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Surgical rehabilitation of reversible facial palsy: Facial–hypoglossal network system based on neural signal augmentation/neural supercharge concept

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Double innervation;
Neural signal augmentation;
Neural supercharge

Summary To obtain symmetric appearance in facial palsy patients, it is important to retain any remaining potential of the compromised facial mimetic muscles. The purpose of the present study was to introduce surgical rehabilitation based on neural signal augmentation/neural supercharge concept for the treatment of reversible facial palsy patients. With construction of facial–hypoglossal network system using end-to-side neurorrhaphy technique, both facial and hypoglossal motor signals are provided to the compromised facial mimetic muscles. It is hypothesised that the remaining potential of incompletely or completely paralysed muscles without atrophy is activated by a neural 'supercharge' effect. To date, nine patients presented with reversible facial palsy have been treated by surgical rehabilitation with facial–hypoglossal network system in our institutes. Facial mimetic muscle function evaluated by the House–Brackmann grading system was improved from grade IV–VI to II–III in this series. The postoperative ENMG findings showed double innervation of the mimetic muscles supplied by the facial and hypoglossal donor motor sources. Hemiglossal dysfunction and mimetic muscle synkinesis associated with tongue motion were never seen with an average follow-up period of 21 months after surgery. This reconstructive concept offers a significant advantage for the treatment of the facial palsy patients with persistent incomplete type and reversible complete type without distinct mimetic muscle atrophy.

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The authors reported an alternative technique of facial nerve rehabilitation surgery based on double innervation of the paralysed mimetic muscle supplied by both facial and hypoglossal donor motor sources.[1] This is a new concept in the treatment of reversible facial palsy to obtain reliable restoration of resting tone and powerful movement from hypoglossal neural source as well as more physiological mimetic recovery from facial neural source. Creation of neural communications between facial and hypoglossal neural networks using end-to-side neurorrhaphy technique not only decreases possible functional damage to these motor donors, but also augments neural motor signals to the damaged facial neural plexus. From this experience, they have focused on the importance of augmentation of neural motor signal to the paralysed mimetic muscles in the treatment of reversible facial palsy patients. Herein the senior author's experience with nine consecutive facial palsy patients with persistent incomplete and reversible complete type who underwent surgical rehabilitation using facial–hypoglossal network system based on neural signal augmentation concept ('neural supercharge') is presented.

Patients and methods

From July 2000 to January 2005, nine patients who presented with reversible facial palsy were surgically treated by the senior author (YY). None had clinical evidence of facial mimetic muscle atrophy. The mean patient age at the time of surgery was 46 years, with a range from 15 to 68 years. There were five men and four women. The patient details are given in Table 1. The interval between the onset of the palsy and the timing of surgery was at least 12 months in incomplete palsy and within 24 months in complete palsy. No patients with incomplete palsy noted any signs of facial palsy recovery after adequate physiologic therapy and all requested surgical rehabilitation. The facial mimetic muscle function in these patients was evaluated before surgery and at postoperative 12-month/final follow-up visit to the outpatient clinic according to the House–Brackmann grading system (H–B GS) (Table 2).[2] Electroneuromyographic (ENMG) studies were carried out for the patients over 12 months after surgery. In this series, the follow-up period after surgery ranged from 12 to 63 months, with an average of 21 months.

Table 1 Etiology of patients presented with reversible facial palsy

Etiology of patients presented with reversible facial palsy	
Incomplete palsy due to Bell's palsy/Hunt syndrome	5
Incomplete palsy due to surgery for parotid gland	2
Complete palsy due to surgery for parotid gland: immediate reconstruction	1
Complete palsy due to surgery for acoustic tumour	1
Total	9

Surgical procedures

In this series, the facial reanimation surgery with construction of a facial–hypoglossal neural

Table 2 House–Brackmann's grading system from Ref[2]

Grade	Description	Characteristics
I	Normal	Normal facial function
II	Mild dysfunction	
	At rest	Normal symmetry and tone
	Motion	Eye: complete closure with minimum effort Mouth: slight asymmetry
III	Moderate dysfunction	
	At rest	Normal symmetry and tone
	Motion	Forehead: slight to moderate movement Eye: complete closure with maximum effort Mouth: slightly weak with maximum effort
IV	Moderately severe dysfunction	
	At rest	Normal symmetry and tone
	Motion	Forehead: none Eye: incomplete closure Mouth: asymmetric with maximum effort
V	Severe dysfunction	
	At rest	Asymmetry
	Motion	Forehead: none Eye: incomplete closure Mouth: slight movement
VI	Total paralysis	No movement

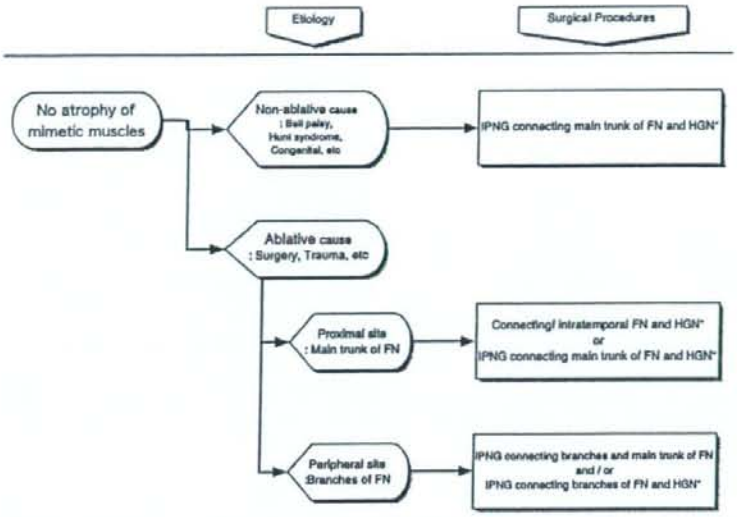

Figure 1 Algorithm for surgical rehabilitation utilizing facial–hypoglossal network system for reversible facial palsy patients. *IPNG, inter-positional nerve graft; FN, facial nerve; and HGN, hypoglossal nerve.

network system to produce double innervation to the paralysed mimetic muscles was carried out according to the algorithm for surgical rehabilitation of reversible facial palsy proposed by the authors' institutes (Fig. 1).

Five patients with persistent incomplete palsy caused by the Bell palsy or Hunt syndrome underwent inter-positional nerve graft (IPNG) connecting the main trunk of facial nerve (FN) and ipsilateral hypoglossal nerve (HGN). The great auricular nerve, approximately 40 mm in length, was grafted in an end-to-side fashion at both sites. End-to-side neurorrhaphy was carried out at the HGN site through lateral surface, on which 30% partial neurectomy was made (partial neurectomy technique) and at the main trunk of FN site through epineural window, which was created by the removal of the epineural sheath (epineural window technique). The grafted nerve was placed with the reverse style (Fig. 2).

Two patients with persistent incomplete palsy secondary to surgical resection of parotid gland underwent IPNG connecting the temporal, zygomatic, buccal and mandibular branches of FN and

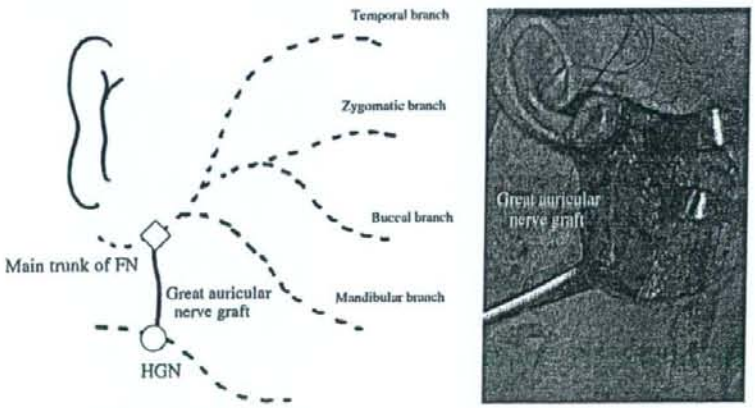

Figure 2 Inter-positional nerve graft connecting main trunk of facial nerve and ipsilateral hypoglossal nerve. (left) Schematic illustration. (right) Intraoperative view. FN, facial nerve; HGN, hypoglossal nerve; ○, partial neurectomy technique; and ◇, epineural window technique.

ipsilateral HGN. In these cases, the continuity of the zygomatic and buccal plexus had been restored with immediate nerve graft by the head and neck surgeon, however, persistent incomplete mid-face facial palsy remained. The sural nerve with four branches created by intraneural dissection under microscope[3] was grafted inter-positionally. End-to-side neurorrhaphy was carried out between the HGN and the proximal side of the graft nerve by partial neurectomy technique and between two branches of the graft nerve and the lateral surface of zygomatic and buccal branches without removal of the epineural sheath (no window technique). End-to-end neurorrhaphy was carried out between remaining two branches of the graft nerve and the temporal and mandibular branches, which were identified at the peripheral region (Fig. 3).

One patient underwent two IPNGs connecting all four branches of FN and both the main trunks of FN and ipsilateral HGN for immediate reconstruction of total facial nerve plexus after surgery to the parotid gland. Two sural nerve grafts with three branches each (created by intraneural dissection under microscope) were placed inter-positionally. In one graft, end-to-side neurorrhaphy was carried out between the HGN and the proximal side of the nerve graft by partial neurectomy technique and end-to-end neurorrhaphy was carried out between two branches of the nerve graft and the buccal and mandibular branches. In the other graft, end-to-end neurorrhaphy was carried out between the cut end of FN stem (main trunk of FN) and the proximal side of the nerve graft and end-to-end neurorrhaphy was carried out between two branches of the nerve graft and the temporal and zygomatic branches. Finally, the remaining third branch of two grafts was connected to each other stem of the grafts with end-to-side neurorrhaphy by epineural window technique (Fig. 4).

One patient with complete palsy secondary to surgery for acoustic neuroma underwent nerve crossover technique connecting the main trunk of FN and HGN and IPNG connecting FN stem and zygomatic and buccal branches of the contralateral FN. The details of this surgical procedure were described in Ref.[1]

Three types of the end-to-side neurorrhaphy included partial neurectomy technique, epineural window technique and no window technique were employed in this series. These techniques were applied to decrease the risk of injuring more axons according to size of the united nerve through its lateral side. Basically, partial neurectomy technique was applied for lateral surface of HGN, epineural window technique for main trunk of FN and grafted nerve, and no window technique for branches of FN.

Results

The postoperative course was uneventful in all patients. No facial nerve damage from the surgical procedures was recognized postoperatively. Physical therapy including mirror exercise supervised by the senior author alone was carried out during the follow-up period. The signs of mimetic muscle recovery started at 8.2 months in average ranging from 6 to 12 months after surgery. Facial

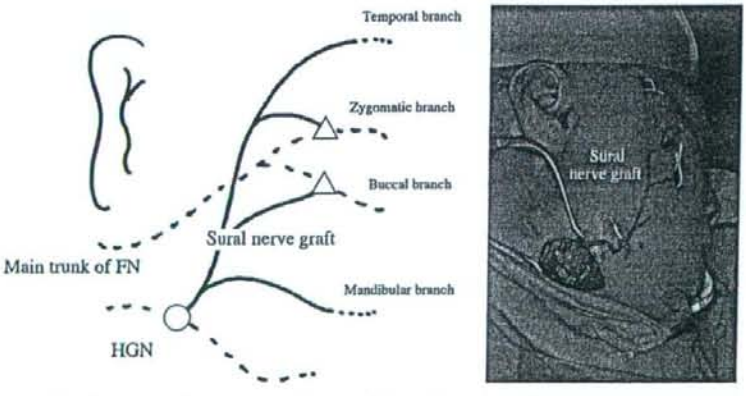

Figure 3 Inter-positional nerve graft connecting four peripheral branches of facial nerve and ipsilateral hypoglossal nerve. (left) Schematic illustration. (right) Intraoperative view. FN, facial nerve; HGN, hypoglossal nerve; ○, partial neurectomy technique; and △, no window technique.

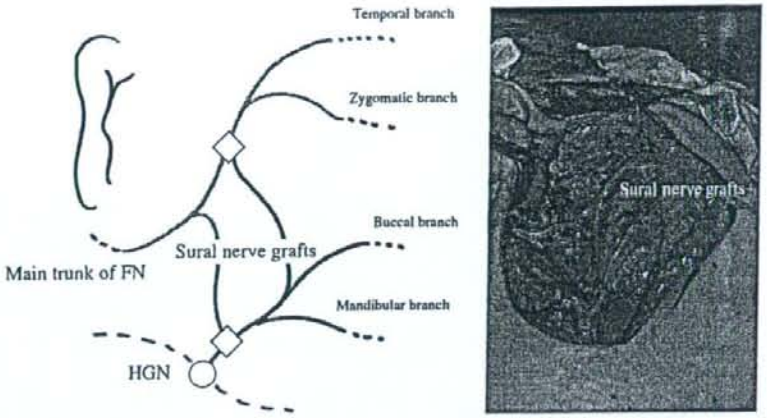

Figure 4 Two inter-positional nerve grafts connecting four peripheral branches of facial nerve and both of main trunk of facial nerve and ipsilateral hypoglossal nerve. (left) Schematic illustration. (right) Intraoperative view. FN, facial nerve; HGN, hypoglossal nerve; O, partial neurectomy technique; and ◇, epineural window technique.

mimetic muscle function evaluated by H–B GS was improved in all patients, from grade VI to II–III in one patient, from grade VI to III in one patient, from grade V to II–III in three patients, from grade V to III in three patients and from grade IV to II–III in one patient. Five patients with postoperative H–B GS II–III had ability to close the eye and move corners of mouth with minimal effort, but slight to no movement of forehead was achieved. Seven patients agreed to have postoperative ENMG studies assessed for facial–hypoglossal network conduction. Action potentials of the peripalpebral and perioral muscles on the paralysed side were obtained with the stimulation of the facial and hypoglossal nerve plexus used for neural motor source in all of these patients. The ENMG findings showed double innervation of the facial mimetic muscles supplied by the facial and hypoglossal neural system. In this series, all the patients have never shown occurrence of hemiglossal dysfunction resulted as dysphagia and speech problems and mimetic muscle synkinesis associated with tongue motion such as eye symptoms and oral incontinence during the follow-up period.

Case reports

Case 1: IPNG connecting main trunk of FN and ipsilateral HGN (see Fig. 2)

A 15-year-old man with minimal recovery from a right Bell's palsy requested surgical rehabilitation. He underwent IPNG connecting the right main trunk of FN and ipsilateral HGN using the right great auricular nerve at 15 months from onset of the palsy. All the surgery was carried out via one skin incision from the tragus to the mandibular notch area. The postoperative course was uneventful. Facial mimetic muscle function evaluated by H–B GS was improved from grade V to II–III. Postoperative 8-month ENMG studies showed double innervation of the peripalpebral and perioral muscles on the operated side. At 18 months after the surgery, he easily closed the eye and moved the corners of the mouth with minimal effort and obtained natural symmetric facial appearance without hemiglossal dysfunction (Fig. 5).

Case 2: IPNG connecting peripheral branches of FN and ipsilateral HGN (see Fig. 3)

A 50-year-old woman had radical total parotidectomy for carcinoma of the parotid gland followed by radiation therapy. Immediate nerve graft to reconstruct the zygomatic and buccal plexus was carried out by the head and neck surgeon. At 14 months after the surgery, she suffered from persistent incomplete palsy of the mimetic muscles supplied by zygomatic and buccal branches and complete palsy of the mimetic muscles supplied by temporal and mandibular branches. The sural nerve was grafted between the HGN and zygomatic and buccal branches with end-to-side fashion and temporal and mandibular branches with end-to-end fashion. Facial mimetic muscle function evaluated by H–B GS was improved from grade V to II–III and any facial muscle synkinesis was not noted. At 21 months after the surgery, she was satisfied with the results (Fig. 6).

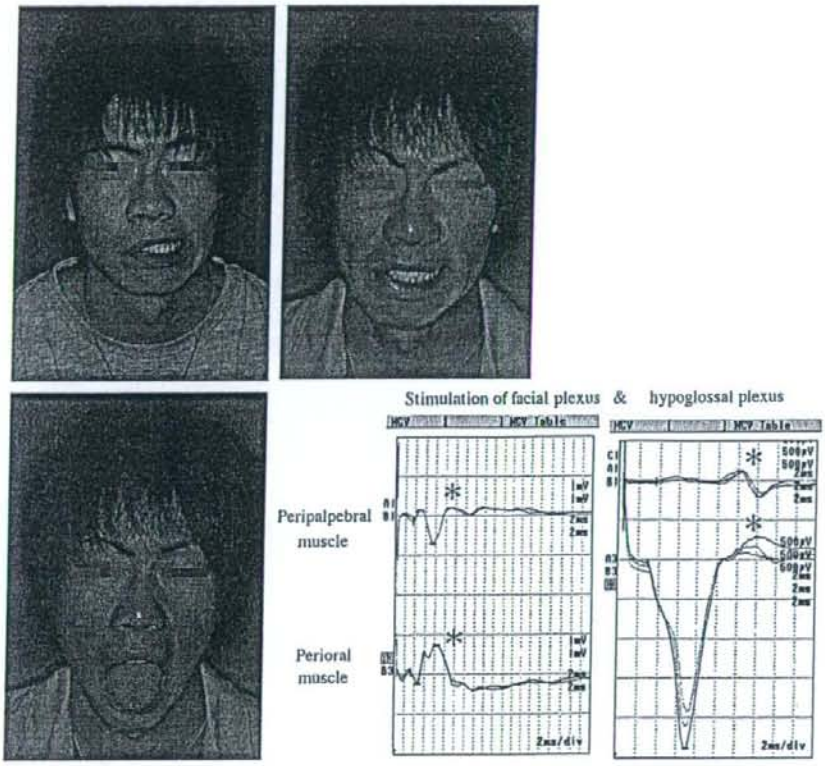

Figure 5 Case 1. (top, left) Preoperative appearance at smiling. (top, right) Appearance 17 months postoperatively at smiling. (bottom, left) No synkinesis of mimetic muscle and tongue atrophy. (bottom, right) Electroneuromyographic findings, 8 months after surgery. *s indicate action potentials obtained after the stimulation of the facial and hypoglossal nerve plexus.

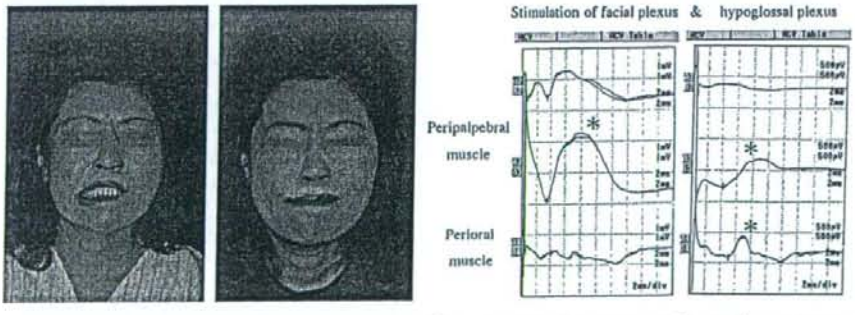

Figure 6 Case 2. (left) Preoperative appearance at smiling. (center) Appearance 13 months postoperatively at smiling. (right) Electroneuromyographic findings, 24 months after surgery. *s indicate action potentials obtained after the stimulation of the facial and hypoglossal nerve plexus.

Case 3: two IPNGs connecting peripheral branches of FN and both main trunk of FN and ipsilateral HGN (see Fig. 4)

A 23-year-old woman underwent the right radical total parotidectomy for carcinoma of the parotid gland and immediate reconstruction of total facial nerve plexus with two sural nerve grafts. The first one was grafted inter-positionally between the HGN and buccal and mandibular branches and the second one between the cut end of FN stem (main trunk of FN) and temporal and zygomatic branches. Finally, neural communication with two grafts was created using the each remaining branch with end-to-side fashion. The signs of mimetic muscle recovery started 8 months later. Double innervation of the peripalpebral and perioral muscles on the operated side was identified by postoperative 18-month ENMG studies. At 28 months after the surgery, facial mimetic muscle function evaluated by H–B GS was improved from grade VI to II–III and she exhibited good restoration of facial symmetry both at rest and animation without any complications in speech and mastication (Fig. 7).

Discussion

Patients who suffer from facial palsy demonstrate various clinical symptoms of asymmetric facial appearance. Loss of forehead wrinkles and ptosis of eyebrow and upper eyelid are caused by reduced frontalis and corrugator supercilii function supplied by the temporal branch. Ectropion of lower eyelid and inability to close the eye are caused by reduced orbicularis oculi function supplied by the zygomatic branch. Dropping of nasal ala and oral commissure and decreased nasolabial fold are caused by reduced nasalis, levator labii superioris, zygomatic major and minor, levator anguli oris and orbicularis oris function supplied by the buccal branch. Asymmetric lip movement is caused by reduced orbicularis oris, depressor anguli oris and depressor labii inferioris function supplied by the mandibular branch. In the surgical treatment of reversible facial palsy, it is most important to challenge taking out the remaining potential of these compromised facial mimetic muscles with maximum effort to obtain symmetric facial appearance. With surgical rehabilitation using facial–hypoglossal network system based on neural signal augmentation/neural supercharge concept, an alternative motor source from hypoglossal plexus is expected to activate the remaining potential of incompletely or completely paralysed mimetic muscles without atrophy, which were caused by supplied nerve injury.

In 1992 and 1994, Viterbo et al.[4–6] introduced the possibility of using end-to-side neurorrhaphy for the treatment of peripheral nerve pathologies. Definite reinnervation was obtained from the lateral face of a healthy nerve without impairment of its function in their experimental studies in rats. Recent bibliography offers many options for experimental models demonstrating occurrence of relevant nerve regeneration through end-to-side coaptation.[7–10] In the clinical fields, hypoglossal–facial nerve crossover or inter-positional nerve graft methods by use of end-to-side neurorrhaphy have been reported for the treatment of reversible complete facial palsy patients.[1,11–17] Furthermore, this type of neurorrhaphy has been applied for motor reinnervation of free functional muscle transplants and motor or sensory reinnervation of injured peripheral nerve in the upper extremity.[18–21] The technique of end-to-side neurorrhaphy takes an important role for the establishment of facial–hypoglossal network system in the surgical

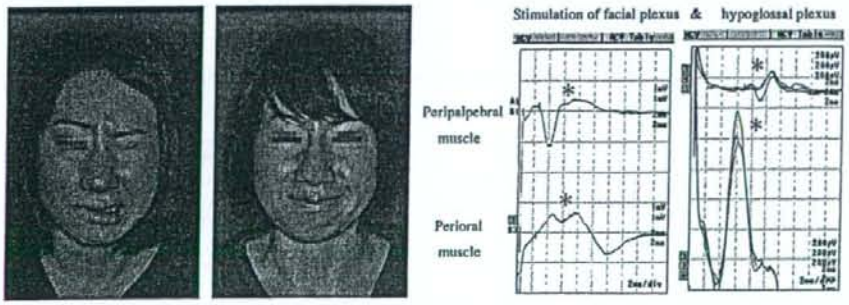

Figure 7 Case 3. (left) Appearance 1 month postoperatively at smiling and closing eye. (center) Appearance 30 months postoperatively at smiling. (right) Electroneuromyographic findings, 18 months after surgery. *s indicate action potentials obtained after the stimulation of the facial and hypoglossal nerve plexus.

rehabilitation of reversible facial palsy. More than one neural motor sources are provided to the compromised muscle without damage both on the originated and donor motor plexus with this technique.

Compared with the previous reports on the treatment of facial palsy patients using the end-to-side neurorrhaphy technique,[11–17] the facial–hypoglossal network system described here aims to organize double innervation on the compromised facial mimetic muscles effectively based on neural signal augmentation/neural supercharge concept. It is controversial that double innervation of a single muscle can be organized or not. In the authors' opinion, healthy muscle does not need any other additional motor neural signal, however, the compromised muscle with the lack of sufficient motor signal requires alternative motor source. In this series, all the patients undergoing surgical rehabilitation based on the neural signal augmentation concept showed significant improvement in facial symmetric appearance at rest as well as voluntary mimetic muscle movement. The postoperative ENMG studies also demonstrated that action potentials of the peripalpebral and perioral muscles on the operated side were noted after the stimulation of both facial and hypoglossal plexuses used as motor neural source. These findings of their clinical and electromyographic studies indicated the possibility of neural supercharge effect to the compromised muscle caused by supplied nerve damage and induction of double innervation of the reinnervated mimetic muscles.

The facial palsy patients treated by the application of hypoglossal motor source with end-to-side neurorrhaphy did not show facial muscle synkinesis associated with tongue motion in the previous literatures[11–17] or in this study. At present, the neuroanatomic basis for this facial–hypoglossal internuclear cooperation is not understood. In 2001, Popratiloff et al.[22] reported a direct bilateral projection of hypoglossal internuclear interneurons onto facial motor neurons by neuronal tracer studies in rats. According to their description, injections of retrograde tracers into the facial nucleus consistently labelled neurons in the hypoglossal nucleus. These findings have not been proved in the human study yet; however, facial–hypoglossal internuclear coordination may take a significant role not to occur synkinesis of facial mimetic muscle associated with tongue motion in surgical rehabilitation of facial palsy.

This is the first description on the surgical treatment of facial palsy patients presented with persistent incomplete type using the neural supercharge effect and reversible complete type using neural signal augmentation from both facial and hypoglossal donor motor sources to induce double innervation of the facial mimetic muscles. Although the authors' clinical science experience was not large and further clinical and basic science experiments concerning with motor neural source supplying the compromised muscle must be required, they believe that facial–hypoglossal network system based on neural signal augmentation/neural supercharge concept offers a definite innovation in surgical rehabilitation of reversible facial palsy.

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Alcaligenes xylosoxidans Cholecystitis and Meningitis Acquired during Bathing Procedures in a Burn Unit: A Case Report

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The information in this article was presented at the 37th Annual Meeting of the Japan Society of Burn, Nagoya, Japan, June 7–8, 2008.

Alcaligenes xylosoxidans, a nonfermentative, Gram-negative rod often found in aqueous environments, has been isolated from respirators, incubators, and disinfectant solutions in the hospital environment. It is known to cause disease in immunocompromised (eg, burn) patients and represents a cross-contamination risk related to wound care. In the authors' burn unit, two patients, admitted with deep dermal burns during a 1-month time period, acquired serious A. xylosoxidans infections. The first involved A. xylosoxidans-associated cholecystitis in an adult with 32% total body surface area (TBSA) burns and the second involved A. xylosoxidans meningitis in an adult with 30% TBSA burns. Both patients received hydrotherapy (bathing) in the same bathing tub, one patient after the other. Culture from environmental sources isolated A. xylosoxidans from the bathing mattress. Bacterial analysis of the isolates, including antimicrobial susceptibility testing and pulsed-field gel electrophoresis, suggested the patients had been infected by the same strain — ie, cross-contaminated — probably during treatment of their burns. The isolated strains were resistant not only to broad-spectrum penicillins and cephalosporins, but also to imipenem, to which past A. xylosoxidans strains have been susceptible. These findings underscore the need for strict infection control to prevent cross-contamination and disease outbreak.

KEYWORDS: burn wounds, Alcaligenes xylosoxidans, bathing, burns unit, cross-contamination

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In the hospital environment, *Alcaligenes xylosoxidans* has been isolated from respirators, incubators, and disinfectant solutions.[1,2] *A. xylosoxidans* infection is thought to occur mostly in immunocompromised patients and those with severe underlying disease conditions.[3,4] The majority of *A. xylosoxidans* strains are multidrug-resistant; thus, strict infection control is required to prevent spread of disease.[3,4] Two cases (one unusual because of the rarity of *A. xylosoxidans*-related cholecystitis) occurred within a 1-month period in patients who had sustained severe burns and had been treated in the burn unit of the authors' facility.

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Case 1. Mr. B, a 78-year-old man, sustained flame burns when his trousers accidentally caught fire in May 2007. He was immediately taken to the authors' burn unit. On initial examination, it was noted that Mr. B had sustained deep burn (DB) to both lower legs, comprising 8% of the total body surface area (BSA); deep dermal burn (DDB) to both thighs, comprising 14% of the total BSA; and superficial dermal burn (SDB) to both forearms and the face, comprising 10% of the total BSA (see Figure 1). A decompression incision was made in both lower legs. The next day, all DB and DDB tissue was surgically removed from the legs and skin grafting was performed. Seven days after surgery, Mr. B underwent hydrotherapy in a hospital bathing tub — 10 days later, he developed a high fever and severe infection in both lower legs. Below-the-knee amputation was performed emergently for both legs due to life-threatening sepsis. Mr. B's general condition improved over the next 2 weeks and he underwent bathing treatment every other day. Forty days after the injury, *A. xylosoxidans* was isolated from the residual wound, prompting immediate culture of environmental surfaces. *A. xylosoxidans* was isolated from the bathing mattress (see Figure 2). Clinical systemic and local inflammatory symptoms were not observed and the entire wound was resurfaced with free skin grafting.

Mr. B was discharged from the burn unit 10 weeks after admission but 1 week later, he developed a temperature of 39.1° C and stomach pain. An abdominal CT scan detected a swollen gallbladder and expanded bile duct (see Figure 3). Hematological studies revealed a white blood cell (WBC) count of 11.9×10^{9}/L and increases in C-reactive protein (CRP) (9.9 mg/dL), alkaline phosphatase (485 IU/L), and gamma-glutamyl transpeptidase (87 IU/L) levels, indicating severe inflammation of the gallbladder (cholecystitis). Percutaneous transhepatic biliary drainage was performed and the remaining bile was drained continuously though a drainage tube. *A. xylosoxidans* was isolated from the bile and treatment with imipenem was initiated but subsequently changed to tazobactam/piperacillin following the results of antimicrobial susceptibility testing. Mr. B's general condition

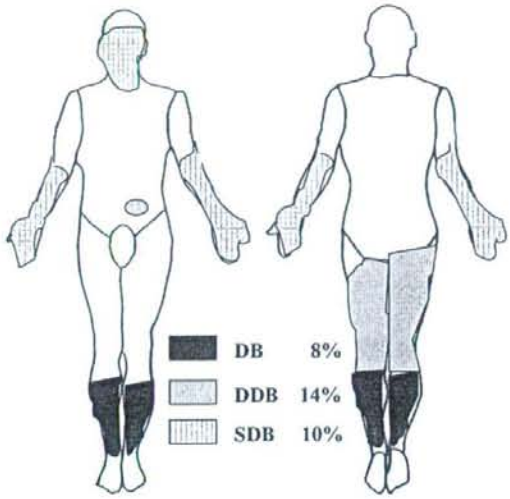


Figure 1. *Case 1: Burn area and depth.*

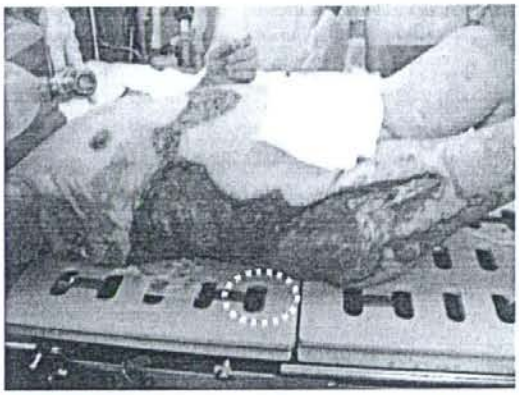


Figure 2. *Picture of bathing set up for hydrotherapy. A. xylosoxidans was isolated from an area of the bathing mattress within the dotted circle.*

KEY POINTS

- The authors describe two burn patients who developed serious infections, including cholecystitis and meningitis, following hydrotherapy.
- Culture results confirmed the presence of *Alcaligenes xylosoxidans* in the wound, spinal fluid, and bile of the patients and on the surface of the bathing mattress.
- The severity of the reported infections and resistance of isolates to previously effective antibiotics underscores the importance of strict infection control practices.

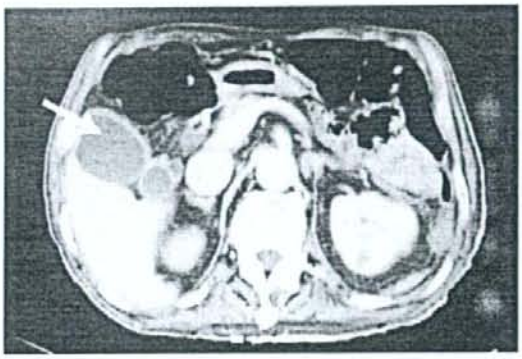


Figure 3. *Case 1: Abdominal CT scan 2 months after the injury. A swollen gallbladder due to cholecystitis was observed (arrow).*

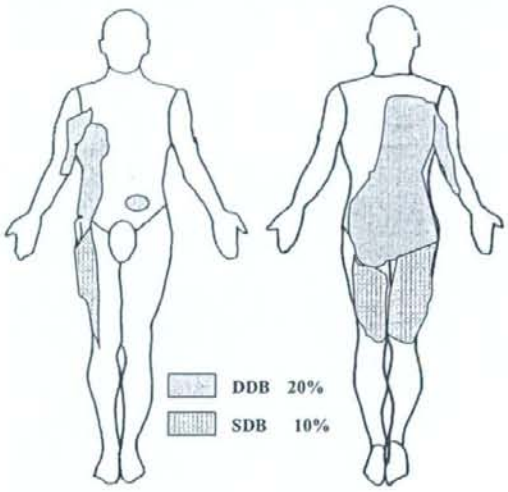


Figure 4. *Case 2: Burn area and depth.*

improved over the next 3 weeks and he was discharged 3 months after the injury.

Case 2. At the end of May 2007, 66-year-old Ms. K sustained scald burns when she accidentally fell over a bowl filled with boiling water. She was taken immediately to the authors' emergency unit. On initial examination, Ms. K had sustained DDB to the back and right upper arm, comprising 20% of the total BSA; and SDB to both thighs, comprising 10% of the total BSA (see Figure 4). Initial surgery was performed the next day to remove all DDB tissue on the back along with free skin grafting. Seven days after surgery, Ms. K received hydrotherapy in the hospital's bathing tub.

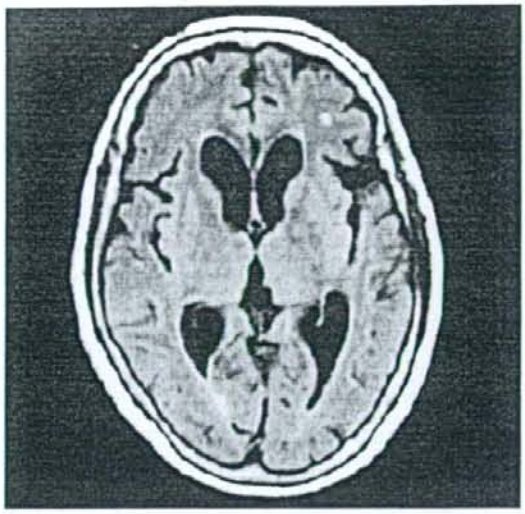


Figure 5. *Case 2: Brain CT scan 2 months after injury.*

Her general condition improved and she was discharged from the burn unit 2 weeks after the injury. The entire wound was resurfaced 10 weeks after the injury with another free skin graft.

However, 12 weeks after the injury, the patient developed a mental disorder (agitated and compromised ability to communicate) with a fever of 38.8° C and a slight headache. A CT scan detected a swollen ventricle (see Figure 5). Hematological studies revealed a WBC count of 13.3×10^{9}/L, and an increased CRP level (2.9 mg/dL), indicating bacterial meningitis. Ventricular drainage was performed, and *A. xylosoxidans* was isolated from the cerebrospinal fluid (CSF). Treatment with amikacin and ceftazidime was initiated and subsequently changed to tazobactam/pippracillin and ceftazidime after antimicrobial susceptibility testing. Four weeks later, recognizing the absence of *A. xylosoxidans* in the CSF, a ventriculo-peritoneal shunt was inserted. Ms. K's general condition improved and she was discharged 5 months after the injury.

Figure 6 shows the clinical course of both patients.

Three isolates of *A. xylosoxidans*, including those from the wound and bile of case 1, the CSF of case 2, and environmental sources, were subjected to antimicrobial susceptibility testing and pulsed-field gel electrophoresis (PFGE) and the results were found to be similar (see Table 1 and Figure 7). The

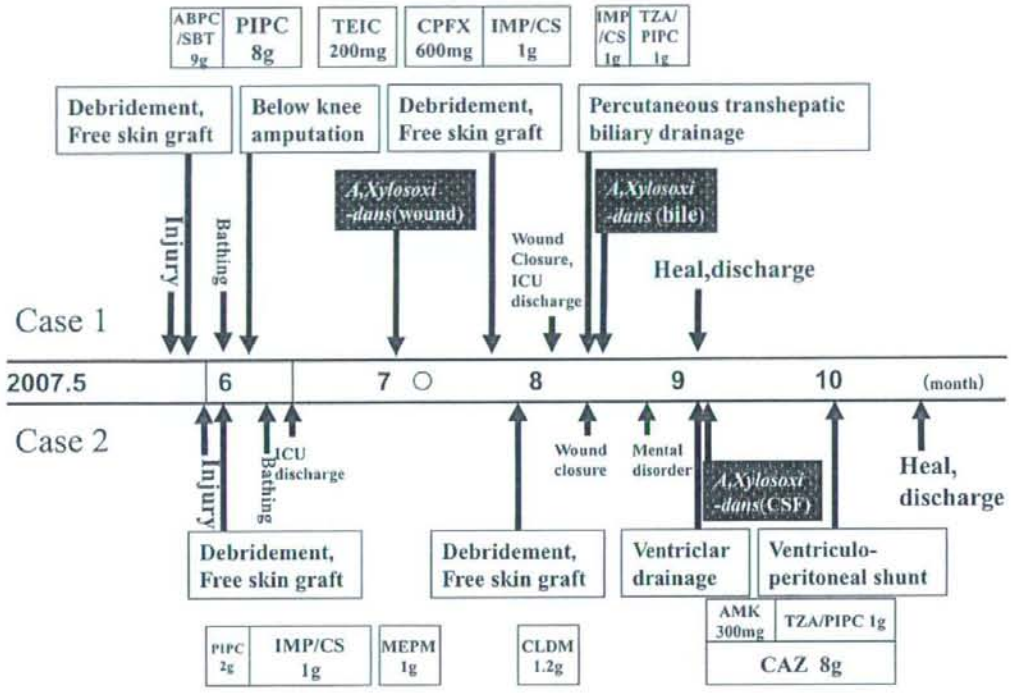


Figure 6. *Clinical course of patients. ABPC/STB = ampicillin/sulbactam; PIPC = pippracillin; TEIC = teicoplanin; CPFX = ciprofloxacin;, IPM/CS = imipenem/cilastatin; TAZ/PIPC = tazobactam/pippracillin; MEPM = meropenem; CLDM = clindamycin; AMK = amikacin; CAZ = ceftazidime; ICU = intensive care unit*

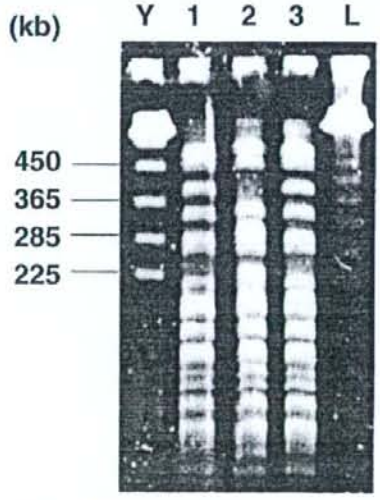
isolates were resistant to almost all antibiotics, including ceftriaxone, cefpodoxime proxetil, cefepime, cefozopran, flomoxef sodium, imipenem, meropenem, aztreonam, entamicin, tobramycin, amikacin, minocycline, and ciprofloxacin; the only antibiotics to which all isolates were uniformly susceptible was tazobactam/pippracillin.

Three isolates from the environmental sources — the CSF of case 2 and the bile of case 1 — were subjected to PFGE of Xba I-genomic DNA to determine their clonal homology (see Figure 7). The PFGE fingerprints of two isolates (Lane 1, environmental sources; and Lane 3, bile of case 1) showed identical patterns. Also, their PFGE fingerprints were closely related to that of Lane 2 (CSF of case 2).

A. xylosoxidans is a nonfermentative, Gram-negative rod often isolated from aqueous environments and known to cause disease in immunocompromised patients. Little has been reported about its pathogenicity in humans, except in cases involving children.[2,5] Reports of CSF infection by *A. xylosoxidans* are even rarer.[6] In case studies, Kumar et al[4] reported that two strains of *A. xylosoxidans* were isolated from CSF, Boukadida et al[7] documented a case of *A. xylosoxidans*-associated neonatal meningitis transmitted by aqueous eosin, and D'Amato et al[8] reported a case of *A. xylosoxidans* meningitis related to a gunshot wound. No case reports describing cholecystitis due to *A. xylosoxidans* were found in the literature.

Burn wound infection is common and difficult to control because cutaneous surfaces are without protective barriers; thus, burn patients may easily acquire bacterial infection, including *Staphylococcus aureus, Pseudomonas aeruginosa, Escherichia coli, Legionella pneumophila, Aerobacter aerogenes, Proteus vulgaris,* and *A. xylosoxidans*.[9-11] Several investigators have reported *A. xylosoxidans* in patients with underlying diseases, including malignancies, cardiac disease, and

	Case 1 wound	Case 1 bile	Case 2 CSF
Pippracillin	I	I	I
Ampicillin	R	-	-
Tazobactam/pippracillin	-	S	S
Ceftriaxone	-	R	R
Ceftazidime	I	I	I
Cefpodoxime proxetil	I	R	R
Cefoperazone	-	I	I
Cefepime	R	R	R
Cefozopran	-	R	R
Flomoxef sodium	R	R	R
Imipenem	R	R	R
Meropenem	R	R	R
Aztreonam	R	R	R
Gentamicin	R	R	R
Amikacin	R	R	R
Tobramycin	-	R	R
Minocycline	R	R	S
Ciprofloxacin	-	R	R
Levofloxacin	R	R	I



Y: Yeast chromosomes, *Saccharomyces cerevisiae*
L: Lambda ladders

Figure 7. *Pulsed-field gel electrophoresis banding patterns of XbaI-genomic DNA from three isolates of A. xylosoxidans. Lane 1, environmental sources; Lane 2, Case 2 CSF; Lane 3, Case 1 bile.*

immunosuppression.[4,5,12] Burn patients also have a high risk of infection because severe burns lead to a compromised immune system, which may cause life-threatening general infections, including sepsis, meningitis, and cholecystitis from common burn wound infections. The results of the susceptibility tests and PFGE patterns of all isolates were similar, suggesting that both patients acquired the organism due to cross-contamination of the same strains of *A. xylosoxidans* from the hospital environment — specifically, from the bathing mattress.

Results of *in vitro* susceptibility studies of the isolates have found that *A. xylosoxidans* is a multidrug-resistant organism. Legrand et al[5] studied susceptibility in 26 blood cultures from 10 patients with *A. xylosoxidans* infections between 1983 and 1988 and reported that the isolates were susceptible to trimethoprim-sulfamethoxazole, as well as the antipseudomonal penicillins, ceftazidime, cefoperazone, and imipenem. In 1996, Duggan et al[12] performed susceptibility studies in 11 cases of *A. xylosoxidans* bacteremia and found that all were susceptible to broad-spectrum penicillins, imipenem, ceftazidime, and trimethoprim-sulfamethoxazole. Aisenberg et al[13] investigated 46 patients with hematogenous *A. xylosoxidans* infection between 1989 and 2003, reporting that most isolates exhibited susceptibility to carbapenems, antipseudomonal penicillins, and trimethoprim-sulfamethoxazole. In 2003, Gomem-Cerezo et al[14] reviewed 44 cases of *A. xylosoxidans* bacteremia diagnosed over a 10-year period and concluded that antibiotic therapy with antipseudomonal penicillins or carbapenems would be a reasonable choice. Based on the authors' review of current drug-susceptibility data, *A. xylosoxidans* appears to have become resistant to novel antibiotics to varying degrees, reducing the number of effective antimicrobial agents. Antipseudomonal penicillins and carbapenems have maintained their effectiveness against the organism until recently. However, strains of *A. xylosoxidans* isolated from the authors' patients were found to be resistant not only to almost all antibiotics, including broad-spectrum penicillins and cephalosporins, but also to imipenem, to which past *A. xylosoxidans* strains were susceptible. Only tazobactam/pippracillin maintained their effectiveness against strains of *A. xylosox-*

idans isolated from the authors' patients and could improve their conditions.

From the authors' experience, including the two case studies presented, as well as information in the literature, *A. xylosoxidans* isolates should be considered as a possible etiology of infection in patients with extensive burn injuries. Various isolates were found to be multidrug-resistant but tazobactam/pipracillin still appear to be effective against the organism. Strict infection control practices are required to prevent cross-contamination and disease outbreaks in acute care facilities because, in addition to the case reported here, the organism has been cultured from several aqueous environments and needs to be monitored.

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Reconstruction of Velopharyngeal Competence for Composite Palatomaxillary Defect With a Fibula Osteocutaneous Free Flap

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Abstract: Velopharyngeal competence reconstruction is indispensable for acquiring the fine speech and ingesting function. However, the maxillary prosthesis becomes unstable in some patients who have undergone extensive palatomaxillary. We present a case of total palatomaxillary defect resulting from squamous cell carcinoma ablation of the palate, which was reconstructed using a fibula-free osteocutaneous flap. Velopharyngeal competence was reconstructed owing to the flap so that the patient could ingest a soft diet and speak without hypernasality 2 weeks after surgery.

Key Words: Total palate defect, velopharyngeal competence, fibula-free osteocutaneous flap

Reconstruction of large maxillary defects is challenging for the reconstructive surgeon. In advanced cases, velopharyngeal competence reconstruction is indispensable for acquiring fine speech and ingesting function.

We present a case of total maxillary and palate defect reconstruction resulting from squamous cell carcinoma ablation.

Case Report

A 74-year-old woman consulted our hospital complaining of palatal neoplasm of 6 month's duration. On examination, a 3.0 × 5.5-cm, hard, round, protuberant tumor was found on the palate (Fig 1). Magnetic resonance imaging showed that the mass had destroyed the palatal bone and invaded the nasal cavity (Fig 2). Histological analysis of a biopsy indicated squamous cell carcinoma.

Surgery consisted of radical excision of the palatal tumor along with palatomaxillary reconstruction using a fibula osteocutaneous free flap. Bilateral maxillary bone was osteotomized, and en bloc resection of palate was performed (Figs 3A, B). An osteocutaneous flap was elevated from the left leg with a 10-cm fibula and a 15 × 5-cm elliptical skin paddle based on the peroneal vessels in a standard manner.

The bone segment was inserted into the maxillary defect to reconstruct the inferior piriform aperture (Fig 4). The skin island was divided into 2 portions and used for oral and nasal side reconstruction. Consequently, the bone segment was sandwiched between 2 flaps (Fig 3C). The peroneal artery

Fig 1 Preoperative view of the palatal tumor. A 3.0 × 5.5-cm, hard, round, protuberant tumor covered with a necrotic tissue was found on the palate.

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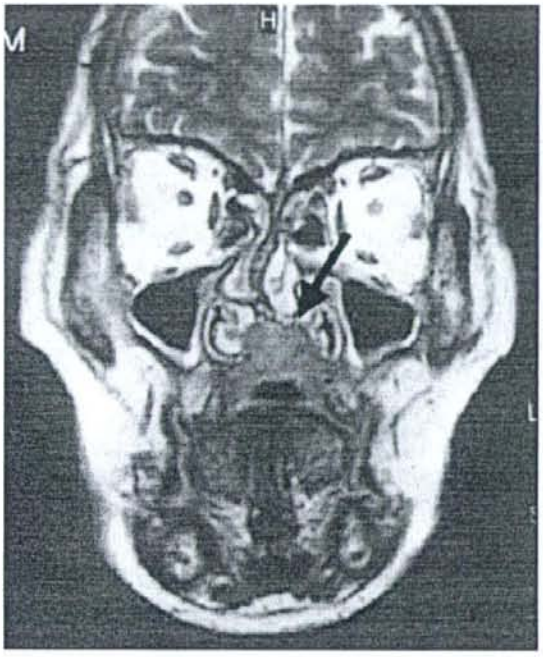

Fig 2 Magnetic resonance imaging showed that the mass had destroyed the palatal bone and invaded the nasal septum and nasal cavity (arrow).

was connected with the facial artery, and each peroneal vein was connected with external jugular and the branch of the internal vein.

The viability of the skin flap was good, and a bone scan revealed good uptake in the transferred fibula bone (Fig 5). The patient could orally ingest a soft diet without back current to the nasal cavity (Fig 6). After 2 months, the patient told through a little hypernasality, which was evaluated, that the fricative [s], [ʃ] was good, the explosive [d], [t], [k] was also good, but [b], [p] was poor. The cause of this speech disturbance might have been incomplete closure of mouth because of short midface and upper lip's sensory disturbance.

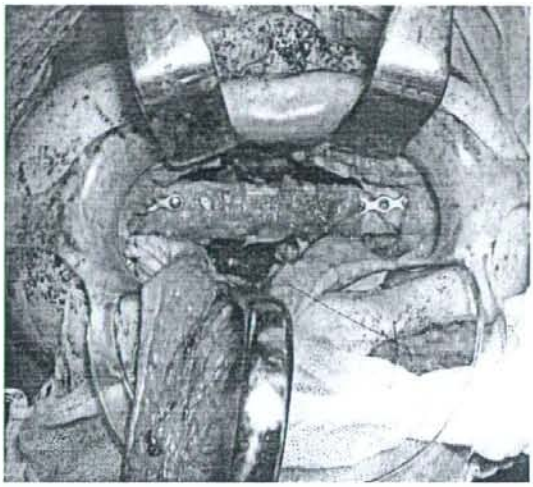

Fig 4 The fibula bone segment was transferred and fixed to the maxilla with titanium miniplates.

The patient intended to wear full dentures after the insertion of an osseointegrated dental implant onto the transferred fibula. However, she died of relapse of neck lymph node metastasis 4 months after surgery.

Discussion

The primary goal of palatomaxillary reconstruction is restoring velopharyngeal competence.[1] In

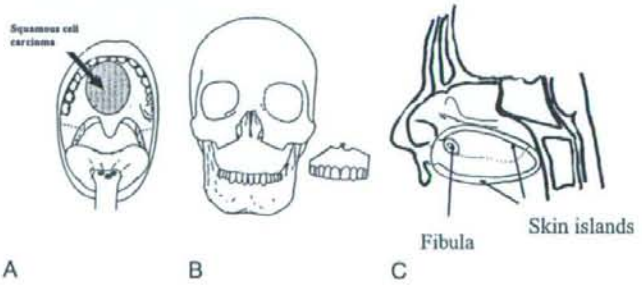

Fig 3 Schematic drawings of resection and reconstruction. Extent of palatomaxillary resection (A). Maxillary bone was osteotomized following the Le-Fort I osteotomy design, and the soft and hard palates were resected (B). The transferred fibula was sandwiched between 2 flaps (C).

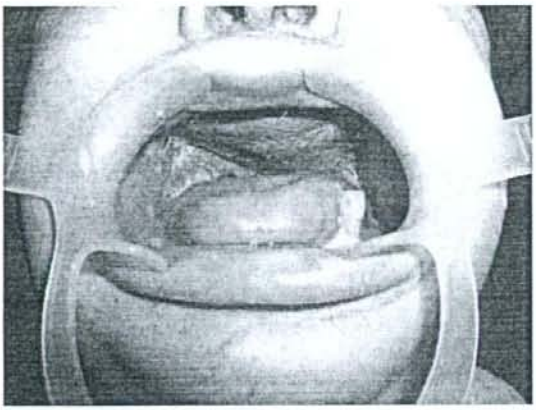

Fig 5 A 1-month postoperative view of the flap. The viability of the skin flap was good without infection or necrosis.

our case, palatomaxillectomy was necessary for oncologic clearance, and microvascular free flap reconstruction was advocated as a more desirable treatment option.[2]

Okay et al[3] indicated that defects after palatectomy, which left no dentition for the retention of an obturator, required vascularized bone-containing free flaps. Velopharyngeal competence function was simultaneously restored by transferring bone-containing free flaps, which enabled the patients to speak and swallow immediately after surgery, and allows for the placement of osseointegrated implants

Several osteocutaneous flaps are available for reconstruction of palatomaxillary defects.[4,5] Among these flaps, an osteocutaneous fibula free flap is a good option for the reconstruction of three-dimensional composite maxillary defects.[5,6] It provides a long straight bone, and the skin island can be harvested large enough for resurfacing of the palatal defect.[5]

Favorable reconstruction using a vascularized flap enables the early restoration of velopharyngeal competence function.

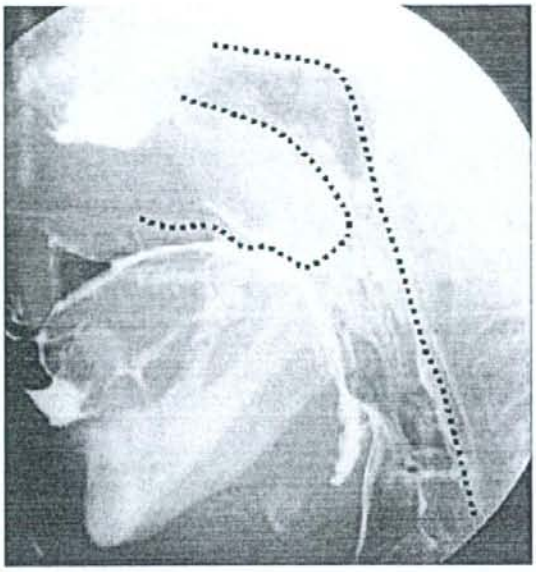

Fig 6 Pharyngeal fluoroscopy 1 month after surgery revealed that the patient could ingest a barium meal without back current to the nasal cavity.

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Upper lip pressure ulcers in very low birth weight infants due to fixation of the endotracheal tube

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KEYWORDS
Pressure ulcers;
Upper lip;
Five very low birth weight infants;
Endotracheal tube

Abstract Pressure ulcers of the upper lip developed in five very low birth weight infants due to fixation of the endotracheal tube. These ulcers left marked scars on the lip, and one patient required revision surgery. All cases occurred in infants whose birth weight was less than 1200 g, and whose gestational age was less than 29 weeks. We investigated these unusual ulcers and concluded that they were not caused by simple pressure but by a shearing force to the lips during fixation of the endotracheal tube. To prevent such pressure ulcers, a new method to stabilize the endotracheal tube is presented in which a flexible laminated arch is laid across the mouth and held to both cheek with adhesive tape and to which the intubation tube is tied.

We warn about the possible complication and recommend this method to prevent undesirable iatrogenic incidents, especially for very low birth weight neonates.

Introduction

A total of 589 neonates were treated in the neonatal intensive care unit (NICU) of the National Organization Nagasaki Medical Center in 2006 and 2007. Of these infants, 110 were very low birth weight (VLBW) infants, who often require long-term intubation and mechanical respiratory support.

In the present paper, we report on pressure ulcers of the upper lip that developed in five VLBW infants due to fixation of the endotracheal tube. We warn about this possible complication and introduce a method for preventing these undesirable incidents.

Cases

All patients required emergent intubation immediately after birth because of severe respiratory disturbance usually caused by respiratory distress

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syndrome (RDS). The endotracheal tube was stabilized with a strip of adhesive tape that circled the endotracheal tube and was attached to the skin of both halves of the upper lip and sometimes extended to the cheek.

Case 1. A 990-g baby boy was born at 26 weeks' gestation with RDS caused by neonatal asphyxia and underwent emergent intubation and received mechanical ventilation. Extubation was performed 56 days after birth, and a scar measuring 7 × 2 mm and crossing the vermillion border was found on the right half of the upper lip. This scar remained as a depressive deformity, and caused contraction and irregularity even after 1 year (Fig. 1).

Case 2. A 525-g baby boy was born at 26 weeks' gestation with RDS caused by immature lungs, and underwent emergent intubation and received mechanical ventilation. A pressure ulcer measuring 7 × 2 mm developed on the right half of the upper lip and was found when the intubation tube was changed 40 days after birth. The ulcer was conservatively treated with ointment. The wound healed within 7 days, but a depressive scar and notch deformity remained even after 3 years (Fig. 2).

Case 3. An 830-g baby girl was born at 26 weeks' gestation with RDS caused by neonatal asphyxia and underwent emergent intubation and received mechanical ventilation. A pressure ulcer measuring 5 × 1 mm developed on the right half of the upper lip and was found when the adhesive tape was changed 61 days after birth. The ulcer was conservatively treated with ointment and healed within 5 days, but a flat scar and de-pigmentation remained even after 1 year (Fig. 3).

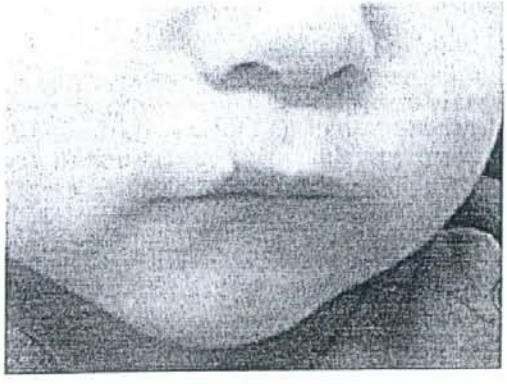

Fig. 1 Case 1. One year after the development of the pressure ulcer. A marked scar remained on the right upper lip which crosses the vermillion border and forms contraction and irregularity.

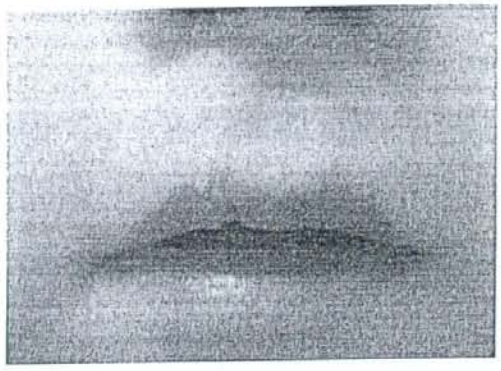

Fig. 2 Case 2. A depressive scar and notch deformity remained even after 3 years.

Case 4. A 660-g baby girl was born at 24 weeks' gestation with RDS caused by neonatal asphyxia who underwent emergent intubation and received mechanical ventilation. Two pressure ulcers measuring 6 × 2 mm and 7 × 2 mm developed on the each half of the upper lip and were found when the adhesive tape was changed 4 weeks after birth. The ulcers were conservatively treated with ointment and healed within 7 days, but bilateral depressive scars and notch deformities remained even after 1 year (Fig. 4). Scar revision surgery was performed 2 years after the pressure ulcer developed.

Case 5. A 1130-g baby boy was born at 28 weeks' gestation with RDS caused by neonatal asphyxia and underwent emergent intubation and received mechanical ventilation. A pressure ulcer measuring 4 × 1 mm developed at the center of the upper lip, and was found when the adhesive tape was changed 28 days after birth. The ulcers

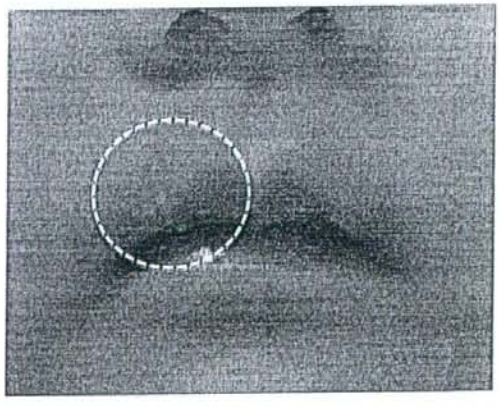

Fig. 3 Case 3. A flat scar and de-pigmentation remained even after 1 year.