Scandinavian Journal of Plastic and Reconstructive Surgery and Hand Surgery
Publication details, including instructions for authors and subscription information:
http://www.informaworld.com/smpp/title~content=t713691514

EVALUATION OF MUSCLE GRAFT USING FACIAL NERVE ON THE AFFECTED SIDE AS A MOTOR SOURCE IN THE TREATMENT OF FACIAL PARALYSIS

Kazuki Ueda; Kiyonori Harii; Hirotaka Asato; Kotarou Yoshimura; Atsushi Yamada

Online Publication Date: 10 March 1999

To cite this Article Ueda, Kazuki, Harii, Kiyonori, Asato, Hirotaka, Yoshimura, Kotarou and Yamada, Atsushi(1999)'EVALUATION OF MUSCLE GRAFT USING FACIAL NERVE ON THE AFFECTED SIDE AS A MOTOR SOURCE IN THE TREATMENT OF FACIAL PARALYSIS',Scandinavian Journal of Plastic and Reconstructive Surgery and Hand Surgery,33:1,47 — 57

To link to this Article: DOI: 10.1080/02844319950159622

URL: http://dx.doi.org/10.1080/02844319950159622

Scand J Plast Reconstr Hand Surg 33: 47–57, 1999

EVALUATION OF MUSCLE GRAFT USING FACIAL NERVE ON THE AFFECTED SIDE AS A MOTOR SOURCE IN THE TREATMENT OF FACIAL PARALYSIS

Kazuki Ueda,[1] Kiyonori Harii,[2] Hirotaka Asato,[2] Kotarou Yoshimura[2] and Atsushi Yamada[3]

From the [1]Division of Plastic Surgery, Jichi Medical School, Tochigi, [2]Department of Plastic Surgery, University of Tokyo, Tokyo, [3]Division of Plastic Surgery, University of Tohoku, Sendai, Japan

(Accepted for publication 20 January 1998)

Abstract. To acquire symmetry of the cheek when smiling, we carried out 39 free vascularised grafts of the muscle, the motor nerve of which was sutured to a stump of the ipsilateral facial nerve, for 39 patients with facial paralysis. We used two methods: an as healthy and fresh as possible facial nerve stump (method 1A, $n = 17$), or an incompletely affected stump (method 1B, $n = 22$). The results are classified into grade 1 to 5 indicating increasing efficiency of muscle function. All patients who had method 1A and 14 patients who had method 1B were evaluated grade 4 or better. Both an incompletely affected facial nerve stump and the proximal stump of a facial nerve that had previously been resected have sufficient function to provide contraction in the grafted muscle.

Key words: facial paralysis, muscle graft, affected facial nerve.

Microneurovascular free muscle graft, originally developed by one of the authors (KH) for the treatment of irreversible facial paralysis, where a primary ipsilateral nerve repair is impossible, has become a widely accepted procedure for dynamic smile reconstruction (12). The important factor in obtaining favourable results from this procedure, however, is selection of a suitable motor nerve in the recipient bed which is capable of innervating the grafted muscle and producing a natural or near-natural smile.

Many pioneering works (10, 13, 19, 20, 27) have led to championing of the two-stage procedure that features a cross-face nerve graft in the first stage and a free muscle graft in the second stage. The cross-face nerve graft can instigate development of functional facial nerve axons from the contralateral, non-affected facial nerve. The main drawbacks of this procedure, however, are that it is time-consuming and requires a sural nerve graft, which can result in paraesthesia or hypoaesthesia of the lateral foot. The numbers and sizes of regenerated myelinated axons at the distal end of a long cross-face sural nerve graft are usually reduced (11), which sometimes causes poor reinnervation of a grafted muscle.

Free muscle graft, in which the motor nerve is sutured to a stump of the ipsilateral facial nerve, however, enables the graft to be inserted in a single stage. In optimal cases, near-natural animation can be obtained because the grafted muscle is innervated by the ipsilateral facial nerve itself (9). In this paper we report our experience of dynamic smile reconstruction of a paralysed face using microneurovascular free muscle graft with nerve suture to the ipsilateral facial nerve stump.

Operative technique

The cheek skin is widely undermined through a preauricular face lift incision to accept the subsequent muscle graft (12). The recipient vessels and nerve are prepared. A muscle segment of required size (usually about 3.5 cm wide, 8 cm long, and 1 to 1.5 cm thick at rest for normal adult Asian face) is introduced between the nasolabial fold and the zygomatic arch as previously reported elsewhere (11, 12). The nutrient vessels of the grafted muscle segment are anastomosed to the superficial, temporal, or the facial vessels. If these vessels are not available, the superficial thyroid vessels and others can be used.

The method varies depending on the quality of the facial nerve stump used as the recipient

 ISSN 0284–4311

Table I. *Clinical details*

Data are expressed as number of patients unless otherwise stated

Variables	1A (healthy nerve) ($n = 17$)	1B (incompletely affected nerve) ($n = 22$)
Mean age (years)	27	32
Range	6–71	13–60
Cause of paralysis:		
Congenital	0	8
Bell's palsy	0	5
Resection of:		
Parotid tumour	6	1
Lymphangioma	4	0
Haemangioma	3	0
Maxillary tumour	2	0
Neurofibroma	1	0
Brain tumour	0	1
Infection	0	4
Trauma	1	2
Unknown	0	1
Muscle transferred:		
Gracilis	7	21
Latissimus dorsi	9	0
Extensor digitor brevis	1	1
Mean duration of follow up (years)	3	2.5
Range	1–7	<1–7

motor source: method 1A requires a facial nerve stump as healthy and fresh as possible, while method 1B uses a partly affected or impaired facial nerve stump. Availability of the ipsilateral facial nerve as recipient motor source for the muscle graft is verified preoperatively by electromyography (EMG) and intraoperatively by modified Karnovsky staining of a biopsy specimen (28). Patients with a discrete EMG interference pattern, which indicates severe neurogenic change, will not respond to this procedure.

Method 1A is chiefly indicated for patients who have a facial nerve deficit as a result of resection of a parotid tumour, haemangioma, or lymphangioma, or extratemporal facial trauma. A relatively healthy or fresh facial nerve stump is available in the ipsilateral cheek. In some cases an intratemporal facial nerve exposed in the facial nerve canal can be used. Method 1B is indicated for patients with incomplete paralysis caused by unresolved Bell's or Hunt's palsy, congenital incomplete paralysis, or infectious diseases.

PATIENTS

Thirty-nine muscles (28 gracilis, nine latissimus dorsi, and two extensor digitor brevis) were grafted between 1973 and 1994. Seventeen patients (nine female and eight male) were treated by method 1A. At the time of operation, their ages ranged from 6 to 71 (mean 27). In almost all cases, paralysis had occurred postoperatively after resection of parotid tumour, haemangioma, or lymphangioma (Table I). The facial muscles were excised or severely injuried accompanying the resection of the tumours. The muscle grafts were done for the treatment of secondary paralysis for all but six patients who had undergone a simultaneous muscle graft after resection of a tumour including the facial nerve. The latissimus dorsi muscle was used for nine of the patients, the gracilis muscle for seven, and the extensor digitor brevis muscle for one. Seven of the latissimus dorsi muscles were used as musculocutaneous flaps for simultaneous coverage of skin defects. The mean follow-up period was three years and two months.

There were 22 (11 male and 11 female) patients treated by method 1B, ranging in age from 13 to 60 (mean 32) at the time of operation. The aetiology of the paralysis was congenital ($n = 8$), incompletely unresolved Bell's palsy ($n = 5$), infective ($n = 4$), postoperative ($n = 2$), and post-traumatic ($n = 2$). The duration of the paralysis was too long to perform a primary ipsilateral nerve repair. The gracilis muscle

was used in all cases but one in which the extensor digitor brevis muscle was used. The mean follow-up period was two years and five months.

Evaluation

Evaluation was based on clinical observations of a patient's acquired facial expressions and EMG findings. Muscle function was classified in the following five categories:

Grade 5 = symmetrical balance and good facial tone at rest, sufficient muscle power upon voluntary contraction, and EMG with a full interference pattern in which no individual action potentials could be identified.

Grade 4 = symmetrical balance and good facial tone at rest, active contraction of muscle acquired but not sufficiently synchronous, and EMG with a reduced interference pattern in which some individual potentials could be identified but not others because of overlapping.

Grade 3 = symmetrical balance and good facial tone at rest, insufficient contraction of the muscle, and EMG with a discrete interference pattern in which each motor potential could be identified.

Grade 2 = reduced symmetrical balance and facial tone at rest, almost no contraction of the muscle, and EMG with no interference pattern.

Grade 1 = no improvement and an electrically silent EMG.

Grade 0 = no follow-up.

RESULTS

Initial contraction of the grafted muscle was observed within six months postoperatively in seven patients treated by method 1A in which the nerve suture was in the extratemporal region. The results were evaluated as grade 5 in five patients and as grade 4 in nine. In one (case 15, Table II) of three other patients treated by method 1A in which the nerve suture was made in the intratemporal facial nerve canal, initial contraction occurred later than in the extracranial group but the final obtained result was designated grade 5. All the patients were classified as grade 4 or better. In no patient was there the uncontrolled spastic movement of the grafted muscle reported by Chuang et al. (2) (Table II).

Initial contraction of the grafted muscle in patients treated by method 1B, on the other hand, was seen at a mean seven postoperative months, or slightly later than after method 1A. Of these patients, six were evaluated as grade 5, a rate slightly inferior to that of method 1A, and eight

Table II. *Results (method 1A)*

Case No.	Age (years)	Sex	Paralysis		Original disease	Muscle	Initial movement (postoperative months)	Grade
			Duration (years)	Type				
1	12	M	10	Incomplete	Lymphangioma	Gracilis	5	5
2	15	M	Not known	Complete	Parotid tumour	Latissimus dorsi	4	5
3	24	F	0.8	Complete	Lymphangioma	Gracilis	4	5
4	39	M	Not known	Complete	Parotid tumour	Latissimus dorsi	Not known	5
5	46	F	5	Complete	Maxillary tumour	Latissimus dorsi	6	5
6	6	F	3	Complete	Lymphangioma	Gracilis	5	4
7	8	M	7	Complete	Haemangioma	Gracilis	Not known	4
8	15	M	Not known	Complete	Neurofibromatosis	Gracilis	Not known	4
9	17	F	17	Incomplete	Lymphangioma	Gracilis	6	4
10	17	F	2	Complete	Haemangioma	Latissimus dorsi	Not known	4
11	23	F	7	Incomplete	Trauma	Extensor digitor brevis	Not known	4
12	35	F	Not known	Complete	Maxillary tumour	Latissimus dorsi	Not known	4
13	37	M	10	Complete	Haemangioma	Latissimus dorsi	5	4
14	71	F	Not known	Complete	Parotid tumour	Latissimus dorsi	Not known	4
15	25	F *	0.75	Complete	Parotid tumour	Latissimus dorsi	9	5
16	50	M*	Not known	Complete	Parotid tumour	Latissimus dorsi	6	5
17	22	M*	3	Complete	Parotid tumour	Gracilis	6	4

* The nerve was sutured at the mastoid portion in the facial nerve canal.

Table III. *Results (method 1B)*

Case No.	Age (years)	Sex	Paralysis		Muscle	Initial movement (postoperative months)	Grade
			Cause	Duration (years)			
1	18	F	Congenital	18	Gracilis	5	5
2	18	F	Trauma	17	Gracilis	(<9)	5
3	22	F	Parotid tumour	17	Gracilis	8	5
4	29	F	Congenital	29	Gracilis	8	5
5	36	M	Otitis media	8	Gracilis	9	5
6	57	F	Otitis media	30	Gracilis	6	5
7	7	M	Bell's palsy	13	Gracilis	5	4
8	15	F	Trauma	8	Gracilis	5	4
9	21	M	Congenital (microtia)	21	Gracilis	9	4
10	27	F	Congenital (microtia)	27	Gracilis	4	4
11	28	M	Bell's palsy	26	Gracilis	9	4
12	36	M	Unknown	33	Gracilis	6	4
13	42	F	Otitis media	20	Gracilis	Not known	4
14	52	F	Bell's palsy	50	Gracilis	6	4
15	25	F	Bell's palsy	6	Gracilis	6	3
16	27	M	Polioencephalitis	26	Gracilis	15	3
17	36	M	Congenital	36	Gracilis	Not known	3
18	60	M	Bell's palsy	10	Gracilis	Not known	3
19	27	M	Congenital	27	Extensor digitor brevis	Not known	1
20	24	M	Brain tumour	7	Gracilis	Not known	0
21	53	F	Congenital	53	Gracilis	5	0
22	57	M	Congenital	57	Gracilis	Not known	0

as grade 4. There was no relation between the results and the causes of paralysis, the periods after onset of paralysis, and age at the time of operation (Table III and IV).

Representative cases are shown in Figs. 1, 2, and 3.

DISCUSSION

Use of the ipsilateral facial nerve on the affected side, if available, is of great benefit for reanimation of a paralysed face. It eliminates the need to take other cranial nerves such as the hypoglossal nerve or the contralateral facial nerve, which is used for cross-face nerve grafting. It is of the utmost importance, however, that the facial nerve on the affected side be evaluated preoperatively to see whether it has sufficient regenerative ability to generate contraction of the grafted muscle. We found that the facial

Table IV. *Relation between causes and results (method 1B)*

Cause of paralysis	Grade						Initial movement (mean (SD) postoperative months)
	0	1	2	3	4	5	
Congenital	2	1	0	1	2	2	6.2 (1.9)
Bell's palsy	0	0	0	2	3	0	6.5 (1.5)
Infection	0	0	0	1	1	2	10 (3.7)
Tumour	1	0	0	0	0	1	8
Trauma	0	0	0	0	1	1	5
Unknown	0	0	0	0	1	0	6
Total	3	1	0	4	8	6	

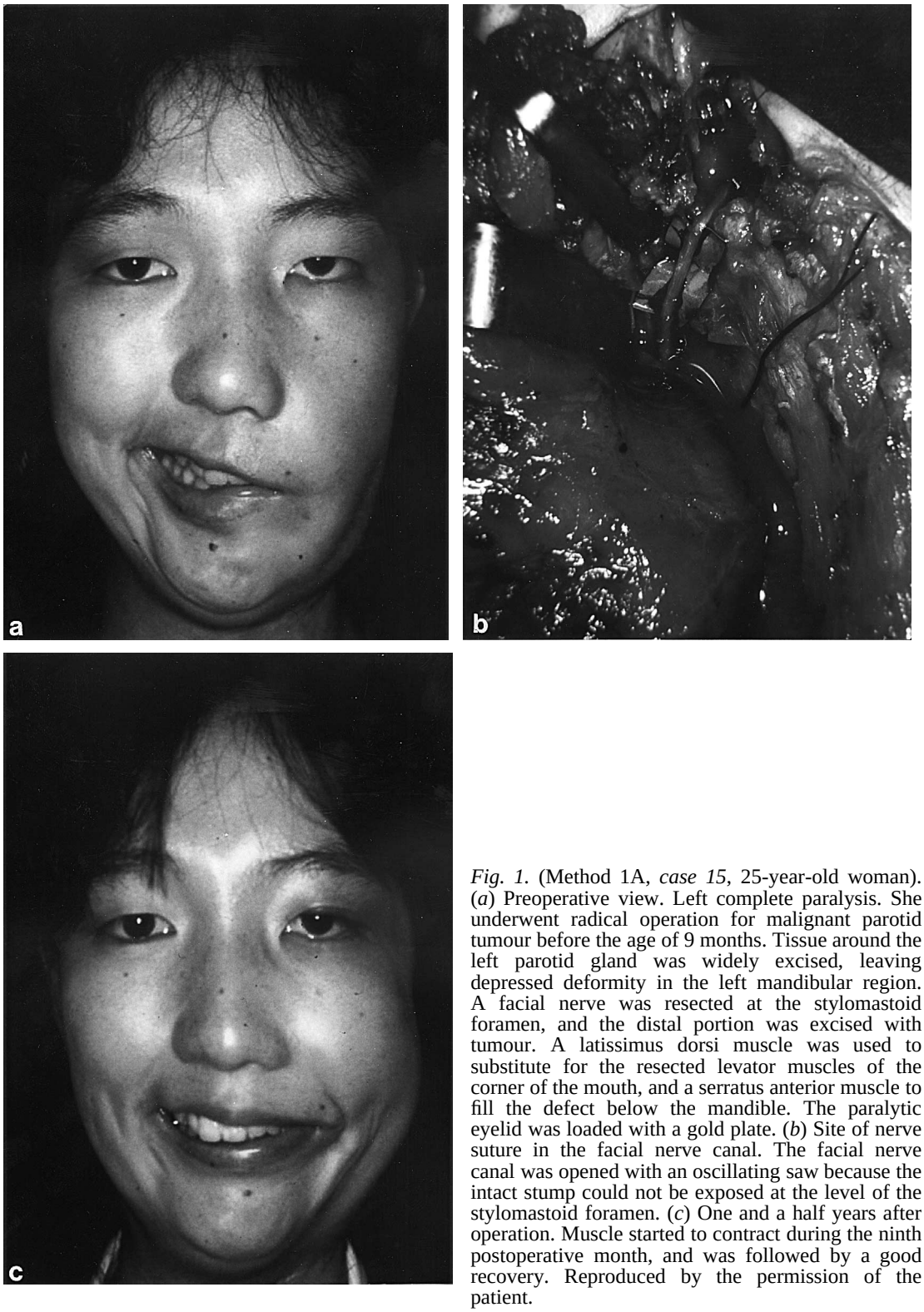

Fig. 1. (Method 1A, *case 15*, 25-year-old woman). (*a*) Preoperative view. Left complete paralysis. She underwent radical operation for malignant parotid tumour before the age of 9 months. Tissue around the left parotid gland was widely excised, leaving depressed deformity in the left mandibular region. A facial nerve was resected at the stylomastoid foramen, and the distal portion was excised with tumour. A latissimus dorsi muscle was used to substitute for the resected levator muscles of the corner of the mouth, and a serratus anterior muscle to fill the defect below the mandible. The paralytic eyelid was loaded with a gold plate. (*b*) Site of nerve suture in the facial nerve canal. The facial nerve canal was opened with an oscillating saw because the intact stump could not be exposed at the level of the stylomastoid foramen. (*c*) One and a half years after operation. Muscle started to contract during the ninth postoperative month, and was followed by a good recovery. Reproduced by the permission of the patient.

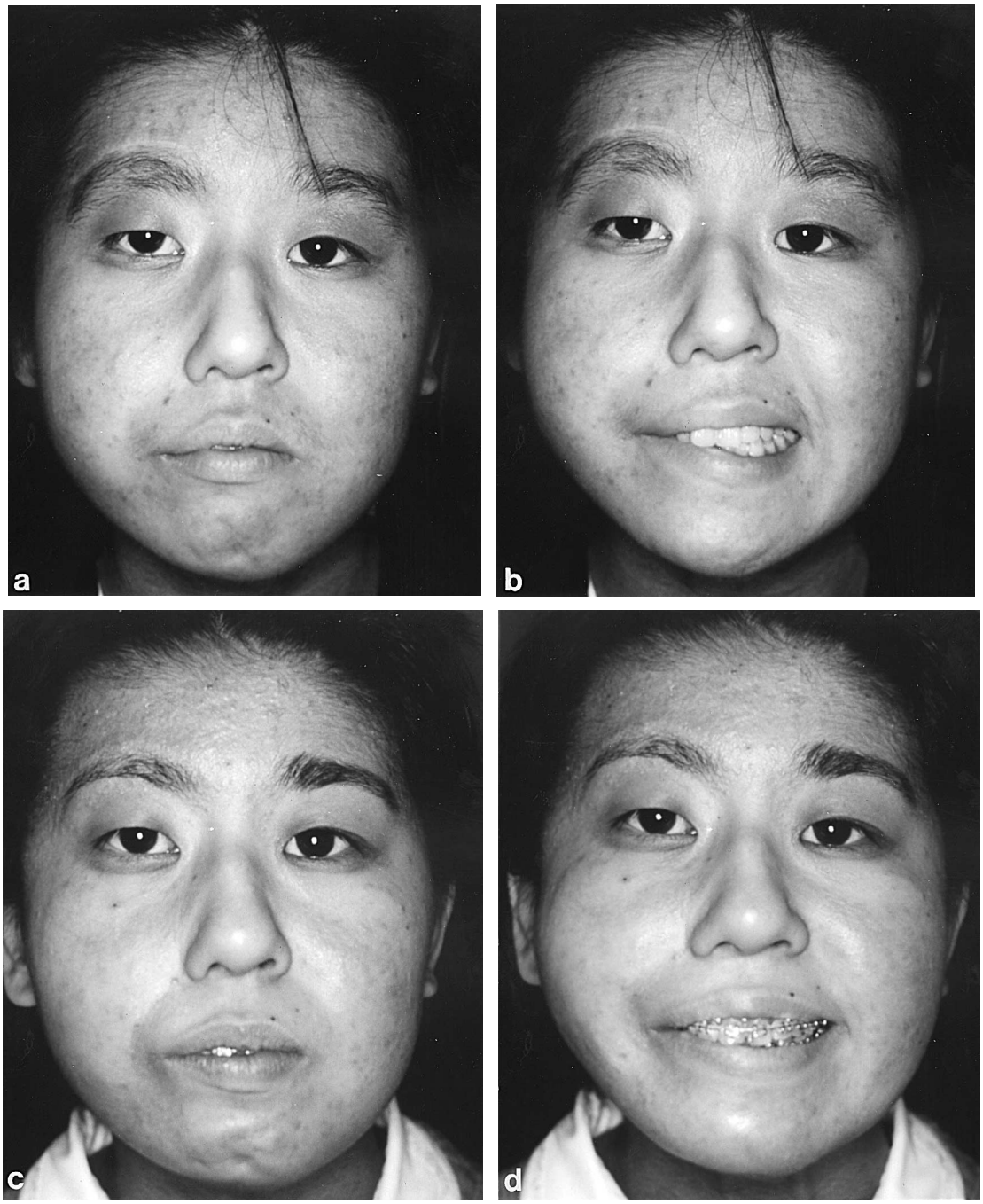

Fig. 2. (Method 1B, *case 1*, 18-year-old woman). (*a,b*) Preoperative view. Incomplete congenital facial paralysis on the right side. She has already had the eyebrow lifted by excision of the skin above the eyebrow. A gracilis muscle was used as a donor muscle. (*c,d*) Six years after operation. Muscle started to contract during the fifth postoperative month. The patient underwent debulking and shortening of the grafted muscle five years postoperatively. Reproduced by the permission of the patient.

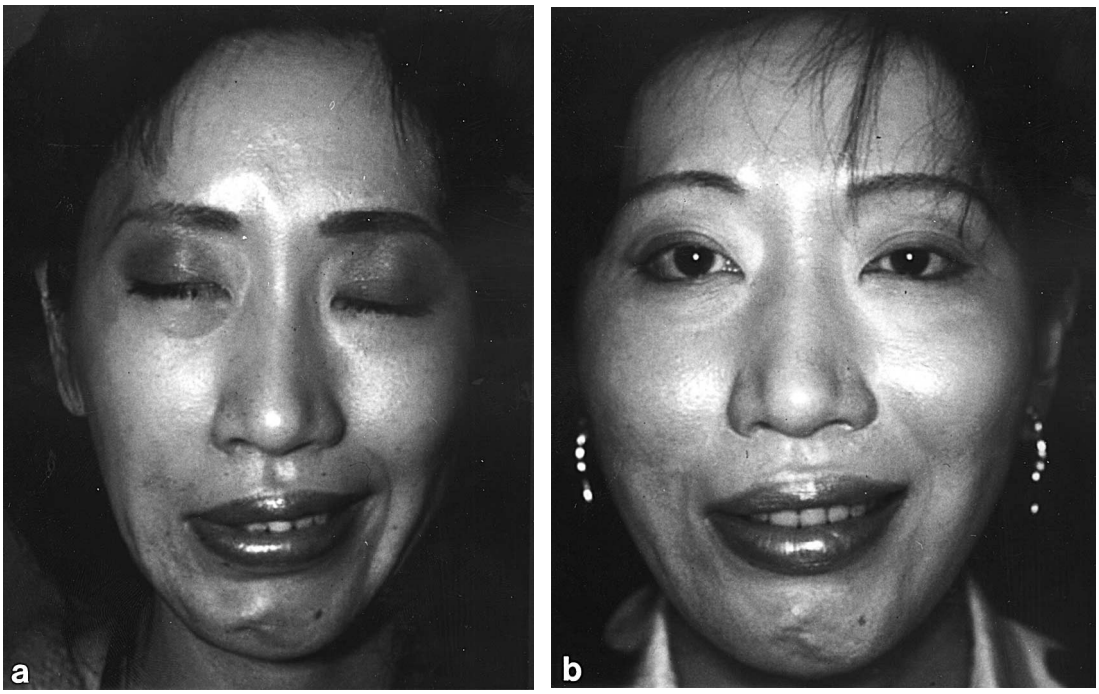

Fig. 3. (Method 1B, *case 3*, 22-year-old woman). (*a*) Preoperative view. Right incomplete facial paralysis. She underwent resection of a cervical tumour at the age 5 years (details unknown), followed by facial paralysis. A gracilis muscle was grafted to the right cheek using the buccal branch on the affected side as a motor source. (*b*) Five years after operation. The patient had her first muscle contraction during the eighth postoperative month. Reproduced by the permission of the patient.

nerve on the affected side still had rather a lot of myelinated and non-myelinated fibres on light and electron microscopic study of the specimens taken during operation (21, Fig. 4). This finding suggested to us that they could be used as the motor source for a muscle graft.

In a method 1A secondary muscle graft, duration of the period between onset of paralysis and operation may be related to the degree of nerve degeneration. For how long can the proximal stump maintain its capacity for axonal regeneration? Degeneration proximal to the resected site is often mentioned as well as the wellknown "Wallerian degeneration" on the distal side. After axonal transsection, primary degeneration starts at the proximal stump within a few days. This includes destruction of the axon and myelin sheaths (28). However, it is thought that this does not progress proximal to the nearest collateral of the stump and remains in only a short segment a few centimetres proximal to the stump (5, 22). We therefore generally use the proximal side of the transsected nerve as a motor source, surgically refreshing it during operation even though some time has elapsed since transsection. It is recommended, however, that the condition of the proximal stump to be used as a motor source be checked by some histopathological method when a long period has elapsed since facial nerve resection (Fig. 5). The reason for this is the lack of a long term follow up study of the durability of regenerative activity of the proximal stump, and also the fact that there have been some cases reported in which there was severe degeneration of the proximal portion (1, 7). Such confirmation is also necessary for primary muscle graft in method 1A when the healthy portion cannot be identified with certainty.

For most patients treated by method 1A EMG is no use for evaluation of the condition of the facial nerve because it is completely paralysed and therefore electrically silent. We therefore recommend use of modified Karnovsky staining

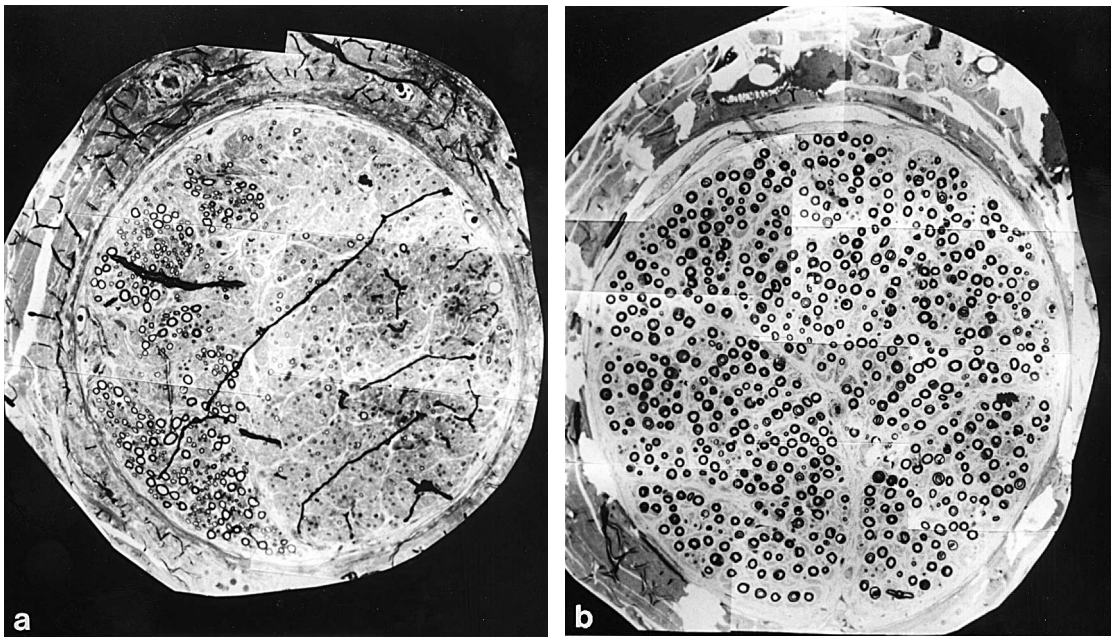

Fig. 4. Specimen of the facial nerve on the affected side (Toluidin blue stain, original magnification ×40). (*a*) A 41-year-old woman with irreversible Bell's palsy for 18 years. A number of myelinated fibres are seen in one half side though few in the other half. (*b*) A 68-year-old woman with congenital paralysis. Myelinated fibres are seen all over the area.

for acetylcholinesterase to confirm the reliability of the proximal stump as a motor source. While it has also been used to differentiate between motor and sensory nerve, it is useful for confirming the basic potential as a motor nerve (15, 26). In our method, it takes about an hour to complete the staining. This short period permits intraoperative decision as to the adequate plane of the nerve stump. During this waiting period, harvesting of the donor muscle can proceed.

We used method 1A for six patients for whom fast and excellent reinnervation of the grafted muscle was obtained even though the paralysis had been present for many years ($5 \sim 17$ years) since onset. The results prove that the proximal stump retains its capacity as a motor source for a long time, and so encourages us to carry out muscle grafts using the facial nerve on the affected side for patients with a long term history of paralysis.

In three of 17 patients in the method 1A group we could not find an intact facial nerve outside the stylomastoid foramen. In these cases the facial nerve canal was opened in the mastoid to expose a facial nerve stump adequate for nerve suture. This procedure permits certain exposure of an exact portion of the facial nerve even though it necessitates a fine elaborate technique for dissection of the nerve. A serious ploblem of this procedure, however, is difficulty in identifying the funicular topology of various branches in the canal because the nerve fibres may not be clearly divided as far as the funiculi. Uncontrolled movement of the grafted muscle could occur. We did not have such an unfavourable result in our series.

Chuang et al. warned of the risk of involuntary muscle contraction when the motor nerve is sutured to a stump of the ipsilateral facial nerve near the stylomastoid foramen (2). They described cases of involuntary contraction of the grafted muscle at rest. Muscle contractility seems to be more active and strong in method 1A compared with the two-stage muscle graft combining cross-face nerve grafting. However, we have never had experience of such unnatural

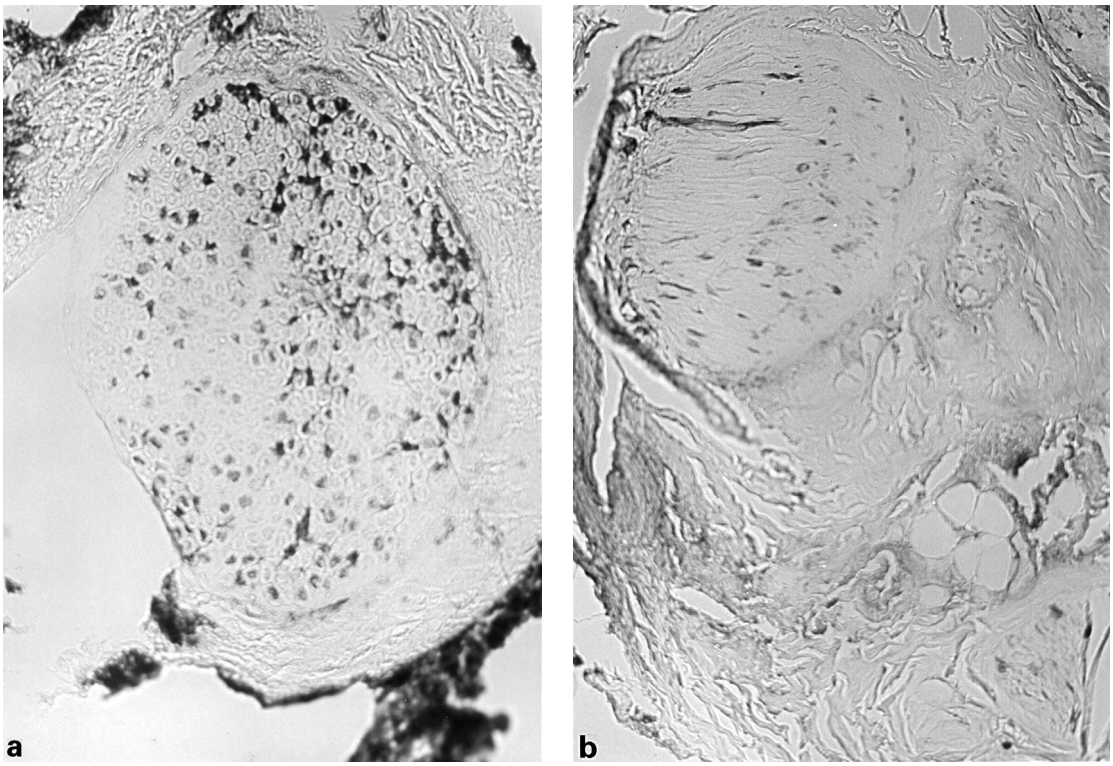

Fig. 5. Specimen of the facial nerve stained by the modified Karnovsky method. Axons of motor nerve were stained as dark spots. (*a*) Normal control, original magnification ×200. (*b*) Affected nerve, original magnification ×150. When dark spots are confirmed to the same extent as in (*b*), the nerve stump can be used as a motor source.

muscle contraction that is not amenable to treatment. Adequate tension of a grafted muscle could be most important to avoid such uncomfortable results.

Our series includes various causes of paralysis–congenital, Bell's palsy, infection, and so on–which may result in various degrees of nerve degeneration. There are two categories of congenital facial paralysis—developmental and acquired. Strict defferentiation of these is difficult when other anomalies or symptoms such as hypoplasia of the face or Moebius syndrome are not present. Falco et al. reported an incidence of 2.1 infants/1000 born with facial paralysis. Of these, 88% were considered as "acquired" cases (3). In their series, 91% of patients with acquired paralysis had undergone forceps delivery. The incidence of developmental paralysis is rather low, but severe neurogenic damage, including severe functional damage to the brain stem (6) or a facial nerve defect in the temporal bone (24), is often reported in patients with developmental paralysis such as Moebius syndrome. Method 1B may therefore be indicated for patients with developmental paralysis. Our series included two cases with definite evidence of developmental paralysis; fortunately, both patients were evaluated as grade 4 (cases 9 and 10, Table III), and so no tendency could be inferred. Bell's palsy is known to have a high recovery rate, and pathological findings consist mainly of oedematous change to the facial nerve in the facial nerve canal without inflammatory changes (8, 14, 16, 23). Most reports about facial nerve abnormalities in the early period after the onset of Bell's palsy referred only to mild nerve degeneration (4, 22). However, severe degenerative changes may develop when the pathogenesis is not elimi-

nated for a long period. Kettel described one case of Bell's palsy with interrupted continuity of the facial nerve (16), and Kumagami and Satou reported lack of the myelin sheath and the axons in three cases in which 82 days, 83 days, and 49 days, respectively had elapsed since onset (17). In short, there are varying degrees of facial nerve degeneration in Bell's palsy, and severe degeneration may be present, particularly in irreversible Bell's palsy. When the paralysis is caused by an infectious disease, inflammatory changes such as increased cellularity caused by lymphocytic infiltration are usually seen, often also accompanied by disappearance of the myelin sheath and axons (17, 22).

To sum up, degenerative changes in the nerve seem to be more or less common irrespective of the origin of the paralysis. We must therefore check the condition of the facial nerve carefully for use as a motor source, and discuss whether method 1B is indicated. Preoperative EMG and intraoperative acetylcholinesterase staining are essential for this purpose. The results of method 1B are important for selection of the treatment procedure for facial paralysis because they will tell us whether the facial nerve on the affected side can be used as a motor source for a muscle graft. What can be said from our experience is that 14 of 22 patients undergoing method 1B attained results of grade 4 or better. We think our results are sufficiently good to encourage the use of method 1B, but further experience will be necessary for generalisation of this procedure.

REFERENCES

1. Aldskogius H. Indirect Wallerian degeneration in intramedullary root fibres of the kitten hypoglossal nerve. Light and electron microscopical observations on silver impregnated sections. Neurobiology 1974; 4: 132–150.
2. Chuang D, Devaraj V, Wei F. Irreversible muscle contracture after functioning free muscle transplantation using the ipsilateral facial nerve for reinnervation. Br J Plast Surg 1995; 48: 1–7.
3. Falco N, Eriksson E. Facial nerve palsy in the newborn: incidence and outcome. Plast Reconstr Surg 1990; 85: 1–4.
4. Fowler E. The pathologic findings in a case of facial paralysis. Acta Otolaryngol 1963; 56: 113–125.
5. Fry F, Coman W. A study of retrograde cell degeneration in the lateral mamillary nucleus of the cat, with special reference to the role of axonal branching in the preservation of the cell. J Comp Neurol 1972; 144: 1–24.
6. Garcia E, Correale J, Arberas C. Familial congenital facial diplegia; electrophysiologic and genetic studies. Pediatr Neurol 1989; 5: 262–264.
7. Grant G, Westman J. Degenerative changes in dedrites central to axonal transsection. Electron microsurgical observations. Experientia 1968; 24: 169–170.
8. Hall A. Pathology of Bell's palsy. Arch Otolaryngol 1951; 54: 475–477.
9. Harii K. Refined microneurovascular free muscle transplantation for animation of paralyzed face. In: Rubin LR, ed. The paralyzed face. St. Louis: CV Mosby, 1991: 178–200.
10. Harii K. Refined microneurovascular free muscle transplantation for animation of paralyzed face. Microsurgery 1988; 9: 169–176.
11. Harii K. Treatment of long-standing facial paralysis by combining vascularized muscle transplantation with crossface nerve grafting. In: Buncke H, Furnas D, eds. Symposium of clinical frontiers in reconstructive microsurgery. St. Louis: CV Mosby, 1984: 159–171.
12. Harii K, Ohmori K, Torii S. Free gracilis muscle transplantation with microvascular anastomoses for the treatment of facial paralysis. A preliminary report. Plast Reconstr Surg 1976; 57: 133–143.
13. Harrison D. The pectoralis minor vascularized muscle graft for the treatment of unilateral facial palsy. Plast Reconstr Surg 1985; 75: 206–216.
14. Jongkees L. On the histology of Bell's palsy. Acta Otolaryngol 1954; 44: 336–343.
15. Kanaya F, Ogden L, Breidenbach W. Sensory and motor fiber differentiation with Karnovsky staining. J Hand Surg 1991; 16A: 851–858.
16. Kettel K. Pathology and surgery of Bell's palsy. Laryngoscope 1963; 73: 837–841.
17. Kumagami H, Satou I. Histopathological findings of peripheral facial paralysis. Pract Otologica Kyoto 1968; 61: 1853–1861.
18. Manktelow R. Free muscle transplantation for facial paralysis. Clin Plast Surg 1984; 11: 215–220.
19. O'Brien B, Franklin J, Morrison W. Cross-facial nerve grafts and microvascular free muscle transfer for long established facial palsy. Br J Plast Surg 1980; 33: 202–215.
20. Park S, Harii K, Ueda K. A light and electron microscopic study of affected human facial nerves. Facial Nerve Research Jpn 1988; 8: 29–32.
21. Powel T, Coman W. A note on retrograde fiber degeneration. J Anat 1964; 98: 29–32.
22. Proctor B, Corgill D. The pathology of Bell's palsy. Trans Am Acad Ophthalmol Otolaryngol 1976; 82: 70–80.
23. Sade J. Pathology of Bell's palsy. Acta Otolaryngol 1972; 95: 406–414.
24. Saitou H, Takeda T, Kishimoto S. Neonatal facial

nerve defect. Acta Otolaryngol 1994; 510 (suppl): 77–81.
25. Sanger J, Riley D, Yoshif N. Histochemical staining of nerve endings as an aid to free muscle transplantation. Microsurgery 1991; 12: 361–366.
26. Terzis J. A unique muscle for correction of facial paralysis. Plast Reconstr Surg 1991; 83: 767–776.
27. William P, Hall S. Prolonged in vivo observations of normal peripheral nerve fibres and their acute reactions to crush and deliberate trauma. J Anat 1971; 1971: 397–408.
28. Yoshimura K, Hatanaka C, Harii K. Differentiation of peripheral nerve fibers in rats and humans using a histochemical staining for cholinesterase. J Jpn PRS 1994; 14: 131–139.

Correspondence to:
Kazuki Ueda, MD
Division of Plastic Surgery
Jichi Medical School
3311-1 Yakushiji, Minamikawachi-machi,
Kawachi-gun
Tochigi, 329-04
Japan
Fax: 81-285-44-3234