A REVIEW OF ELECTRICAL STIMULATION AND ITS EFFECT ON LINGUAL, LABIAL AND BUCCAL MUSCLE STRENGTH

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ABSTRACT

Background. Lingual, labial and buccal weakness (LLBW) is a widespread consequence of several neurological insults. LLBW impact on oral motor functions such as speech production and swallowing is well documented in the literature. Therefore, it is important for the speech-language pathologists to have access to evidence-based approaches for treatment. Thus, it is imperative that the speech-language pathology field search for effective treatment approaches and explore new treatment modalities that can improve therapy outcomes. One relatively new modality in this field is neuromuscular electrical stimulation (NMES). **Aims.** The purpose of this paper is fivefold: (a) to provide an overview of the general effects of NMES on skeletal muscles; (b) to review the effect of NMES on orofacial musculature evaluating the potential appropriateness of NMES for use in strengthening lingual, labial and buccal muscles; (c) to identify future directions for research with consideration of its potential role in improving speech intelligibility and the oral preparatory phase of swallowing in patients with oral motor weakness; (d) to provide a brief anatomic and physiologic bases of LLBW; (e) to provide background information for orofacial myologists who may encounter individuals with LLBW. **Main Contribution.** NMES is a modality that is commonly used in physical therapy and occupational therapy fields that assists in treating several motor and sensory muscular disorders including muscular weakness. The literature reviewed demonstrate that very limited data related to the use of NMES on orofacial muscles exist despite the fact that these muscles can be easily accessed by electrical stimulation from the surface. **Conclusions**. This review of the research using electrical stimulation of muscles highlights the need for experimental treatment studies that investigate the effect of NMES on orofacial weakness.

KEYWORDS: Neuromuscular electrical stimulation, labial weakness, lingual weakness, dysarthria

INTRODUCTION

One of the consequences following various neurological insults is paresis or paralysis to muscles of the mouth and face. Three of these muscle groups are known as lingual, labial, and buccal regions. Lingual, labial, and buccal weakness (LLBW) experienced by many individuals across the life span can lead to an increased risk of speech or swallowing impairment. Quality of life may also be affected when these individuals are unable to communicate using intelligible speech. These individuals may also, experience a life threatening disorder as a result of swallowing impairment that leads them to avoid previously enjoyed foods and beverages. Consequently, individuals with a swallowing disorder may avoid specific food consistencies that may pose a health risk and those with reduced speech intelligibility or oral phase dysphagia may avoid potentially embarrassing social situations.

The purpose of this paper is fivefold: (a) to provide an overview of the general effects of NMES on skeletal muscles; (b) to review the effect of NMES on orofacial musculature evaluating the potential appropriateness of NMES for use in strengthening lingual, labial and buccal muscles; (c) to identify future directions for research with consideration of its potential role in improving speech intelligibility and the oral preparatory phase of swallowing

in patients with oral motor weakness; (d) to provide a brief anatomic and physiologic bases of LLBW; (e) to provide background information for orofacial myologists who may encounter individuals with LLBW.

Anatomic and physiologic bases of LLB muscles

The lingual, labial, and buccal (LLB) regions consist of many muscles that are active during speech and swallowing. The following is a brief anatomic and physiologic overview of muscles that are of interest to this review.

Labial muscles are considered part of the muscles of facial expression. A number of muscles constitute the lips and control their function (Figure 1) – i.e., Orbicularis oris, Levator labii superioris, Levator anguli oris, Zygomaticus minor, Zygomaticus major, Risorius, Depressor anguli oris, Depressor labii inferioris, and Mentalis (Seikel, King & Drumright, 2009). These muscles receive motor supply via the facial nerve (Duffy, 2012)(Figure 4).

The buccal muscle is also considered part of the muscles of facial expression. The buccinator is a thin quadrilateral muscle that forms the buccal area and connects the maxilla and the mandible to form the anterior part of the cheek or the lateral wall of the oral cavity (Figure 1). This muscle is also innervated by the facial nerve (Seikel et al., 2009))(Figure 4). This muscle is active during mastication, and it assists in holding the cheek to the teeth during chewing (Duffy, 2012) to prevent food from entering the lateral sulcus of the oral cavity.

The tongue consists of eight intrinsic and extrinsic muscles (Figures 1 and 2). Four intrinsic muscles that are not attached to any bone control the shape of the tongue, and four extrinsic muscles that are attached to bones control the position of the tongue (Seikel et al., 2009). The extrinsic muscles are the genioglossus, which arises from the mandible and protrudes the tongue; hyoglossus, which arises from the hyoid bone and depresses the tongue; styloglossus, which arises from the styloid process and elevates and retracts the tongue; palatoglossus, which arises from the palatine aponeurosis, and depresses the soft palate, moves the palatoglossal fold towards the midline, and elevates the back of the tongue (Seikel et al., 2009). The intrinsic muscles are the superior longitudinal muscle that runs along the superior surface of the tongue under the mucous membrane, and elevates, assists in retraction of, or deviates the tip of the tongue; the inferior longitudinal muscle that lines the sides of the tongue and is joined to the styloglossus muscle; the verticalis muscle that is located in the middle of the tongue and joins the superior and inferior longitudinal muscles; the transversus muscle that divides the tongue at the middle and is attached to the mucous membranes that run along the sides (Seikel et al., 2009). The motor functions of all intrinsic and extrinsic muscles of the tongue are supplied by the hypoglossal nerve (Figure 5), with the exception of the palatoglossus, which is innervated by the vagus nerve (Seikel et al., 2009))(Figure 5).

Neurological bases for LLBW. LLBW can result from lesions to the muscles, the neuromuscular junction, the lower motor neurons of the cranial nerves, the upper motor neurons originating in the motor cortex or from damage to the extra pyramidal motor system (Duffy, 2012; Webster, 1999; Brookshire, 1997). A brief overview of the effect of these lesions on LLBW is presented in the following section.

A. Upper motor neuron damage. Unilateral lesions of the motor cortex or pyramidal tract cause mild to moderate contralateral LLBW or paralysis. On the other hand, bilateral upper motor neuron lesions exhibit severe LLBW that leads to severe dysarthria, dysphagia and poorly controlled laughing and crying (Duffy, 2012; Webster, 1999; Brookshire, 1997).

Upper motor neuron lesions occur in conditions affecting motor neurons in the brain or spinal cord such as stroke, multiple sclerosis, traumatic brain injury, and cerebral palsy. Symptoms include decreased control of active movement, muscular spasticity, and decreased control of active movement, particularly slowness (Duffy, 2012; Webster, 1999; Brookshire, 1997).

B. Lower motor neuron damage. Damage to cranial nerves that innervate LLB muscles or their nuclei in the pons and medulla results in LLBW (Brookshire, 1997). As the cranial nerves lie very close to one another in the brainstem, a lesion will usually damage more than one pair of nuclei. Thus, a number of

muscles may be affected bilaterally (Webb, Adler & Love, 2008). Symptoms include muscle paresis or paralysis, fibrillations, fasciculations, and hypotonia or flaccidity. Eventually these muscles become atrophied (Duffy, 2012; Webster, 1999; Brookshire, 1997). Damage affecting the hypoglossal, facial and trigeminal nerves result in weakness of lingual, labial, and buccal musculature and dysfunctions of speech and decreased eating and chewing capacity (Webb, Adler & Love, 2008).

C. Extra pyramidal motor system damage. There are two major disorder complexes associated with disease of the extra pyramidal motor system: Parkinson's disease (PD) and Huntington's disease (HD). Parkinson's disease results from the death of dopaminergic neurons in the substantia nigra pars compacta (Webb et al., 2008). It is characterized by a resting tremor, but the most debilitating symptom is severe bradykinesia or akinesia (Webb et al., 2008). Parkinsonism is characterized by varying degrees of (1) rigidity, (2) bradykinesia, (3) tremor, and (4) postural defects. Huntington's disease is also known as Huntington's Chorea because it is characterized by continuous choreiform movements of the body (especially the limbs and face) (Webb et al., 2008).

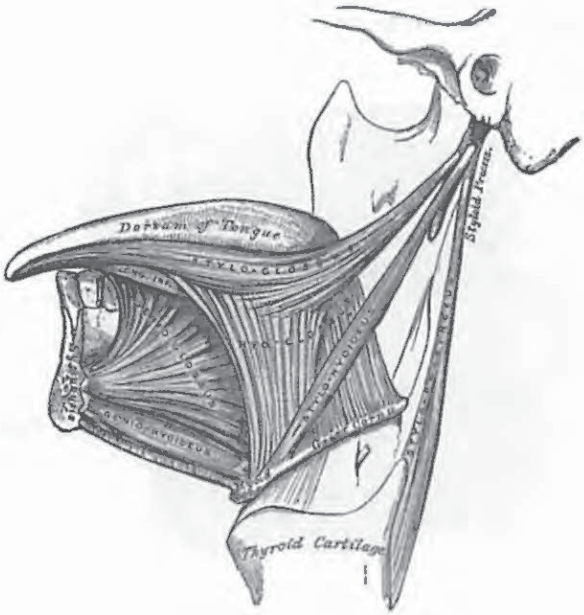
D. Neuromuscular junction diseases. In neuromuscular junction diseases, the end plate potential fails to effectively activate the muscle fiber due to an autoimmune reaction against acetylcholine receptors, resulting in muscle weakness and fatigue (Sha & Layzer, 2007). The most common example of this disorder is myasthenia gravis that is caused, commonly, by autoantibodies against the acetylcholine receptor (Sha & Layzer, 2007). Myasthenia gravis often causes a unique muscular weakness, in which the patient's muscular strength deteriorates with prolonged use, but is recovered following a break from usage (Brookshire, 1997).

Figure 1 Labial and Jaw Muscles



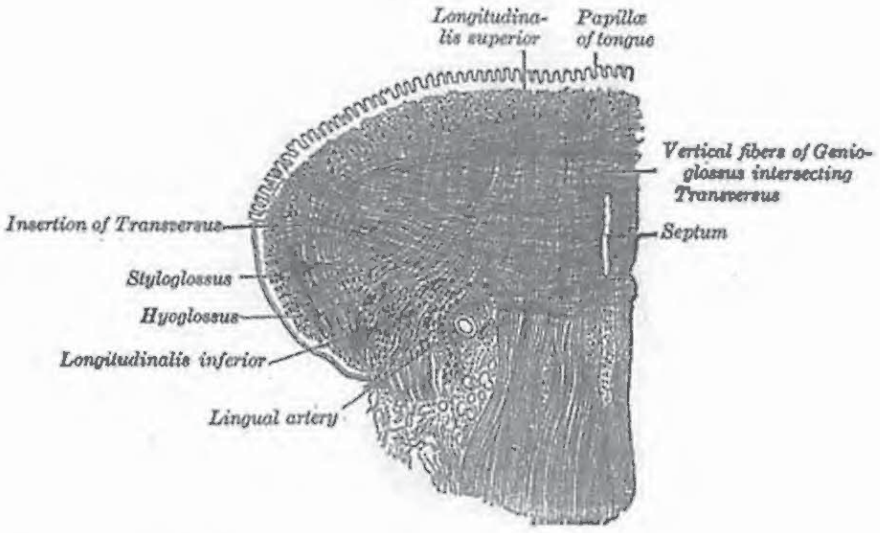
Adapted from (Gray, H., 1918) (The figure was adopted from Wikipedia Commons website were there an open permission for reuse of the material)

Figure 2 Extrinsic Lingual Muscles



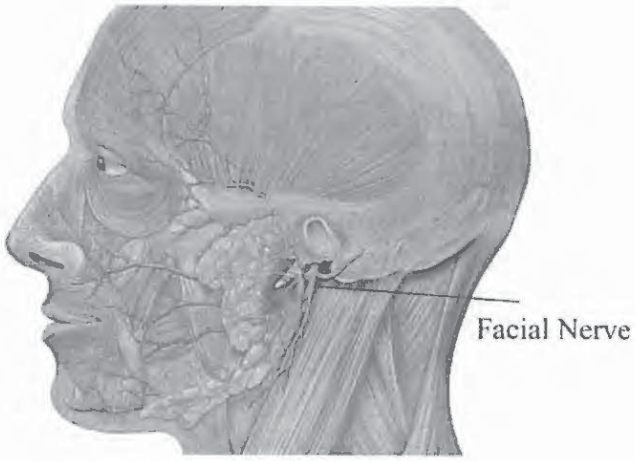
Adapted from (Gray, H., 1918) (The figure was adopted from Wikipedia Commons website were there an open permission for reuse of the material)

Figure 3 Intrinsic and Extrinsic Lingual Muscles



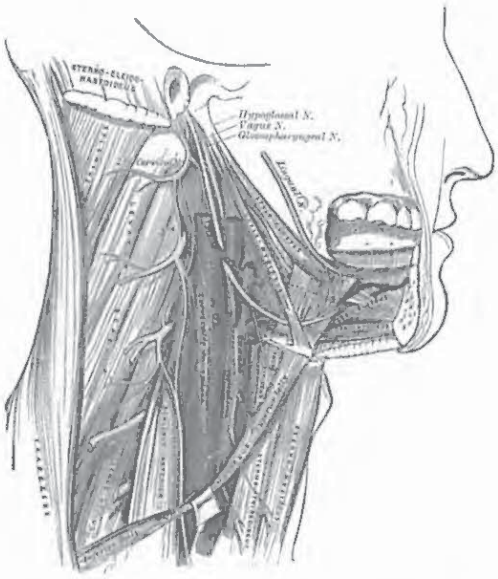
Adapted from (Gray, H., 1918) (The figure was adopted from Wikipedia Commons website were there an open permission for reuse of the material)

FIGURE 4 Neural Innervation of Facial Muscles



Adapted from (Lynch, p., 2006) (The figure was adopted from Wikipedia Commons website were there an open permission for reuse of the material)

FIGURE 5 Neural Innervation of Lingual Muscles



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Prevalence of LLBW

To date, no reliable estimates exist on the incidence and prevalence of individuals who suffer from weakness of the labial, buccal and lingual muscles. However, the presence of lingual, labial and buccal weakness in a society can be inferred from the prevalence of disorders causing these weaknesses. Since LLBW is often associated with neurological impairments (Brookshire, 1997; Duffy, 2012) including neurological degenerative conditions such as Parkinson's disease (McAuliffe, Ward, Murdoch & Farrell, 2005; Solomon, Robin & Luschei, 2000) and amyotrophic lateral sclerosis (ALS) (Dworkin, 1980; Cha & Patten, 1989; Khan & Prayson, 2003) as well as non-progressive brain damage such as traumatic brain injury (TBI) (Theodoros, 2001; Goozée, Bruce, Murdoch, Deborah, & Theodoros, 2001) and cerebrovascular accidents (CVAs) (Brookshire, 1997; Duffy, 2012), the incidence may be inferred from data on these neurological disorders.

For example, studies estimate that in the United States, 1.5 to 2 million individuals sustain a TBI each year (Langlois. Kegler & Butler, 2003; Stierwalt & Murray, 2002; Coronado, 2011). A CDC survey in 2012 (CDC, 2012) indicates that 2.7% of non-institutionalized U.S. adults (approximately 6.2 million persons) had a histories of stroke; consequently many of these individuals may present with LLBW.

There are several neurological degenerative conditions that may lead to LLBW, one of which is Parkinson's disease (PD) (Ramig, Fox, & Sapir, 2004). Samii, Nutta & Ranson (2004) estimate that 89% of individuals with PD have a speech or voice disorder that is caused by a neuromuscular impairment. Additional studies estimated that the prevalence of PD in industrialized countries is 0.3% of the general population and about 1% of the population older than 60 years (Samii, Nutt, & Ranson, 2004; Levine, Fahrbach, & Siderowf, 2003). Another common cause for LLBW is Huntington's disease. Individuals with this disorder present with an unpredictable and weak speech pattern that may severely compromise clarity of speech. Qin & Gu (2004) estimated that Huntington's disease affects 5 out of 100,000 people and symptoms usually occur in the late 40s.

Furthermore, LLBW is highly prevalent in patients with ALS (Kidney, Alexander, Corr, O'Toole, & Hardiman, 2004). Because the disease usually does not affect cognitive abilities, patients with ALS are aware of their progressive loss of functioning and may become anxious and depressed. Khan & Prayson (2003) estimated that the disease's worldwide prevalence ranges from 0.5 to 3 in 100,000, with a few areas having a higher prevalence.

Given the widespread nature of LLBW and its impact on oral motor functions such as speech production and swallowing, it is important for the speech-language pathologists to have access to evidence-based approaches for treatment. Thus, it's imperative that the speech-language pathology field search for effective treatment approaches and explore new treatment modalities that can improve therapy outcomes.

One relatively new modality in this field is neuromuscular electrical stimulation (NMES). The effect of NMES on muscular strength and movement has been investigated in several areas of the head and neck region. Some examples include (1) the back of the tongue in sleep apnea (Isono, Tanaka, Nishino, 1999; Mezzanotte, Tangel, White, 1992; Miki, Hida, Shindoh, Kikuchi, Chonan, Taguchi, Inoue, Takishima, 1989; Oliven, Schnall, Pillar, Gavriely, Odeh, 2001; Randerath, 2006); (2) regions of the face associated with facial palsy (Kavanagh, Newellm, Hennessy & Sadick, 2012) and; (3) the larynx and submental areas associated with dysphagia (Humbert, et al., 2006; Nam, Beom, Oh & Han, 2013; Carnaby-Mann & Crary, 2007; Ludlow et al., 2007). Although, the literature supports a positive correlation between the use of NMES and increased muscular strength and range of motion (Binder-Macleod, Halden & Jungles, 1995; Binder-Macleod & Lee, 1997; Doucet, Lam & Griffin 2012; Khan, 1987), limited research has investigated the effect of neuromuscular stimulation on the lingual, labial and buccal muscles in terms of speech production and intelligibility.

GENERAL ELECTRICAL STIMULATION DEFINITION AND ITS EFFECT ON THE SKELETAL MUSCLES

Definition

Neuromuscular electrical stimulation (NEMS) is a treatment that uses small electrical current to activate nerves innervating muscles effected by paralysis resulting from spinal cord injury (SCI), head injury, stroke and other neurological disorders. The application of NMES causes muscles to contract as if they were exercising. NMES is delivered to muscles as a waveform of electrical current via electrodes.

Using electrical stimulation to produce human movement is not a novel approach. Cambridge (1997) noted that in 1790, Luigi Galvani first observed motion after applying electrical wires to leg muscles severed from the body of a frog. In 1831, Michael Faraday showed that electrical currents could stimulate nerves to create active movement (Cambridge, 1997). Liberson, Holmquest, Scot & Dow (1961) used electrical stimulation for muscle function -- one of the early clinical experiments of NMES -- to stimulate the peroneal nerve in the leg in an effort to correct foot drop during ambulation in persons with stroke-related hemiplegia.

There are, broadly, two types of NMES electrodes: surface (cutaneous) and intramuscular (percutaneous). In the rehabilitation field, surface NMES electrodes are most commonly used. The electrodes are generally incorporated in pads that adhere to the skin. The electrical current (in the NMES devices) has customizable characteristics which allow for manipulation of the stimulus frequency, amplitude, and pulse width of the electric current to be delivered. The amplitude and pulse width determine the number of muscle fibers that are activated (Sheffler & Chae, 2007).

Benefits & Applications of NMES

NMES approaches are generally used in patients who have an intact peripheral and motor-neuron system but are unable to activate their musculature for volitional functions. Current therapeutic clinical application of NMES is limited to neurologic impairments that involve the upper and lower motor neuron such as spinal cord injury (SCI), stroke, brain injury, multiple sclerosis, and cerebral palsy (Sheffler & Chae, 2007) and involve muscle weakness or paralysis.

According to Randerath (2006), "Muscle training using electrical neurostimulation (ENS) has been found to effectively strengthen skeletal muscles in pathological or posttraumatic situations. In healthy muscles, neuromuscular electrical stimulation can induce the activity of motor units which are difficult to activate voluntarily" (p.161). The literature supports the therapeutic application of NMES to enhance muscle strength, retard muscle atrophy, and reduce spasticity (Binder-Macleod, Halden & Jungles, 1995; Binder-Macleod & Lee, 1997). NMES also has been found to help improve muscle strength, increase range of motion, reduce edema, decrease atrophy, heal tissue, and decrease pain (Doucet, Lam & Griffin, 2012; Khan, 1987).

Some types of electrical stimulation, such as functional electrical stimulation, provide therapeutic effects that persist when the NMES device is not in use (Daly, et al., 1996). Daly et al. (1996) reported that clinicians observed improvements in voluntary neuromuscular function and improvements in the condition of soft tissue after using electrical stimulation. Additionally, therapists observed motor recovery in people with incomplete spinal cord injury, stroke, or traumatic brain injury after the use of motor prostheses (Daly, et al., 1996).

Furthermore, Maffiuletti (2010) listed several additional applications for NMES in the rehabilitation field. These applications include (1) the preservation of muscle mass and function during prolonged periods of disuse or immobilization (Gibson et al., 1988), (2) for recovery of muscle mass and function following prolonged periods of disuse or immobilization (Snyder-Mackler et al., 1995), (3) for improvement of muscle function in different healthy populations such as elderly subjects (Caggiano et al., 1994) and adult subjects (Currier and Mann 1983) and (4) for use with recreational and competitive athletes (Babault et al., 2007; Delitto et al., 1989; Maffiuletti et al., 2002a; Pichon et al., 1995).

Thus, according to Maffiuletti (2010), (re)training programs that utilized NMES have been used in the following areas:

- Cardiovascular medicine patients with chronic or refractory heart failure (Harris et al., 2003; Quittan et al., 2001), cardiac transplant (Vaquero et al., 1998), chronic obstructive pulmonary disease (Roig and Reid 2009; Vivodtzev et al., 2008);
- Orthopedic medicine patients with anterior cruciate ligament reconstruction (Delitto et al., 1988; Eriksson and Haggmark 1979; Fitzgerald et al., 2003; Lieber et al., 1996; Snyder-Mackler et al., 1991), bone fracture (Gibson et al., 1988), knee osteoarthritis (Gibson et al., 1989; Zizic et al., 1995), rheumatoid arthritis (Piva et al., 2007), total knee arthroplasty (Petterson and Snyder-Mackler 2006; Stevens et al., 2004), total hip arthroplasty (Suetta et al., 2004), meniscectomy (Gould et al., 1983), patellofemoral pain (Callaghan et al., 2001);
- Neurological medicine patients following stroke (Glinsky et al., 2007; Newsam and Baker 2004), spinal cord injury (Belanger et al., 2000; Crameri et al., 2000; Dudley et al., 1999), cerebral palsy (Merrill 2009; Stackhouse et al., 2007);
- General medicine patients with hemophilia (Querol et al., 2006), cancer (Crevenna et al., 2006) and critically ill patients (Gerovasili et al., 2009);
- Geriatric medicine healthy (Amiridis et al., 2005; Caggiano et al., 1994) and unhealthy (Stevens et al., 2004) elderly subjects;
- Space medicine Astronauts (Convertino 1996; Mayr et al., 1999), simulated microgravity (Duvoisin et al., 1989);
- Sports medicine healthy and injured athletes of individual and team sports (Delitto et al., 1989; Maffiuletti, 2006. (224). Table 1 presents a summary of studied NMES applications.

Although electrical stimulation has the capacity to produce movement in denervated, paralyzed, or spastic muscles, there is uncertainty concerning the mechanisms by which NMES produces these changes. However, it is obvious that neuromuscular electrical stimulation elicits muscle contraction by initiating action potentials in intramuscular nerve branches that mimic the action potential coming from the central nervous system (Hultman et al., 1983). The order of motor unit activation with NMES depends on two factors: (1) the combined effects of axon diameter (Clamann et al., 1974 & Eccles et al., 1958); and (2) the distance between the axon and the active electrode (Gorman & Mortimer, 1983; Delitto & Snyder-Mackler, 1990).

Limitations of Electrical Stimulation

Doucet, Lam & Griffin (2012) noted that the most significant limitations of any non-physiologically induced muscle activation is the overall decreased efficiency of contraction and the high tendency for development of neuromuscular fatigue. However, the application of NMES can be customized to reduce fatigue and optimize force output by adjusting the associated stimulation parameters. Therefore, strategies must be designed as part of electrical stimulation regimens to offset the high degree of fatigue associated with NMES.

NMES represents a non-physiologic means of muscle contraction that bypasses the processes associated with volition. Thus, Enoka (1988) suggested three lines of evidence that question the mechanisms by which NMES might elicit increases in strength secondary to the presence of strength related neural adaptations: (1) a time course of strength gains that precedes changes in muscle size, (2) a lower requisite training intensity compared with that necessary for voluntary training, and (3) increased strength of the non-exercised contralateral limb that accompanies the strengthening of the test limb with NMES.

Finally, another major limitation for the use of NMES modality is that the long-term effectiveness following discontinuation is not well investigated (Doucet, Lam & Griffin, 2012). According to Doucet et al. (2012), few studies have follow-up data after treatment. Therefore, NMES may not be a long-term intervention for muscle re-education or

restoration of movement. However, for patients with spinal cord injuries, Shields & Dudley-Javoroski (2006) have suggested that only long-term use of NMES helps to offset the muscle atrophy and complications of disuse.

LINGUAL ELECTRICAL STIMULATION

Electrical stimulation has also been applied with the tongue. Application to the tongue is not a novel idea. Historically, electrical stimulation of the tongue has been used as early as 1955 to examine the sensory effects of such stimulation (Pierrel, 1955). Pleasonton (1970) used electrical stimuli (delivered short electrical pulses) to investigate patterns of sensitivity to contrast the tip and dorsum, midline and lateral areas, and right and left sides of the tongue using electrodes that were placed on the tongue.

Table 1. Summary of Studied NMES applications

Author	Applications
• Randerath (2006)	• strengthening skeletal muscles in pathological or posttraumatic situations • inducing motor units that are difficult to activate voluntarily
• Binder-Macleod, Halden & Jungles (1995) • Binder-Macleod & Lee (1997)	• enhancing muscle strength • retarding muscle atrophy • reducing spasticity
• Doucet, Lam & Griffin (2012)	• reducing edema • healing tissue • decreasing pain
• Daly et al. (1996)	• motor recovery in SCI, CVA, or TBI after the use of motor prostheses
• Gibson et al. (1988) • Snyder-Mackler et al. (1995)	• preservation of muscle mass and function during prolonged periods of disuse or immobilization
• Caggiano et al. (1994)	• improving of muscle function in healthy elderly subjects
• Babault et al. (2007) • Delitto et al. (1989) • Maffiuletti et al. (2002a) • Pichon et al. (1995)	• strengthening skeletal muscles in recreational and competitive athletes
• Oliven et al. (2009) • Hu et al. (2008) • Steier et al. (2011) • Oliven, Schnall, Pillar, Gavriely, and Odeh (2001)	• maintaining open airway in patients with obstructive sleep apnea
• Kavanagh et al. (2012)	• facial NMES to improve zygomatic major thickness.
• Humbert et al. (2006) • Ludlow et al. (2007) • Nam et al. (2013) • Barnaby-Mann and Crary, (2007) • Clark, et al. (2009) • Ludlow (2010)	• dysphagia rehabilitation

Most of the studies found in the literature related to lingual electrical stimulation investigated its effect on sleep apnea. Lingual neuromuscular electrical stimulation effects on airway patency were investigated in patients with obstructive sleep apnea (Isono, Tanaka & Nishino, 1999; Mezzanotte, Tangel & White 1992; Miki, et al., 1989; Oliven, Schnall, Pillar, Gavriely, Odeh & 2001; Randerath, 2006). In these studies, different approaches to the delivery methods for applying electrical pulses to the lingual muscles have been investigated using: (1) intramuscular NMES (Oliven et al., 2001; Oliven et al., 2009; Hu et al., 2008; Decker et al., 1993), (2) submental surface NMES (Steier et al., 2011; Yang, Meng, & Zhu, 2000; Guilleminault et al., 1995; Decker et al., 1993), (3) lingual surface NMES (Isono et al., 1999; Schnall et al., 1995), and (4) sublingual NMES (Oliven, Schnall, Pillar, Gavriely, Odeh, 2001).

The aim of these studies was to explore the effect of NMES in maintaining open airway in patients with obstructive sleep apnea. The airway is typically obstructed in patients with sleep apnea due to pharyngeal and lingual weaknesses (Remmers et al., 1978; Oliven et al., 2003); thus, applying electrical stimulation to the genioglossus muscle (Oliven et al., 2003) pushes the tongue superiorly and anteriorly, leading to opening of the airway. Although these studies focused on the single time effect in relation to obstructive sleep apnea, they demonstrated that surface electrical stimulation can be safely utilized to stimulate extrinsic lingual muscles.

ELECTRICAL STIMULATION AND FACIAL MUSCLES

Electrical stimulation has also been investigated with facial muscles though not extensively. The facial muscles are superficial and would be targeted easily by electrical stimulation. However, few studies to date have investigated the effect of NMES on facial muscles, generally, and muscles of speech production and swallowing, specifically. In a study that targeted the zygomatic major muscle, Kavanagh et al. (2012) concluded that following a 12-week course of facial NMES, the thickness of the muscle was increased as measured by ultrasound and there were subjective improvements in facial characteristics.

A limited number of studies have investigated the application of NMES within the facial palsy population (Cronin & Steenerson, 2003; Hyvärinen et al., 2008; Alakram & Puckree, 2011). In a retrospective case review of 24 patients with facial paralysis who received neuromuscular facial retraining, Cronin & Steenerson (2003) concluded that all patient groups made significant improvements in function with improved symmetry in dual-channel electromyographic readings. The subjects also were found to have increased facial movement based on percentages of movement measured in the study.

In a study that targeted participants with chronic facial nerve paralysis with sensory level NMES, a significant improvement was observed in the upper branch of the facial nerve motor action potential distal latency on the affected side in all patients. An improvement of one grade on the House-Brackmann scale was observed, and some patients also reported subjective improvement (Hyvärinen et al., 2008).

Another study investigated the NMES as a treatment approach for Bell's palsy in the acute phase of the disorder in conjunction with other modalities (i.e., heat, massage, exercises). Results of the study demonstrated that the effects of electrical stimulation, as used in that study, were found to be clinically, but not statistically significant (Alakram & Puckree, 2011). To date, no studies exist in the field of communication sciences and disorders that have examined the effect of NMES on the strength of muscles that are involved in articulation and swallowing -- such as labial muscles (i.e., the orbicularis oris, levator and depressor muscles), buccinator, and masseter -- despite the fact that these muscles can be easily targeted by surface NMES.

ELECTRICAL STIMULATION AND DYSPHAGIA

Recently, surface electrical stimulation has been explored as a treatment option in patients with neurogenic dysphagia, a disorder that involves oropharyngeal weakness. Most of the studies published are related to neuromuscular electrical stimulation and pharyngeal phase dysphagia and airway protection (Humbert et al, 2006; Ludlow et al,

2007; Nam et al., 2013). These studies investigated laryngeal and submental electrode placements. Systematic reviews presented a number of promising findings and findings having no benefit of NMES over traditional, non-electrical stimulation treatment. These reviews concluded that their findings warrant the need for more controlled trials to assess effectiveness of NMES (Barnaby-Mann & Crary, 2007; Clark, Lazarus, Arvedson, Schooling, & Frymark, 2009; Ludlow, 2010).

In a study exploring the effect of electrical stimulation on swallowing in chronic pharyngeal dysphagia, Ludlow, et al. (2007) noted that the depression in hyolaryngeal elevation due to electrical stimulation may serve as resistance during muscle activation in swallowing, thus improving muscle strength. Humbert, et al. (2006) and Nam, et al. (2013) examined the effect of submental placement of electrodes on laryngeal movement. Humbert, et al. (2006) showed that surface submental stimulation alone produced no elevation of the hyoid, with only some anterior movement. However, Nam, et al. (2013) demonstrated that stimulating both the suprahyoid and the infrahyoid muscle resulted in a significant increase in superior laryngeal elevation but not in anterior hyoid excursion.

In a meta-analysis conducted by Carnaby-Mann & Crary (2007) that examined the evidence on neuromuscular electrical stimulation for swallowing rehabilitation, the authors concluded that there is a statistically significant summary size effect that supports the use of neuromuscular electrical stimulation to treat swallowing disorders. Furthermore, clinicians who are using surface NMES for dysphagia treatment reported positive clinical outcomes with no treatment-related complications and high patient and professional satisfaction (Crary et al., 2007) suggesting a functional benefit of this treatment modality.

Additionally, Ludlow and her colleagues (Martin et al., 2010) investigated the effect of intramuscular electrical stimulation in the larynx in 10 patients with dysphagia post radiation therapy and 9 healthy volunteers. This study demonstrated that intramuscular stimulation can augment hyo-laryngeal elevation and might be able to increase airway protection during swallowing. Their data provided promising findings for the use of intramuscular electrical stimulation in individuals with dysphagia.

The effect of NMES on swallowing in dysphagia studies is controversial, possibly because the laryngeal elevator extrinsic muscles are deep within the neck and cannot be easily accessed by surface NMES. However, studies have demonstrated that surface NMES placed in the laryngeal and submental region have reached the supralaryngeal muscles. Hence, NMES resulted in a movement of the hyoid bone despite any argument that this movement assisted in the physiology of swallowing or acts against it. Additionally, since intramuscular electrical stimulation is applied directly in the targeted muscles, it showed that direct electrical stimulation to the muscles produce favorable effects.

SUMMARY AND CONCLUSION

Lingual and facial muscles of mastication and expression are important for communication and swallowing. Speech-language pathologists are in need of modalities to assist in the treatment when muscle weaknesses exist. NMES is a modality that is commonly used in physical therapy and occupational therapy fields that assists in treating several motor and sensory muscular disorders including muscular weakness.

The literature reviewed demonstrate that very limited data related to the use of NMES on orofacial muscles exist despite the fact that these muscles can be easily accessed by electrical stimulation from the surface. Thus, the present review of the research using electrical stimulation of muscles highlights the need for experimental treatment studies that investigate the effect of NMES on orofacial weakness. NMES has been shown to have promising potential as a treatment modality for muscle weakness.

Theoretically, this positive effect should translate into improved strength when applied in the orofacial muscles. However, before incorporating this procedure into clinical practice, research is needed to investigate its effects on LLBW. Furthermore, research is needed to evaluate the impact of NMES on patients with diminished motor function of the

labial, lingual and buccal muscles to demonstrate effectiveness, efficacy, and safety of this procedure. Additionally, there is a need for future studies that evaluate electrode placement effect of NMES on LLBW as well as the most effective characteristics of the applied current used for stimulation.

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