Effect of Neuromuscular Electrical Stimulation in A Patient with Sjogren's Syndrome with Dysphagia: A Real Time Videofluoroscopic Swallowing Study

Shun-Man Cheung, MD; Chung-Jen Chen[1], MD; Yi-Jung Hsin, MD; Yi-Ta Tsai, MD; Chau-Peng Leong, MD

Severe dysphagia in a 54 year-old woman with Sjogren's syndrome with involvement of multiple cranial nerves significantly improved after treatment with neuromuscular electrical stimulation (NMES) in combination with a swallowing rehabilitation program. The swallowing response was assessed in real time using a videofluoroscope. Immediate improvement in the tongue retraction force, clearing of the valleculae, increase in laryngeal elevation and shortening of pharyngeal transit time were noted during stimulation. The patient returned to independent oral feeding after 46 sessions of NMES. After follow-up for 1 year, we found that the patient maintained adequate oral feeding and did not show signs of pulmonary complications. *(Chang Gung Med J 2010;33:338-45)*

Key words: neuromuscular electrical stimulation, dysphagia, Sjogren's syndrome

Sjogren's syndrome (SS) is an autoimmune disorder that primarily affects the lacrimal and salivary glands, resulting in xerophthalmia and xerostomia (the sicca complex). This syndrome occurs predominantly in women and has the highest incidence in women between 40 and 60 years old. The sicca complex may occur singularly (primary SS) or may be accompanied by other connective tissue diseases (secondary SS).[1]

SS is typically characterized by an insidious onset of the sicca complex. A decrease in the production of saliva leads to dysphagia, abnormalities in taste, and stasis of food in the buccal area. Further, a decrease in secretions in the gastrointestinal tract results in reduced esophageal motility.[2] Dysphagia with involvement of multiple cranial nerves has rarely been reported in SS patients.

The presence of dysphagia is associated with increased risks of dehydration, malnutrition, aspiration pneumonia and mortality. Appropriate swallowing rehabilitation can reduce the risk of developing complications. Traditional dysphagia treatment has varying degrees of success. In 1996, Freed et al introduced transcutaneous neuromuscular electrical stimulation (NMES), which provides controlled stimulation to strengthen the swallowing muscles.[3] Electric stimulation has been widely used in muscular rehabilitation by medical practitioners for a long time. NMES is used to strengthen the muscles through stimulation of peripheral motor nerves, causing a transfer of neurotransmitters which trigger contraction of the muscle. Freed et al theorized that NMES activated type II muscle fibers. These fibers are predominately in several swallowing muscles

From the Department of Physical Medicine and Rehabilitation; [1]Division of Rheumatology, Allergy and Immunology, Department of Internal Medicine, Chang Gung Memorial Hospital-Kaohsiung Medical Center, Chang Gung University College of Medicine, Kaohsiung, Taiwan.
Received: Jun. 4, 2008; Accepted: Dec. 8, 2008
Correspondence to: Dr. Chau-Peng Leong, Department of Physical Medicine and Rehabilitation, Chang Gung Memorial Hospital-Kaohsiung Medical Center. 123, Dapi Rd., Niaosong Township, Kaohsiung County 833, Taiwan (R.O.C.)
Tel.: 886-7-7317123 ext. 8841; Fax: 886-7-7996988; E-mail: cpleong@adm.cgmh.org.tw

which participate in high-speed, forceful contraction during swallowing. These muscle fibers generally can not be recruited by traditional rehabilitation exercise. They concluded that NMES resulted in significantly more dramatic improvement than tactile stimulation.[3]

NMES is now widely used in the field of dysphagia rehabilitation. Satisfaction is reported to be high among patients and professionals.[4] Stroke is the major etiology of dysphagia treated with NMES.

Leelamanit et al applied NMES to the thyrohyoid muscle during swallowing in 23 patients with moderate to severe "reduced laryngeal elevation dysphagia",[5] and evaluated the outcome by videofluoroscopic swallowing study (VFSS) and clinical swallowing assessment. Twenty of their patients showed marked improvement. The authors concluded that synchronous electrical stimulation during swallowing improved the dysphagia resulting from reduced laryngeal elevation.

Oh et al studied the response of cortical activities by measuring the corticobulbar output maps in patient with dysphagia before and after electrical stimulation (ES).[6] Swallowing function significantly improved after ES for 2 weeks which correlated with the corticobulbar output maps. This study suggested that ES improved swallowing function via cortical reorganization.

Suiter et al showed no significant gains in myoelectric activity of the submental muscle following NMES.[7] Some authors demonstrated that NMES at rest and during swallowing may reduce hyoid elevation, and suggested that NMES may increase the risk of penetration and aspiration.[8,9] It is important to identify the risk of aspiration and evaluate the treatment effect before NMES.

We describe a patient with primary SS showing involvement of multiple cranial nerves. We present the findings of VFSS and the outcome of NMES treatment.

CASE REPORT

A 54-year-old woman was admitted to our emergency room because of an acute attack of vomiting, swallowing disturbance, choking, and vocal hoarseness.

The patient's medical history showed hypertension under control with regular medication, lumbar spondylosis with multiple radiculopathy, and bilateral osteoarthritis of the knee joints.

The patient had a full physical and neurological examination at the time of admission. She was alert, there were no skin lesions, the conjunctiva showed no anemia and the sclera were not icteric. There was no lymph node enlargement. The lungs were clear, the cardiovascular examination showed no abnormalities,and there was no hepatosplenomegaly.

The results of a neurological examination revealed dysarthria, dysphonia, and dysphagia. On cranial nerve testing, the patient exhibited bilateral impairment in the gag reflex and a slight symmetrical decrease in the tongue protruding ability. There was no facial sensory impairment or asymmetry. No smell, visual or hearing impairment was found. The muscle strength was preserved and the muscle tone was unaltered. High cortical function including language, memory and gait pattern were normal. Because of positive signs and symptoms of multiple cranial nerve involvement, including the bilateral glossopharyngeal nerve, vagus nerve and hypoglossal nerve, we performed brain computed tomography, cerebral magnetic resonance imaging, and cerebrospinal fluid analysis; the results revealed no active vasculopathy or infection in the central nerves system (CNS). Fiberoptic endoscopy (FES) revealed palsy of the right vocal cord. Electromyography revealed a decrease in the amplitude of the left biceps (0.3-0.6 mv). A sensory nerve conduction velocity study revealed a decrease in the left median conduction velocity (40 m/sec). Further, a motor nerve conduction velocity assessment showed a mild decrease in amplitude of the left median (5.3-5.5 mv), ulnar (3.0-3.4 mv), and bilateral peroneal (1.0-1.2 mv) compound muscle action potentials; however, the H reflex and F wave response were normal (30.0-30.6 msec for the bilateral H reflex, 24.9-25.8 msec for the bilateral median nerves and 25.9-26.1 msec for the bilateral ulnar nerves). The blink-reflex revealed absence of contralateral R2 (late) potenitals bilaterally.

Laboratory examination revealed that the hemoglobin level, red and white blood cell count, platelet and differential cell count, and renal and liver function were normal. The titer of the thyroid function test was elevated (free T_4: 5.56 ng/dl). A technetium-99m (^{99m}Tc) thyroid scan revealed diffuse goiter with increased uptake of the isotope; this finding was con-

sistent with those noted in hyperthyroidism due to Graves' disease. Iodine-131 (^{131}I) therapy was initiated.

During hospitalization, the patient experienced pain on the right side of the face, oral and ocular dryness, and one episode of pneumonia. We performed diagnostic tests for autoimmune disease. Schirmer's test results were positive (right: 3 mm/5 mins, left: 5 mm/5 mins) and decrease of saliva production (2.03 g/2 mins) was noted. There were increased titers of immunoglobulin (Ig) IgG, IgA, IgM, and IgE, normal titers of anti-SSA, anti-SSB, and anti-CENP antibodies, and positive expression of anti-fodrin IgA. A whole body scan performed using gallium-67 revealed increased gallium accumulation in the lacrimal glands, parotid glands, and kidneys bilaterally.

The Table 1 shows the results of the clinical swallowing evaluation. Testing revealed a moderate risk of aspiration because of delayed initiation of swallowing, and reduced hyoid and laryngeal elevation. The patient also had dysphonia characterized by a breathy voice, hypernasality and hoarseness. Because of the positive signs and symptoms, the positive results on the blink reflex study, electrodiagnostic study, fiberoptic endoscopic study, and videofluoroscopic study, we diagnosed the patient with SS showing involvement of the bilateral glosopharyngeal nerves, right vagus nerve and hypoglossal nerve.

She was treated with methylprednisolone, oxychloroquine, and cyclophosphamide administered intravenously. The symptoms of xerophthalmia and xerostomia improved within 2 months. However, she still complained of dysphagia and dysphonia. A nasogastric feeding tube was required and oral food intake was impossible.

We could not perform VFSS to evaluate the patient's swallowing ability because she exhibited severe gastroesophageal reflux. The patient initially underwent a swallowing rehabilitation program which involved oral exercise, posture correction, dietary modification, and thermal stimulation. However, the dysphagia did not show any improvement after 10 sessions of conventional swallowing therapy. Therefore, we performed dual-channel NMES (using VitalStim™, Chattanooga Group, Hixson, TN37343, U.S.A.) for swallowing training. For this procedure, we placed 2 channels, one immediately above and the other below the thyroid notch; all 4 electrodes were vertically aligned along the midline of her neck. We used the highest stimulation intensity that she could tolerate; however, the electrical stimulation program was occasionally interrupted due to the severe gastroesophageal reflux. Dysphagia and dysphonia persisted even in the third month of hospitalization.

VFSS was performed after 10 irregular sessions

Table 1. Clinical Assessment and Videofluoroscopic Study before and after Neuromuscular Electrical Stimulation

	Before	After
Clinical assessment:		
Oral motor function:		
Tongue movement	Bilaterally reduced	Normal
Lip closure	Normal	Normal
Showing teeth	Normal	Normal
Jaw movement	Normal	Normal
Puffing	Normal	Normal
Oral pharyngeal function:		
Salivary production	Normal	Normal
Palate movement	Abnormal	Normal
Hypernasality	Moderate	None
Drooling	None	None
Hyoid elevation	Fair	Good
Swallowing reflex	Delayed (> 3 sec)	Normal (< 1sec)
Voice gargling	With hoarseness	With hoarseness
Gag reflex	Bilaterally reduced	Yes
Nasal reflux	No	No
Laryngeal elevation	Poor	Good
Choking	5 c.c.	None
Videofluoroscopic swallowing study:		
Oral stage:		
Sulcus poolings	Anterior	None
Pharyngeal stage:		
Tongue retraction	Reduced	Normal
Pooling in vallecula	Yes	No
Pooling in pyriform sinuses	Yes	No
Residue on pharyngeal wall	Yes	No
Reduce laryngeal elevation	Yes	No
Pharyngeal transit times	> 3 sec	< 1 sec
Penetration aspiration scale	Scale 5	Scale 1

of NMES. Usually, a standard VFSS is performed in the lateral view after administration of two 3-ml thin liquid barium boluses, two 3-ml thick barium boluses, 2 puree boluses, 2 semisolid boluses, and 1 solid bolus, and in the anteroposterior view, after the administration of one 3-ml thin barium bolus. The results in our patient revealed a normal oral preparatory phase, with no reduction in tongue function, but a delay in the induction of pharyngeal swallowing (> 3 s), a reduction in tongue base retraction, pooling in the vallecula, and a reduction in laryngeal elevation. Barium penetration was observed when the test was performed using the 3-ml thin barium and 3-ml thick barium boluses (Fig. 1A); therefore, we did not perform the test using the puree and solid boluses. On the basis of these results, we diagnosed the condition as a disorder in the pharyngeal phase of swallowing. We attempted to record in real time the effects of NMES on the swallowing ability by performing VFSS, with the patient's consent. The electrodes were initially positioned in the same manner as in NMES. The initial stimulation was administered at the maximum intensity that the patient could tolerate. We then requested her to swallow 3-ml of thin barium once again during NMES, and we recorded the signals on VFSS. The thin barium bolus easily passed through the pharynx, without causing stasis in the valleculae or pyriform sinuses; further, no penetration or aspiration occurred. On the other hand, during the second test using a 3-ml thick barium bolus, some barium residue remained in the valleculae and slight penetration was noted. VFSS revealed improved tongue base retraction ability and laryngeal elevation (Fig. 1B). Next, the position of the electrodes was changed (channel 1 was placed horizontally above the thyroid notch and channel 2, parallel to channel 1); however, no difference was noted in the patient's ability to swallow the 3-ml thick barium bolus.

After this examination, we continued to administer NMES for 1 h, 3 times a week, in combination with a swallowing rehabilitation program that involved postural education, diet modification, oral exercise, and swallowing practice. She was able to maintain regular oral intake of food with different consistencies and successfully passed the "3-oz water swallow test". After a total of 46 NMES sessions, the nasogastric tube was removed. The final VFSS follow-up test, wherein the patient swallowed

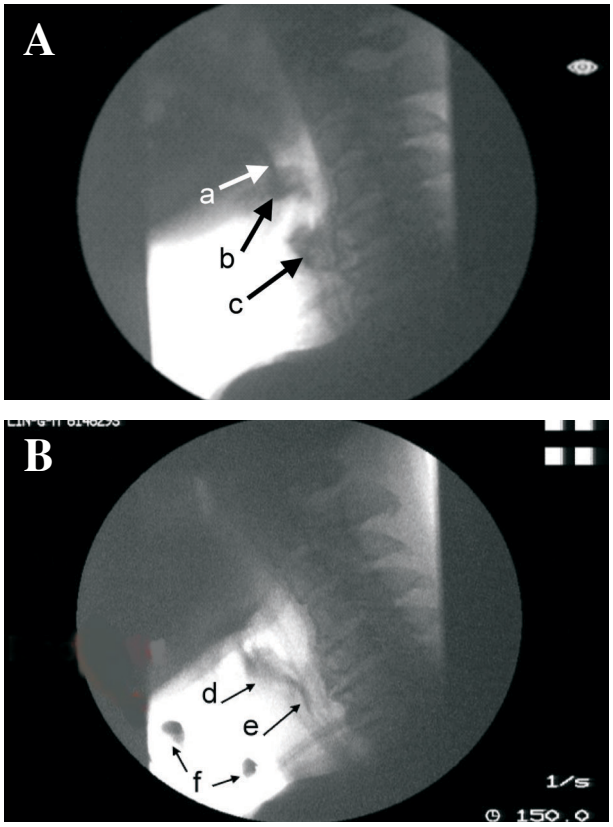

Fig. 1 Lateral view in the real time videofluoroscopic swallowing study (VFSS) with a 3 mL thin barium blous. The swallowing response (A) before electrical stimulation reveals (a) reduction in tongue base retraction, barium paste on the tongue base, (b) barium pooling in the vallecula, (c) reduction in laryngeal elevation, and barium stasis in the pyriform sinuses. (B) During electrical stimulation, (d) there is an increase in laryngeal elevation, (e) and barium passes the upper esophageal sphincter. The electrodes (f) are aligned vertically along the midline of the neck.

5 types of food of different consistencies, revealed that the oral and pharyngeal phases of swallowing were normal (Fig. 2). After 12-month of follow-up, the patient was able to maintain adequate oral food intake and her body weight increased.

DISCUSSION

A diagnosis of primary SS (PSS) requires at least 4 of the following 6 diagnostic criteria: (1) ocular dryness; (2) oral dryness or salivary gland swelling; (3) ocular signs, including positive results

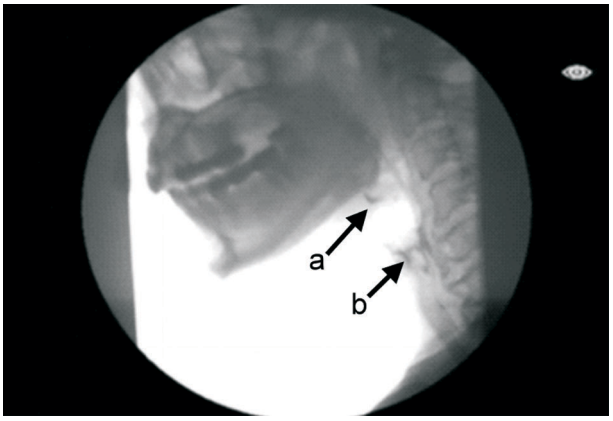

Fig. 2 Final videofluorosopic swallowing study reveals normal barium passage with no pooling in (a) the valecullar and (b) pyriform sinuses.

on a Schirmer's test or rose bengal dye test for the detection of keratoconjunctivitis; (4) specific histopathological features in the salivary gland; (5) evidence of salivary gland dysfunction, including abnormal flow of saliva and abnormal findings on scintigraphy or sialography; and (6) the presence of anti-Ro/SSA or anti-La/SSB antibodies. Further, the patient should either test positive for autoantibodies or yield positive biopsy results.[10] In this case, the diagnosis was confirmed by a Schirmer's test and saliva scan. The patient presented with many clinical manifestations consistent with those of PSS but tested negative for anti-Ro/SSA and anti-La/SSB antibodies. The patient did not consent to a biopsy of the salivary gland, so we assessed the serum levels of anti-alpha-fodrin antibodies, which were positive. A postitive anti-alpha-fodrin antibody test is an important marker for the diagnosis of SS in patients who test negative for anti-Ro/SSA and anti-La/SSB antibodies.[11] Our patient fulfilled the criteria required for the diagnosis of PSS.

Neurological involvement is noted in approximately 20% patients with PSS; this is because these manifestations often precede the diagnosis (81–93%), which makes the diagnosis of SS with neurological involvement more difficult. In PSS patients, peripheral nervous system (PNS) involvement occurs more frequently than that of the CNS.[12] PNS involvement is dominated by sensorimotor neuropathy, involvement of the cranial nerves such as the optic, trigeminal, facial, or cochlear nerve, multiple mononeuropathy, and polyradiculopathy.[12,13] CNS involvement in the disease may be focal or multifocal, manifesting as spinal cord involvement, chronic myelopathy, and motor neuron disease.[12] The impairments noted in our patient mainly involved the pharyngeal and laryngeal regions and were accompanied by an abnormal gag reflex, decrease in tongue protrusion ability, and facial pain. Dysphagia and dysphonia were the earliest symptoms in our patient. The differential diagnosis should consider other neurological diseases such as stroke, Parkinson's disease, brain tumor, and cranial neuropathy, peripheral lesions of the upper digestive tract, and connective tissue diseases. Electrophysiological analysis revealed an abnormal blink reflex in our patient, but the masseter reflex was normal. These findings are consistent with those reported previously for PSS patients with trigeminal neuropathy.[14] An FES examination revealed right vocal cord palsy, suggestive of right recurrent laryngeal nerve palsy.

Typically, VFSS reveals that the pharyngeal transit time is significantly prolonged in patients with salivary gland dysfunction.[2] The first VFSS in our patient revealed that the oral preparatory phase was normal but the pharyngeal transit time was delayed, with bolus pooling in the valleculae, penetration, and absence of laryngeal elevation. A diagnosis of neuromuscular dysfunction affecting the pharyngeal phase of swallowing was preferred, rather than pure salivary gland dysfunction. Esophageal manometric assessment reveals abnormalities in esophageal motility, and gastroesophageal reflux is often noted in Sjogren syndrome.[15,16] In our patient the esophageal motility was normal on VFSS, which is not consistent with previous study.

The conventional swallowing rehabilitation strategy adopted for neurological dysphagia includes speech therapy, patient education, practice of swallowing maneuvers, diet modification, and postural modification. We attempted to treat our patient using conventional methods, without obvious improvement. Therefore, we subsequently performed NMES for the treatment of dysphagia.

ES for muscle training exerts positive effects on muscle strength, endurance, and coordination. The effects of ES also include increases in the content of muscle contractile proteins, enzyme consumption in aerobic pathways, and the size and number of mito-

chondria.[17,18] NMES is a novel strategy for swallowing training, and it has been reported to be effective in the treatment of oropharyngeal dysphagia.[3,5,19] However, Shaw et al retrospectively evaluated 18 patients with dysphagia, according to their pretherapy overall dysphagia score (0-functional; 1-mild dysphagia, difficulty noted with at least one food consistency; 2-moderate dysphagia, swallowing is relatively safe but food consistency modification is required; 3-significant dysphagia, predominantly enteral tube feeding but still able to swallow small amounts of certain consistencies; 4-severe dysphagia, enteral tube feeding). They concluded that NMES (VitalStim therapy) seems to help those with mild to moderate, but not severe, dysphagia.[20]

According to the clinical swallowing assessment and VFSS, our patient had severe dyshagia. She was successfully treated with NMES but needed many treatment sessions.

Humbert et al pointed out in their study that surface electrical stimulation to the laryngeal regions causes significant hyoid and laryngeal descent at rest, and also reduces hyoid and laryngeal elevation during swallowing in healthy adults.[8] Ludlow et al demonstrated the effects of ES in chronic pharyngeal dysphagia, and showed significant hyoid depression occurred during stimulation at rest.[9] Aspiration and pooling were significantly reduced with a low sensory threshold level of stimulation, but not at the maximum level in that study. The authors suggested that carefully screening to determine safety is needed before treatment.

We were able to evaluate the therapeutic effects of NMES with real-time VFSS in our patient. The electrodes were placed vertically in the first test and horizonatally in the second test along the midline of the neck, above and below the thyroid cartilage. We recorded the patient's responses prior to and during stimulation. Stimulation was noted to cause immediate improvement in the tongue retraction force, clearing of the valleculae, an increase in laryngeal elevation, and a decrease in the pharyngeal transit time. These results showed that NMES may trigger the swallowing reflex and increase the contractility of the muscles involved in swallowing, thus shortening the pharyngeal transit time.

Our patient tolerated NMES well, so the nasogastric tube could be removed and the patient could tolerate oral feeding with multiple consistencies. The clinical assessment and the last VFSS follow up after NMES are shown in the Table. Our patient was able to swallow well even after the NMES treatment program and rehabilitation program were discontinued, and she did not show any clinical signs of dysphagia or pulmonary complications. We believe that the favorable outcome observed in our patient with regard to dysphagia is the result of the combined use of NMES and rehabilitation training.

REFERENCES

1. Mahoney EJ, Spiegel JH. Sjogren's disease. Otolaryngol Clin North Am 2003;36:733-45.
2. Rhodus NL, Colby S, Moller K, Bereuter J. Quantitative assessment of dysphagia in patients with primary and secondary Sjogren's syndrome. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995;79:305-10.
3. Freed ML, Freed L, Chatburn RL, Christian M. Electrical stimulation for swallowing disorders caused by stroke. Respir Care 2001;46:466-74.
4. Crary MA, Carnaby-Mann GD, Faunce A. Electrical stimulation therapy for dysphagia: descriptive results of two surveys. Dysphagia 2007;22:165-73.
5. Leelamanit V, Limsakul C, Geater A. Synchronized electrical stimulation in treating pharyngeal dysphagia. Laryngoscope 2002;112:2204-10.
6. Oh BM, Kim DY, Paik NJ. Recovery of swallowing function is accompanied by the expansion of the cortical map. Int J Neurosci 2007;117:1215-27.
7. Suiter DM, Leder SB, Ruark JL. Effects of neuromuscular electrical stimulation on submental muscle activity. Dysphagia 2006;21:56-60.
8. Humbert IA, Poletto CJ, Saxon KG, Kearney PR, Crujido L, Wright-Harp W, Payne J, Jeffries N, Sonies BC, Ludlow CL. The effect of surface electrical stimulation on hyolaryngeal movement in normal individuals at rest and during swallowing. J Appl Physiol 2006;101:1657-63.
9. Ludlow CL, Humbert I, Saxon K, Poletto C, Sonies B, Crujido L. Effects of surface electrical stimulation both at rest and during swallowing in chronic pharyngeal dysphagia. Dysphagia 2007;22:1-10.
10. Vitali C, Bombardieri S, Moutsopoulos HM, Coll J, Gerli R, Hatron PY, Kater L, Konttinen YT, Manthorpe R, Meyer O, Mosca M, Ostuni P, Pellerito RA, Pennec Y, Porter SR, Richards A, Sauvezie B, Schiodt M, Sciuto M, Shoenfeld Y, Skopouli FN, Smolen JS, Soromenho F, Tishler M, Wattiaux MJ, Tomsic M, Van de Merwe JP, Yeoman CM. Assessment of the European classification criteria for Sjogren's syndrome in a series of clinically defined cases: results of a prospective multicentre study. The European Study Group on Diagnostic Criteria for Sjogren's Syndrome. Ann Rheum Dis 1996;55:116-21.

11. Witte T. Antifodrin antibodies in Sjogren's syndrome: a review. Ann N Y Acad Sci 2005;1051:235-9.
12. Delalande S, de Seze J, Fauchais AL, Hachulla E, Stojkovic T, Ferriby D, Dubucquoi S, Pruvo JP, Vermersch P, Hatron PY. Neurologic manifestations in primary Sjogren syndrome: a study of 82 patients. Medicine 2004;83:280-91.
13. Gemignani F, Marbini A, Pavesi G, Di Vittorio S, Manganelli P, Cenacchi G, Mancia D. Peripheral neuropathy associated with primary Sjogren's syndrome. J Neurol Neurosurg Psychiatry 1994;57:983-6.
14. Valls-Sole J, Graus F, Font J, Pou A, Tolosa ES. Normal proprioceptive trigeminal afferents in patients with Sjogren's syndrome and sensory neuronopathy. Ann Neurol 1990;28:786-90.
15. Anselmino M, Zaninotto G, Costantini M, Ostuni P, Ianniello A, Boccu C, Doria A, Todesco S, Ancona E. Esophageal motor function in primary Sjogren's syndrome: correlation with dysphagia and xerostomia. Dig Dis Sci 1997;42:113-8.
16. Rosztoczy A, Kovacs L, Wittmann T, Lonovics J, Pokorny G. Manometric assessment of impaired esophageal motor function in primary Sjogren's syndrome. Clin Exp Rheumatol 2001;19:147-52.
17. Munsat TL, McNeal D, Waters R. Effects of nerve stimulation on human muscle. Arch Neurol 1976;33:608-17.
18. Nash MS, Bilsker S, Marcillo AE, Isaac SM, Botelho LA, Klose KJ, Green BA, Rountree MT, Shea JD. Reversal of adaptive left ventricular atrophy following electrically-stimulated exercise training in human tetraplegics. Paraplegia 1991;29:590-9.
19. Burnett TA, Mann EA, Cornell SA, Ludlow CL. Laryngeal elevation achieved by neuromuscular stimulation at rest. J Appl Physiol 2003;94:128-34.
20. Shaw GY, Sechtem PR, Searl J, Keller K, Rawi TA, Dowdy E. Transcutaneous neuromuscular electrical stimulation (VitalStim) curative therapy for severe dysphagia: myth or reality? Ann Otol Rhinol Laryngol 2007;116:36-44.

神經肌肉電刺激對乾燥症合併吞嚥困難病例之治療效果：同步吞嚥攝影檢查之研究

張舜雯 陳忠仁[1] 辛宜蓉 蔡易達 梁秋萍

一位 46 歲乾燥症合併多發性顱神經侵犯的患者，出現嚴重的吞嚥障礙。使用吞嚥肌之神經肌肉電刺激治療，配合吞嚥復健，獲得明顯改善。以同步電視螢光幕吞嚥攝影研究其吞嚥反應，發現在電刺激時，能立即改善舌根後縮的力量，食團滯留在會厭谿變少，喉上提的力量增加，並縮短了食團通過咽部的時間。患者經此電刺激治療 46 次後成功拔除鼻胃管，重獲經口進食的功能。停止治療後追蹤一年，患者仍可維持正常吞嚥功能，沒有發生肺部合併症。(長庚醫誌 2010;33:338-45)

關鍵詞：神經肌肉電刺激，吞嚥困難，乾燥症

長庚醫療財團法人高雄長庚紀念醫院 復健科，[1]風濕免疫科；長庚大學 醫學院
受文日期：民國97年6月4日；接受刊載：民國97年12月8日
通訊作者：梁秋萍醫師，長庚醫療財團法人高雄長庚紀念醫院 復健科。高雄縣833鳥松鄉大埤路123號。
Tel.: (07)7317123轉8841; Fax: (07)7996988; E-mail: cpleong@adm.cgmh.org.tw