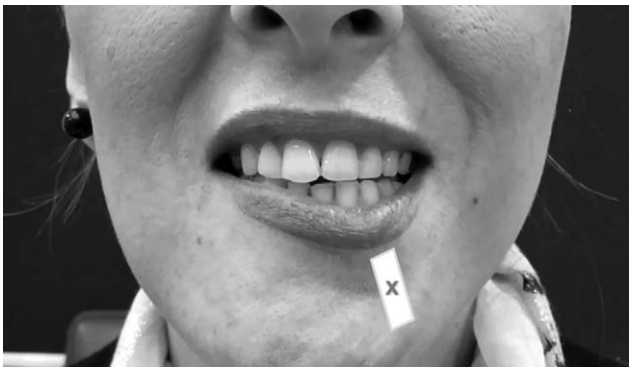

FIGURE 2. Patient photo: congenital unilateral lower lip palsy patient, before treatment.

45% of patients did not come back for chemodenervation. Treatment cost can be a reason for the high dropout rate (Fig. 1).

Other treatment options for unilateral lower lip palsy include selective neurectomy of the healthy contralateral side. Another reconstructive option is reconstruction of the lower lip depressor activity with digastric muscle transfer.

Patients with CULLP have high level of concern regarding their mouth symmetry during speech and smiling. Chemodenervation treatment resulted with higher patient satisfaction with speech in comparison to smiling. One possible reason can be the anatomy of the smile as explained by Rubin et al in 1974.[10] There are there 3 types of smile, namely "Mona Lisa," canine and full denture smile. The only smile affected by lower lip palsy is the full denture smile, therefore it can be speculated patients that have the other smile patterns are not affected as much. Whereas unilateral lower lip palsy almost always causes serious asymmetry during speech, especially while pronouncing sounds that incorporate i and e.

Although this study was carried out in CULLP patients only, results are also applicable to other DLI related asymmetries. Contralateral DLI chemodenervation is also a very valid option for patients with acquired unilateral lower lip palsy such as Bell palsy, iatrogenic marginal mandibular palsy.

CONCLUSION

Most CULLP patients are concerned regarding their asymmetric appearance while smiling or speaking. Chemodenervation of the contralateral DLI muscle reduces concern levels and has high patient satisfaction. Chemodenervation of the contralateral healthy DLI muscle is a valid, practical treatment option.

REFERENCES

1. Breslow GD, Cabiling D, Kanchwala S, et al. Selective marginal mandibular neurectomy for treatment of the marginal mandibular lip deformity in patients with chronic unilateral facial palsies. *Plast Reconstr Surg* 2005;116:1223–1232
2. Hussain G, Manktelow RT, Tomat LR. Depressor labii inferioris resection: an effective treatment for marginal mandibular nerve paralysis. *Br J Plast Surg* 2004;57:502–510
3. Krane NA, Markey JD, Loyo M. Neuromodulator for the treatment of congenital unilateral lower lip palsy. *Ann Otol Rhinol Laryngol* 2019;128:62–65
4. Isken T, Gunlemez A, Kara B, et al. Botulinum toxin for the correction of asymmetric crying facies. *Aesthetic Surg J.* 2009;29:524–527.
5. Chen CK, Tang YB. Myectomy and botulinum toxin for paralysis of the marginal mandibular branch of the facial nerve: a series of 76 cases. *Plast Reconstr Surg* 2007;120:1859–1864
6. Udagawa A, Arikawa K, Shimizu S, et al. A simple reconstruction for congenital unilateral lower lip palsy. *Plast Reconstr Surg* 2007;120:238–244
7. Lindsay RW, Edwards C, Smitson C, et al. A systematic algorithm for the management of lower lip asymmetry. *Am J Otolaryngol - Head Neck Med Surg* 2011;32:1–7
8. Terzis JK, Ph D, Kalantarian B. Microsurgical strategies in 74 patients for restoration of dynamic depressor muscle mechanism: a neglected target in facial reanimation. *Plast Reconstr Surg* 1999;105:1917–1931
9. Godwin Y, Tomat L, Manktelow R. The use of local anesthetic motor block to demonstrate the potential outcome of depressor labii inferioris resection in patients with facial paralysis. *Plast Reconstr Surg* 2005;116:957–961
10. Rubin LR. The anatomy of a smile: its importance in the treatment of facial paralysis. *Plast Reconstr Surg* 1974;53:384–387

Facial Nerve Palsy Associated With Orthognathic Surgery

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Abstract: The authors report a 25-year-old female who presented facial palsy after undergoing bimaxillary orthognathic surgery for retrognathism correction. Orthognathic surgery is a procedure used to treat dentofacial deformities which aims to achieve an adequate relationship between dental archs, improving function (such as chewing, breathing, and speaking) and facial aesthetics. Even though there are some complications that can occur during the intraoperative and postoperative periods like bleeding, tooth, soft-tissue damage, nerve damage, bad split, infection, and nonunion, facial nerve injuries are considered rare complications after this kind of surgical procedure. Despite being uncommon, rarely described, transient, and spontaneously resolved in almost all patients, facial nerve palsy is one of the most serious complications because it directly affects patient's quality of life and social interaction.

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Orthognathic surgery (OgS) is an elective procedure performed to correct dentofacial deformities and improve function (such as chewing, breathing, and speaking), posture, and aesthetic of facial lower and mid-thirds.[1] Even though there are some complications that can take place during the intraoperative and postoperative periods like bleeding, tooth, and soft-tissue damage, nerve damage, bad split, infection, and nonunion, facial nerve injuries are considered rare complications after OgS.[2]

Despite being uncommon, rarely described, generally transient and spontaneously resolved in almost all patients, facial nerve palsy is one of the most serious complications associated to OgS, once it directly affects patient's quality of life and social interaction,[3] causing distress and psychologic problems due to aesthetic and functional alterations.[4] Therefore, it can be considered severe and must not be neglected.[5]

Facial nerve injury after OgS usually involves the peripheral facial nerve distal to the stylomastoid foramen.[6] The peripheral facial palsy (PFP) is characterized by unilateral weakness or paralysis of facial expression muscles and can cause asymmetry on the face and difficulty of eating, drinking and controlling salivary flow. Facial nerve compression, infection, complete or incomplete nerve transection, nerve traction, and nerve ischemia from injection of vasoconstrictors are some possible etiologies for this condition.[6,7]

A patient with PFP following OgS is described, as well as its management and an 11-month clinical follow-up. The medical records, postoperative photographs, and literature were reviewed in detail to collect data on the clinical course, treatment, and outcomes.

CLINICAL REPORT

A healthy 25-year-old woman whose main complaint was the difficulty of breathing and facial pain associated with the temporomandibular joint underwent a bimaxillary OgS to correct class II dentofacial deformity (retrognathism). The procedure consisted in a Le Fort I osteotomy for maxillary rotation, and bilateral sagittal split osteotomy (BSSO) for mandibular advancement. The patient underwent preparatory orthodontic treatment for 2 years.

Under general anesthesia, the operation was preceded by a local vasoconstrictor infiltration (Bupivacaine 0.5%, with Epinephrine 1:200,000; Cristália, Itapira, Brazil) in the perimandibular region of the ascending ramus and at the angles of the mandible, and in the upper jaw from the pyriform apertures to the retrozygomatic region. Reciprocating electric saw was used to perform the osteotomies. After Le Fort I osteotomy, a surgical splint (intermediary) was used to guide the upper jaw to its new position, where it was fixed by 1.5 mm titanium plates and 1.5 × 5 mm screws after intermaxillary fixation (IMF). In the mandible, delicate chisels and a Smith splinter were used complete the BSSO. After the osteotomy was finished, another surgical splint (final) was used to guide the distal segment to its predetermined advancement (7 mm) and IMF was applied. The segment was then fixed in place using 2.0 mm titanium plates and 2.0 × 5 mm screws. After releasing the IMF, the occlusion was found to be acceptable.

The intraoperative course was uneventful, with a total surgery time of 4 hours, between intubation and extubation. Ice packs were applied during the first 48 hours postoperatively.

The postoperative period was characterized by the presence of a remarkable swelling, comparing to what we generally observe in our patients that undergo bimaxillary OgS, especially in the left side of the face. In the first day after surgery, as the patient complained about having difficulty in closing her left eye, we implemented eye protection with tear-like eye drops during the day and patches to protect the eyes during sleep and rest periods. No ophthalmologic evaluation was performed due to the unavailability of an ophthalmologist to see her in a short time through our public health system, and also because there was no symptomatology that could justify her referral, although it is known that there is a risk of developing corneal ulcer in these situations.

Following our service routine for all patients that undergo bimaxillary OgS, she was maintained under intravenous antibiotic (1 g Cephalotine) and received corticoid therapy with Dexamethasone 10 mg every 8 hours during the 2 days of hospitalization.[8,9] After hospital discharge, it was prescribed oral cephalexin (three times daily for more 5 days),[10] and the analgesia was through 100 mg Nimesulide and 500 mg Dipyrone administered, respectively, 2 and 4 times daily for 3 days. Also, the patient was instructed to perform a 0.12% chlorhexidine gluconate mouthwash twice a day for 7 days.

On the 3rd postoperative day, the patient still had a great edema, was unable to wrinkle her forehead, what already suggested a PFP, and a severe asymmetric smile was also present. Stapedial reflexes and taste functions were normal, she had no clinical signs of viral infection and denied having Bell palsy before.

On the 7th postoperative day, the patient returned to the Department of Oral and Maxillofacial Surgery for control. After facial expression tests, we could rule out a central lesion, once all branches were affected. Clinical examination showed a lack of facial muscle motility on the entire left side of her face, difficulty in closing the left eye, moving the forehead and smiling (Fig. 1A-B). Panoramic, lateral, and frontal X-rays were taken, but no signs of unfavorable splits were noticed (including the styloid process). At this time, we could identify that all facial branches were involved, to a greater or lesser extent and, according to House and Brackmann,[11] this palsy could be classified as a severe dysfunction asymmetry, once the front does not move, there is an incomplete closure of the eye and the mouth only shows slight movements.

It was thought that the paralysis would have as probable causes either the substantial postoperative swelling, compression due to the use of retractors during the osteotomy or compression during the BSSO opening. A viral infection Bell's palsy (although less likely, once she had no clinical manifestations and the timing of a possible injury coincided with the surgery) could also be considered. Anyway, it is generally difficult to exclude facial nerve infections with the herpes zoster virus in the differential diagnosis because the major differences in this condition (presence of small blisters, or vesicles, on the external ear and hearing disturbances) may occasionally be lacking.

As it was not possible to determinate the cause of facial palsy, Prednisolone 20 mg was prescribed (2 capsules in the morning for 5 days, then 1 capsule in the morning for more 5 days). Tablets of Acyclovir 200 g were also prescribed (2 g per day for 5 days). According to the algorithm we use to approach facial nerve palsy,[12] when the exact cause is unknown, even with a high probability of being related to the surgical procedure, the conduct is to prescribe oral steroids and optionally offer antiviral therapy in addition to steroids. No electroneurography was performed once in the protocol followed, asking for electrodiagnostics is considered optional. In addition, once the patient was operated in the public health system of our country, it would take much time for her to get the examination done.

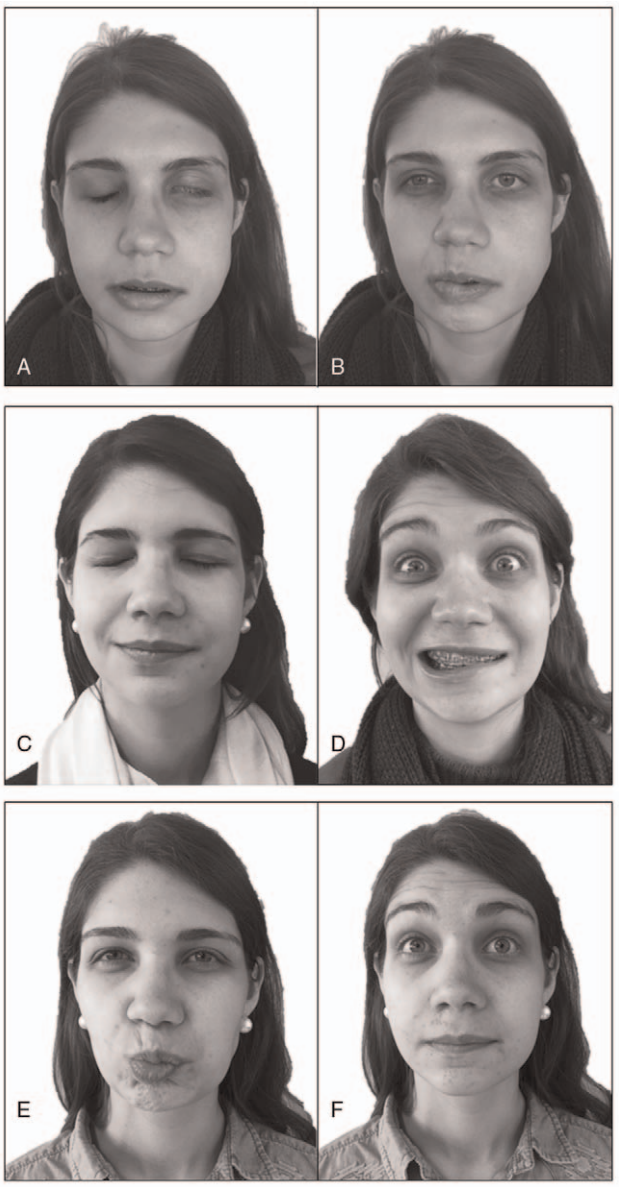

FIGURE 1. (A-B) 7 days after surgery: clinical examination showed a lack of facial muscle motility on the left side of the face: incomplete closure of the eye and mouth able to perform only slight movements (difficulty to smile). (C) 21 days after surgery: the patient presented important improvements (eyes completely closed). (D) 35 days after surgery: patient moving the forehead normally but still with some difficulty on smiling. (E-F) 50 days after surgery: patient's facial motility completely recovered (whistling and normally moving forehead and eyebrow).

In the 15th postoperative day, the patient was stable and started motor and functional physiotherapy. After 21 days she had already presented important improvements in her facial motility and was already able to completely close her left eye. At this point, eye protection maneuvers were discontinued (Fig. 1C).

After 35 days, she presented a very good evolution, but motor physical therapy was maintained because her mouth was not completely recovered yet (Fig. 1D).

Fifty days after surgery, a complete recovery of all left-sided facial muscle motility was achieved, with total regression of the facial palsy (Fig. 1E-F). The patient also reported almost complete regression of paresthesia, but with remnants on the other side. She did not mention major influences of paralysis in relation to her normal functions. After 11 months, no further signs of paresthesia could be identified. Written informed consent was obtained from the patient to publish this clinical report. The study was approved by the Research Ethics Committee of the institution. This study was financed in part by the Coordenação de Aperfeiçoamento de Pessoal de Nivel Superior, Brazil (CAPES), Finance Code 001.

DISCUSSION

The OgS is commonly used to treat skeletal and dentofacial deformities by repositioning upper and lower jaws, improving aesthetics and solving functional problems. Once the injury of the marginal mandibular ramus of the facial nerve is a well-known complication of extraoral approaches to the mandibular ramus, intraoral approaches can minimize them and prevent complications such as facial nerve palsy. Nevertheless, patients with facial palsy after intraoral procedures have been reported in the literature.[1–5]

Most of the authors report that facial nerve palsy after OgS is a rare complication, with an incidence ranging between 0.10% and 0.75%. According to them, the facial nerve compression the most likely etiology, probably due to the close relationship between the posterior border of the mandibular ramus and the facial nerve in the open-mouth position adopted for BSSO.[3,6,13–15]

In addition, other possible etiologies for this condition include complete or incomplete nerve transection, nerve traction, and nerve ischemia caused by excessive injection of vasoconstrictor agents deep in the perimandibular region.[7] Besides that many other factors can lead to facial nerve injuries: unfavorable fractures (bad splits), infection, manipulation of instruments too far behind mandibular ramus, hematoma, edema, variations in the anatomic course of the facial nerve or compression by substantial postoperative swelling at the perimandibular level.[3,7,16] Also, unprotected osteotomy and slippage of cutting burs into perimandibular soft tissues during medial osteotomy can cause complete or incomplete nerve transection of the facial nerve trunk.[3,7,15]

It is important to distinguish between facial deficits due to central lesions and those due to peripheral lesions. The central type is characterized by partial paralysis, once the contralateral lower facial muscles are paralyzed, while orbicularis oculi, the corrugator supercilii and the frontalis muscles, which receive bilateral cortical fibers, continue to work. Therefore, if a person cannot frown, raise the eyebrow, open and close the eyes, it is probably not a central facial palsy. On the contrary, the peripheral palsy is subdivided into intrapetrosal and extrapetrosal types. A lesion in the intrapetrosal region usually produces, associated with motility loss, deficient taste sensitivity, and hyperacusia, while the extrapetrosal type is characterized by total paralysis with a lack of voluntary motility of all mimetic muscles ipsilateral to the lesion.[16,17]

The PFP is characterized by unilateral weakness or paralysis of facial expression muscles. It can cause asymmetry on the face and difficulty of eating, drinking and controlling salivary flow. The treatment of PFP is multidisciplinary must aim at the comfort and total recovery of the patient.[18]

When PFP is caused by infection, a pharmacologic treatment with antibiotics and antivirals should be chosen. In patients with compression of the facial nerve by postoperative edema (traumatic facial paralysis), one must wait for the movements to return gradually.[19] Treatment with systemic steroids and physiotherapy has also been recommended for facial nerve palsy after OgS as a mean of decreasing intraneural pressure and edema.[6] Timing of

initiation of steroid therapy plays an important role in enhancing nerve regeneration and a significantly better improvement can be expected when steroid treatment is begun early.[20,21]

Full recovery of nerve function can be expected in mild nerve injuries and physical therapy maneuvers such as facial massage and facial exercises performed twice a day also have been suggested. Connective tissue massage is also an effective adjuvant treatment to traditional physiotherapy.[6] Systemic steroids such as dexamethasone have been shown to play a role in nerve injuries.[21,22] Injection of vitamin B12 might also be beneficial in enhancing the recovery from facial nerve palsy.[23] Surgical exploration and repair are indicated only for severe injuries when facial nerve transection is noted.[6,24] The use of electrical stimulation has been linked to positive outcomes, and the type should depend on the pathology of the facial nerve.[15] Biofeedback, ultrasound therapy, acupuncture, and magnets have also been used in combination with physiotherapy, but their specific efficacy requires further investigation.[17]

Even though it is difficult to determine which of the possible etiologies actually caused the facial nerve palsy, some were raised as causative agents in the reported patient: The first hypothesis is that during the surgical procedure, when moving the proximal segment of the mandible after opening the sagittal osteotomy, it may have been taken too far left and posterior, compressing the facial nerve which is surrounded by the parotid gland. Compression of the facial nerve as a result of the positioning of the retractors on the medial side of the ramus up to the posterior edge can also be considered. Another theory is that facial nerve compression occurred from the substantial postoperative swelling at the perimandibular level. And a final possibility is a thermal shock, once the use of constant ice packs in the postoperative period directly on the face could also have been the causal factor, even though it was applied bilaterally.

According to the literature, it takes on average 2 to 3 months for complete recovery of muscular motility. However, there are some reports where patients took less (3–4 weeks) or much more time to completely recover facial movements (4 up to 12 months).[5,15,25,26]

Even though there is evidence of facial nerve palsy following bimaxillary surgery with mandibular advancement, this kind of complication seems to be rarer in class II than in class III patients who undergo mandibular setback.[17] Despite being transient and resolving spontaneously in almost all patients,[27] the assumption that postoperative facial nerve palsy always disappears is incorrect and the course of treatment should be handled with extreme care.[3,14]

The risk of facial nerve palsy after OgS should always be included in the informed consent considering that it is one of the most serious complications for a patient requiring correction of a dentofacial deformity, once their quality of life and social interaction are directly affected.[3] Also, its early evaluation should always be considered in the immediate postoperative period so the treatment can be started as soon as possible. Careful handling of fragments, as well as management of postoperative edema with medicines and protection against direct use of ice should be mandatory.

REFERENCES

1. Andriola FO, Kulczynski FZ, Deon PH, et al. Changes in cervical lordosis after orthognathic surgery in skeletal class III patients. *J Craniofac Surg* 2018;29:598–603
2. Kim SG, Park SS. Incidence of complications and problems related to orthognathic surgery. *J Oral Maxillofac Surg* 2007;65:2438–2444
3. Ruiz LP, Lara JC. Facial nerve palsy following bilateral sagittal split ramus osteotomy for setback of the mandible. *Int J Oral Maxillofac Surg* 2011;40:884–886
4. Jorge JJJ, Boldorini PR. Paralisia facial periférica. *Rev Fac de Ciênc Méd Sorocaba* 2005;7:9–14
5. Sammartino G, Califano L, Grassi R, et al. Transient facial nerve paralysis after mandibular sagittal osteotomy. *J Craniofac Surg* 2005;16:1110–1115
6. Hsu HA, Chang YC, Lee SP, et al. Myofascial pain syndrome may interfere with recovery of facial nerve palsy after orthognathic surgery - a case report. *J Oral Maxillofac Surg* 2012;70:e653–e656
7. Cousin GC. Facial nerve palsy following intra-oral surgery performed with local anaesthesia. *J R Coll Surg Edinb* 2000;45:330–333
8. Weber CR, Griffin JM. Evaluation of dexamethasone for reducing postoperative edema and inflammatory response after orthognathic surgery. *J Oral Maxillofac Surg* 1994;52:35–39
9. Zandi M. Comparison of corticosteroids and rubber drain for reduction of sequelae after third molar surgery. *Oral Maxillofac Surg* 2008;12:29–33
10. Danda AK, Ravi P. Effectiveness of postoperative antibiotics in orthognathic surgery: a meta-analysis. *J Oral Maxillofac Surg* 2011;69:2650–2656
11. House JW, Brackmann DE. Facial nerve grading system. *Otolaryngol Head Neck Surg* 1985;93:146–147
12. Baugh RF, Basura GJ, Ishii LE, et al. Clinical practice guideline: Bell's palsy. *Otolaryngol Head Neck Surg* 2013;149:S1–S27
13. Sakashita H, Miyata M, Miyamoto H, et al. Peripheral facial palsy after sagittal split ramus osteotomy for setback of the mandible - a case report. *Int J Oral Maxillofac Surg* 1996;25:182–183
14. de Vries K, Devriese PP, Hoving J, et al. Facial palsy after sagittal split osteotomies - a survey of 1747 sagittal split osteotomies. *J Craniomaxillofac Surg* 1993;21:50–53
15. Choi B-K, Goh RCW, Chen PKT, et al. Facial nerve palsy after sagittal split ramus osteotomy of the mandible: mechanism and outcomes. *J Oral Maxillofac Surg* 2010;68:1615–1621
16. Consolo U, Salgarelli A. Transient facial nerve palsy following orthognathic surgery: a case report. *J Oral Maxillofac Surg* 1992;50:77–79
17. Rai KK, Shivakumar HR, Sonar MD. Transient facial nerve palsy following bilateral sagittal split ramus osteotomy for setback of the mandible: a review of incidence and management. *J Oral Maxillofac Surg* 2008;66:373–378
18. Movérare T, Lohmander A, Hultcrantz M, et al. Peripheral facial palsy: speech, communication and oral motor function. *Eur Ann Otorhinolaryngol Head Neck Dis* 2017;134:27–31
19. Santos APN, Ganda AMF, Campos MIC. Relationship between facial paralysis and temporomandibular disorder: case report. *Rev Odontol UNESP* 2009;38:123–127
20. Al-Bishri A, Dahlin L, Sunzel B, et al. Systemic betamethasone accelerates functional recovery after a crush injury to the rat sciatic nerve. *J Oral Maxillofac Surg* 2005;63:973–977
21. Bracken MB, Shepard MJ, Collins WF, et al. A randomized controlled trial of methyl prednisolone or naxolone in treatment of acute spinal cord injury. Result of the second National Acute Spinal Cord Injury Study. *N Engl J Med* 1990;322:1405–1411
22. Kobayashi M, Costanzo RM. Olfactory nerve recovery following mild and severe injury and the efficacy of dexamethasone treatment. *Chem Senses* 2009;34:573–580
23. Jalaludin MA. Methylcobalamin treatment of Bell's palsy. *Methods Find Exp Clin Pharmacol* 1995;17:539–544
24. Jones JK, Van Sickels JE. Facial nerve injuries associated with orthognathic surgery: a review of incidence and management. *J Oral Maxillofac Surg* 1991;49:740–744
25. Palmen E. Facial nerve paralysis following a sagittal split osteotomy of the mandible. In: Proceedings of the 8th International Conference on Oral and Maxillofacial Surgery. Chicago, IL: Quintessense, 1985: 418–442
26. Behrman SJ. Complications of sagittal osteotomy of the mandibular ramus. *J Oral Surg* 1972;30:554–561
27. Westermark A, Bystedt H, von Konow L. Inferior alveolar nerve function after sagittal split osteotomy of the mandibule: correlation with degree of intraoperative nerve encounter and other variables in 496 operations. *Br J Oral Maxillofac Surg* 1998;36:429–433