stranjalis and Sakas, 2002; Tekeli et al., 2008).

In a review of 882 patients that sustained facial fractures, no relationship could be identified between isolated mandible fractures and the individual cranial bones fractured (Haug et al., 1994).

The observations of this study support the notion that compression may be a potential mechanism of nerve entrapment that could cause pain and/or hypoaesthesia or anaesthesia. Most authors believe that the impairment of sensibility associated with the condylar fracture is due to pressure of the condylar head on one or more branches of the mandibular nerve. This pressure may affect the sphenomandibular ligament, the stylomandibular ligament, the medial pterygoid muscle, or even the foramen ovale. In this study, compression towards the medial pterygoid muscle or the sphenomandibular ligament was highly unlikely, because, in our patients, the CT and MRIs showed that those structures were located far anterior and inferior to the condyle. Furthermore, the stylomandibular ligament, as described by Zielinksi, was located too posteriorly to play a role in the mechanism of compression (Zielinski, 1969).

The fracture described in Case 1 was a low subcondylar fracture that involved displacement with a fragment angulation that could exert pressure between the medial side of the ascending ramus and the foramen ovale. In Case 2, the medial-anterior—inferior displacement of the condyle was found close to an extended large pterygoid plate. In both cases the position of the condyle was superior to the sphenomandibular ligament or the median pterygoid muscle.

Isberg described IAN and LN entrapment that occurred when these nerves were subjected to mechanical irritation, including compression, rubbing, traction, or friction. These perturbations were typically caused by a muscular spasm of the pterygoid muscles (Isberg, 2001). The predominant symptom of nerve entrapment is pain.

Johansson measured histologic frontal plane sections to determine the distance between the foramen ovale and the medial pole of the condyle (Johansson et al., 1990). This distance (5–10 mm) was related to potential mechanical irritation during medial disc displacement due to compression. The irritation resulted in pain symptoms upon movement of the joint.

According to Piagkou, several anatomical features might allow compression of the IAN and the LN. These included an ossified pterygospinous or pterygoalar ligament, a large lateral plate lamina of the pterygoid process, and medial fibres on the lower belly of the lateral pterygoid. A contraction of the lateral pterygoid, due to the connection between nerve and anatomic structures (soft and hard tissues), might lead to mandibular nerve compression. This would result in paraesthesia, anaesthesia, or pain (Piagkou et al., 2011).

None of these authors reported patients with IAN or LN nerve disturbances related to traumatic condylar displacement (Johansson et al., 1990; Isberg, 2001; Piagkou et al., 2011).

In a literature review of IAN and/or LN impairments secondary to subcondylar fractures, no study reported pain in the course of the LN or IAN. Schmidseder described pain in the innervation area of the buccal nerve, which improved over time. Schmidseder also reported neuralgic pain in the ATN area (Schmidseder and Scheunemann, 1977). The neuralgic pain completely disappeared after resection of the displaced condyle. Egyedi also reported a case where burning pain occurred in the long buccal nerve area. The pain occurred at the start of a meal and improved over the meal duration and completely disappeared after a condylectomy (Egyedi, 1963).

Nerve entrapment through compression mechanism might explain the pain we observed in Case 1 of this review. That pain occurred at the start of the chewing cycle, and was due to incomplete condylar repositioning.

Compression at the trigeminal ganglion is a known cause of trigeminal neuralgia. It results in masseteric paresis in 10% of cases (de Siqueira et al., 2007), due to compression of the motor branch. Just outside the cranium, the motor and sensory branches of V3 unite to form a short main trunk, the mandibular nerve. The medial pterygoid nerve branches from the main trunk and courses close to the otic ganglion. Involvement of the motor branch in Case 1 suggested a high compression mechanism; this allowed us to

rule out a role for the medial pterygoid muscle in the sensory nerve deficit. We speculate that Case 1 is an example of a compression that occurred at a high level, close to the foramen ovale, where the condyle compressed the nerve against the rim of the medial wall of the foramen ovale, where it extended to the pterygoid base.

Both Piagkou and Krmpotic-Nemanic pointed out that the lateral laminae of the pterygoid process can be extremely large; the lateral lamina in the pterygospinosus process region varied in width from 5 to 17 mm (Krmpotic-Nemanic et al., 1999; Piagkou et al., 2011). In this study, the mandibular nerve in Case 2 may have been at greater risk of compression because this anatomical variant was also found in Case 2, unilaterally at the site of the fracture, representing a clinical example of Krmpotic-Nemanic's theory. Krmpotic-Nemanic et al. found large lateral plates in 6/200 (3%) sites, with 1 bilateral occurrence in 100 skulls (Krmpotic-Nemanic et al., 1999).

Kim et al. found that the bifurcation of the LN and IAN was located around the mandibular notch at 14.3 mm inferior to the foramen ovale; this is well below the position of the condyle on the CT and MRI (Kim et al., 2004). Therefore, it is understandable how the combined impairment of the LN and IAN might occur, because these nerves are found in a single bundle at the compression site. In our Cases 1 and 2, this might explain why both the LN and the IAN were involved in the sensory deficits.

We analyzed the available radiographs in several previously published case reports (Laws, 1967; Zielinski, 1969; Goga et al., 1990; Griffiths and Townend, 1999; Chernyshev, 2004). Three of these cases were similar to our cases, because the CT scans showed the condyle overlying, or nearly overlying, the foramen ovale (Goga et al., 1990; Griffiths and Townend, 1999; Chernyshev, 2004). The Laws study did not include CT scans, but they provided a Townes View radiograph that showed the same angulation as that observed in our cases (Laws, 1967). The Zielinski study also had no CT scan, and they only showed a lateral radiograph of a low subcondylar fracture with an anterior displacement of the fragment; unfortunately, the medial component of the displacement was not shown (Zielinski, 1969).

Panoramic radiographs were found in only 2 studies other than the cases of this review (Goga et al., 1990; Griffiths and Townend, 1999). In the cases of IAN and LN impairment, the condylar fragment showed very little vertical loss and only a moderate anterior inclination. The medial displacement could not be seen on the panoramic radiographs.

In all cases of subcondylar fractures with IAN and/or LN sensory deficits, the imaging showed that the condyle position was close to the skull base, medially angulated. These suggested the possibility that the base of the broken fragment might exert pressure upon closing the mouth. This exerted pressure was necessary to compress the nerve.

We propose that sensory impairment of the IAN and LN after a subcondylar fracture is rare, due to the necessity of fulfilling the following conditions simultaneously:

- the subcondylar fragment must be supported by the ascending ramus
- the condylar fragment must be sufficiently long to reach the foramen ovale or the lateral pterygoid plate
- the angulation of the fragment must be displaced medially and capable of exerting pressure
- the pressure must be exerted against a bony structure; this structure could be the medial wall of the foramen ovale in the case of a narrow foramen, or it could be the base of the pterygoid plate close to the foramen ovale or lower, against a large lateral laminae pterygoidei.

When any one of these conditions is not met, nerve compression is not possible, due to the following reasons:

- a dislocation fracture creates a 'loose' fragment that cannot exert pressure
- the fractured fragment is short and cannot reach a bony structure
- the fragment is displaced at an angle that does not allow connection with the nerve
- an associated fracture elsewhere in the mandible prevents the broken fragment from creating sufficient pressure upon closing the mouth.

Because conservative treatment did not resolve nerve impairment, but open reduction or condylectomy did resolve all cases of nerve impairment, the treatment of choice should be the open reduction. In Case 1 it became obvious that, once consolidated, reduction of the fracture and repositioning of the condyle is very difficult. In these cases, a condylectomy is a good alternative to finish off the sensory deficit. Although the strategy for the management of condylar fractures is still controversial (Sforza et al., 2011), we would propose that sensory disturbances should be the 5th absolute indication of open reduction of condylar fractures next to the 4 others listed by Zide and Kent (1983).

5. Conclusion

Inferior alveolar nerve and lingual nerve anaesthesia following a subcondylar fracture are rare complications because it requires compression of the nerve by the condyle at a particular angle. This requires a unilateral displacement fracture of the condyle. The presence of an enlarged lateral pterygoid plate enhances the risk of compression. The IAN and LN anaesthesia can be eliminated after open reduction of the fracture. Therefore, IAN and LN anaesthesia constitute a strict indication for an early open fracture reduction. These findings might enhance the understanding of mandibular nerve neuropathies and promote the development of novel treatments in cases with an enlarged lateral pterygoid plate.

Conflict of interest

The authors declare no conflicts of interest.

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