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Facial Paralysis after Trauma: A Simplified Method to Find and Repair the Facial Nerve

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ABSTRACT

Objective
This study aims to identify the digastric nerve as an alternative to access the facial nerve in case of post-traumatic facial nerve paralysis.

Study design
Retrospective study.

Setting
Tertiary care hospital.

Subjects and Methods
Eleven peripheric facial paralysis cases between 2005-2016 following trauma to the parotid gland are presented. Initial emergency treatments were done elsewhere but all returned back to treat facial paralysis after 11-18 months to our institute. The digastric nerve was identified and a facial-hypoglossal nerve anastomosis was performed.

Results
Pre-operative House-Brackmann scores were 6 for all cases. Post-operative scores were between 3 and 4. Pre-operative needle electromyography revealed no motor unit action potentials; after 7 months post-operatively, we had motor unit action potentials in every case.

Conclusion
In cases of trauma and secondary approaches of the parotid gland, a safe step for finding the facial nerve is to find the digastric nerve and follow it through the main trunk.

Keywords
Facial nerve; Wound and injury; Hypoglossal-facial nerve anastomosis (HFNA).

Abbreviations
HFNA: Hypoglossal-Facial Nerve Anastomosis ; TMJ: Temporomandibular Joint; MUPs: Motor Unit Potentials; HB: House-Brackmann.

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INTRODUCTION

The facial nerve lies beneath the parotid gland and a thick glandular capsule serves as a protection against blunt trauma. The mandible is the second barrier for protection. The force required to injure the parotid gland is mostly more than the force required to fracture the mandible or temporomandibular joint (TMJ). In severe cases, the soft tissues are usually more affected than the adjacent structures, making surgical intervention necessary.[1-3]

Blunt trauma or gunshot injuries to the parotid gland may cause acute or delayed facial paralysis. Delayed treatment of the nerve is almost always difficult because the scar formation prevents visual identification or stimulation of the facial nerve or its branches.[4]

Postero-anterior or antero-posterior facial nerve dissections are the two common methods to find the facial nerve in the parotid surgery.[5] The tragal pointer or the tympanomastoid suture are the landmarks of the first method while the peripheral branches of the nerve are followed by the parotid gland in the latter one. Locating the nerve may be troublesome even in elective cases, whereas it is almost impossible in the delayed trauma cases because of extreme fibrosis. Hypoglossal-facial nerve anastomosis (HFNA) is a common method to repair the nerve once a long period of time has been passed from the onset of the trauma. Identification and access to both the facial and the hypoglossal nerves are mandatory in this procedure.[6]

We, in this study, present eleven cases of post-traumatic facial nerve paralysis that had end-to-end HFNA in our institute. To best of our knowledge, the digastric nerve is used as a referral point for the facial nerve but its importance in delayed trauma and fibrotic cases are underestimated. In this study, our aim is to emphasize the importance of identification of the digastric nerve in traumatic surgical cases as the first step of the facial neural surgery. Also a brief review of the literature and comparison of the pre-operative and post-operative facial nerve functions are introduced.

MATERIALS AND METHODS

Eleven peripheric facial paralysis cases between 2005-2016 following trauma to the parotid gland are presented. Institutional review board (IRB) approval was obtained from our institute. Initial emergency treatments were done elsewhere but all returned back to treat facial paralysis after 11-18 months. None of them received surgical intervention for the treatment of facial paralysis before or after the trauma. All cases underwent a thorough otolaryngologic examination and the pre-operative House-Brackmann (HB) scores were noted (Table 1). All cases received needle electrode electromyography to evaluate the motor function of the facial nerve (Table 1). The stimulus was given from the pretragal point and both the frontalis muscle and the *levator labii superioris/levator labii alaque nasi* muscles. After electrophysiological tests, all subjects were planned to receive surgical intervention.

All clinical subjects were operated under the condition of general anesthesia. Each patient received a dose of midazolam 1.5 mg intravenously for premedication. Nitrous oxide, oxygen, and remifentanil were used for induction of general anesthesia. No ad-

Table 1. *Subject Characteristics*

	Age/Gender	Duration of Facial Paralysis (Months)	Main Injury	Cause of Injury	Initial HB Score	Initial MUAP	Planned Surgery	Post-operative 7th month HB Scores	Post-operative 7th month MUAP
1	27 Male	16	Mandible fracture	Car accident	6	-	CN 7-12 Anastomosis	3	+
2	34 Male	11	Temporomandibular fracture	Gunshot	6	-	CN 7-12 Anastomosis	4	+
3	22 Female	13	Temporal bone fracture	Car accident	6	-	CN 7-12 Anastomosis	3	+
4	26 Male	18	Mandible fracture	Car accident	6	-	CN 7-12 Anastomosis	4	+
5	28 Male	16	Parotid region hematoma	Assault	6	-	CN 7-12 Anastomosis	4	+
6	29 Female	17	Mandible fracture	Car accident	6	-	CN 7-12 Anastomosis	4	+
7	31 female	15	Parotid region hematoma	Assault	6	-	CN 7-12 Anastomosis	4	+
8	22 Male	14	Mandible fracture	Gunshot	6	-	CN 7-12 Anastomosis	3	+
9	24 Female	15	Temporomandibular fracture	Car accident	6	-	CN 7-12 Anastomosis	4	+
10	28 Male	12	Temporomandibular fracture	Car accident	6	-	CN 7-12 Anastomosis	3	+
11	29 Male	12	Mandible fracture	Car accident	6	-	CN 7-12 Anastomosis	3	+

HB: House-Brackmann
CN: Cranial nerve
MUAP: Needle Electrode Electromyography Motor Unit Action Potentials

ditional muscle relaxant was given to enhance nerve stimulation. As a standard protocol, each patient was placed in the supine position with a shoulder support to the trauma side to facilitate surgery. Modified Blair incision was the type of incision we preferred to expose the parotid gland. Skin and subcutaneous tissues were elevated and the flap over the parotid gland was prepared. The anterior edge of the sternocleidomastoid muscle was identified and followed-up reaching the digastric muscle posterior belly and the mastoid apex. We preferred to use 2.5x surgical binoculars to ensure adequate magnification. Digastric nerve was identified on the posterior belly of the digastric muscle and under the parotid gland (Figure 1). It was followed-up until the main facial nerve trunk was reached. Meticulous dissection was carried out to remove all possible fibrous tissue or adhesions. In all our cases presented in this study, the facial nerve was transected near the main truncus. Therefore, end-to-end HFNA was planned for all patients. The hypoglossal nerve was found under the digastric muscle and sutured to the facial nerve trunk with 10/0 non-absorbable sutures. In the extreme fibrosis of the parotideal region, submandibulary fossa dissection was carried out to identify the neural structures.

Post-operative medications were considered as the standard for antibiotic regimens (cefuroxime axetil 500 mg b.i.d, 7 days), oral analgesics (acetazolamide 500 mg t.i.d, 7 days) and corticosteroids (prednisolone 1 mg/kg initially for 7 days and then decreased till completion).

All subjects were followed monthly for the first year, electromyographic evaluations were done in every three months and the HB scores were noted.

RESULTS

Seven cases were male and 4 cases were female. They were between 22-34 years old (Table 1). Seven cases were after car accidents, two were due to gunshot and two were after the assault. Eight had acute facial paralysis and the rest had delayed paralysis. All of them initially suffered from edema and hematoma.

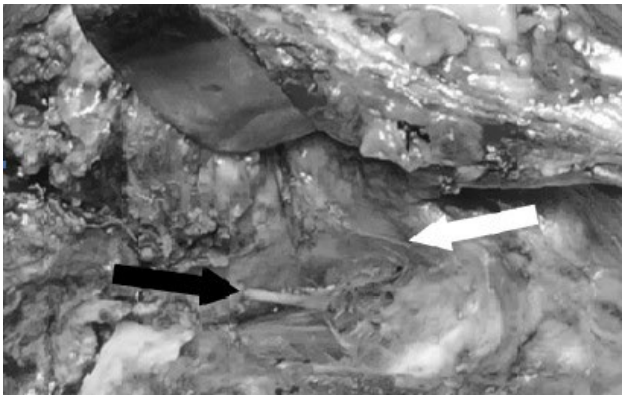
Pre-operative HB scores were 6 for all cases. Post-operative scores were between 3 and 4 (average 3,54). Pre-operative needle electromyography revealed no motor unit action potentials; after 7 months post-operatively, we had motor unit action potentials in every case. All cases are monitored in the first year after the surgery and no complications have occurred.

DISCUSSION

Trauma to the parotideal region rarely requires treatment. The extreme force that causes injury usually also causes penetration. Blunt trauma requiring emergency treatment may be caused by traffic accidents, severe falls, and criminal cases. Facial paralysis is the most important clinical presentation for cases of blunt trauma.[1]

The facial neural treatment plan is based on several factors. The timing of the surgery creates major differences. If the time elapsed from the transsection of the nerve is long enough to create fibrosis and neural degeneration, primary repair of the nerve may not be succeeded.[7] Secondly, physical examination of the neural integrity is crucial for optimal surgery, the HB scoring system is developed to uniform the function of the nerve; however, subjective judgment may not give enough or objective information on the paralysis status, especially with regard to the treatment and prognosis. Therefore, electrophysiological tests are supportive for the diagnosis of the neural-muscular system but not a method to diagnose the disease or confirm the etiology.[8] The aim of electrophysiological tests is to localize the lesion, determine the severity of the injury, and differentiate whether an injured nerve is still degenerating or regenerating. Cases with fibrillation potential and degeneration more than 90% should be treated surgically. The regenerative and repair processes of the nerve begin almost immediately, but nerve regeneration begins only after Wallerian degeneration. In severe cases; transection of the axon divides the nerve into proximal segment connecting to a cell body and a distal one. Two segments retract in the direction opposite to the transection site. The endoneurial tube is disrupted and axon cannot regenerate into its original sheath. Axonal regrowth begins as early as 24 hours post-injury. During regeneration, axonal regrowth may be impeded by fibrous tissues. Within 3 days post-injury, axonal sprout starts to grow from the proximal stump and the rate of axonal regeneration is generally estimated to be 1 mm per day. By 48 to 96 hours post-injury, axonal continuity is lost and nerve conduction is

Figure 1. *Intraoperatif view of the anatomic landarks. Black arrow: Facial nerve; White arrow: Digastric nerve*



lost. Myelin disintegration lags slightly behind that of axons but is well advanced by 36 to 48 hours. Disintegrated debris of axon and myelin sheath is removed by phagocytosis of macrophages within 12-14 days post-injury.[9]

In all of our cases, the earliest interventional time after the trauma was 11 months. Histologically, this is adequate for neural and structural fibrosis to occur. The HB scores and the neurophysiological tests revealed complete transection of the facial nerve on the trauma side. Therefore, once we confirmed the diagnosis by the physical examination and neurophysiological tests, we planned termino-terminal HFNA. The idea of HFNA is not to gain the facial muscular function completely but to gain the muscular tonus to some extent, although the cosmetic and functional results may not be satisfactory. Urgent surgical intervention on the time of the trauma, on the other hand, provides a better surgical field without no fibrosis or synechiae and primary repair or cable grafting of the facial nerve brings out better cosmesis and functional results.[10,11]

The middle deep temporal nerve branches can be identified on the deep side of the temporalis, within 9 to 12 mm posterior to the jugal point of the zygoma and utilized for facial reanimation. This technique may reanimate the facial muscles with independent activation.[12]

Experimentally, biodegradable metallic magnesium filaments, placed inside hollow nerve conduits, supported nerve repair by providing contact guidance support for axonal regeneration. With a nerve gap of 6 mm and 6 weeks post-repair, magnesium filaments had partially resorbed. Regenerating cells had attached to the filaments and axons were observed in distal stumps in all animals. Magnesium filaments were completely resorbed and no evidence of scarring was seen.[13]

Flasar et al studied 11 cases after HFNA; first movements were seen between 6 and 10 months after surgery in individual patients. The maximal improvement was achieved at 18 months. Motor unit potentials (MUPs) were first recorded after the 2nd month and present in all 11 patients 8-10 months post-surgery. Polyphasic regeneration potentials first appeared at 4-10 months post-surgery. The MUP amplitudes increased between the 3rd and 15th months after surgery to values of control muscles. The MUP duration was significantly increased above normal values between the 3rd and 24th months after surgery.[14]

Our electrophysiological results were consistent with the published previous data. However, we were unable to obtain the long-term results. The electrophysiological results were not totally parallel to the physical examination and HB scores, we were unable to achieve House-Brachmann scores higher than 3.

For treatment plan, we always preferred HFNA in late cases. The trauma was in the parotideal region, so we expected dense fibrosis in this region only. We found the digastric nerve over the digastric muscle, which could be accessed in several sites including the submandibular region, and as a step of the HFNA, also the hypoglossal nerve could easily be identified in this area of the neck and be prepared as well. These steps usually took 30 minutes; which we believe, is not a long time for safe and clear surgery. We did not attempt to use the other nerve finding methods because of the long duration of the paralysis and dense fibrosis. The posterior belly of the digastric muscle is innervated by the digastric branch of the facial nerve. The digastric nerve is the second branch of the facial truncus, following the posterior auricular nerve; it is also located on the surface of the digastric muscle.[10] Few studies have detailed methods to identify the facial truncus by following the digastric nerve. A study by Kanatas and McCaul[10] described this method, but no patient information was provided. In another study by Saha et al.,[11] this technique was introduced on cadavers and living patients. They reported that the facial truncus was 3.50-3.87 mm from the tympanomastoid suture line and 16.61-16.36 mm from the tragal pointer, whereas 7.41-8.03 mm from the digastric muscle. The authors claimed that the posterior belly of the digastric muscle was the point farthest from the truncus, making manipulation easier.

Experimentally, end-to-end and end-to-side HFNA neurorrhaphy methods are compared but all techniques resulted in partial functional recovery, but complete restoration was not obtained. There were no significant differences between the experimental groups in axon diameter or myelin thickness. The hypoglossal nerve fiber count after end-to-side was markedly more than that after end-to-end neurorrhaphy corresponding to the electrophysiological examination.[15] We preferred end-to-end (termino-terminal) method in all our surgeries, this may explain why the HB scores were no better than 3 post-operatively. We believe further research is needed to compare the long-term results of end-to-end or end-to-side HFNA.

The facial nerve truncus and its branches are in the most accessible state immediately after the trauma, and it is also possible to use primary suturing or nerve grafting to repair the nerve. As time passes, it is almost impossible to locate or repair the nerve, so mostly after the first year, a facial-hypoglossal nerve anastomosis required. After this surgery, hemiparalysis or paresis of the tongue and facial asymmetry occurs frequently.[9]

We have some limitations in this study; firstly, all cases have initially been treated elsewhere, it would notably be better if we could have assessed the cases from the onset of the trauma till the end of the post-operative first year. Secondly, we do not have the electrophysiological tests of the patients before they have applied to our institute. Finally, we monitored all cases for one year. Nevertheless, the aim of this manuscript is to present an easy method of facial nerve identification, so we believe, these limitations should be considered for further studies.

CONCLUSIONS

Access to the facial nerve is crucial in facial neural injury and parotid surgery. In cases of trauma and secondary approaches of the parotid gland, dense fibrosis and synechiae block the vision and the surgical field is vague. Alternatively, an unaffected site is chosen to find a branch of the facial nerve, which is the digastric nerve and

is followed by the main facial trunk. This method is not hard, the learning curve is easy and does not increase the overall operative time but a safe and clean dissection and structural identification could be performed.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

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