Neuromuscular Characterisation of Dysphagia Following Stroke

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Abstract

Dysphagia following stroke consists a complex set of symptoms that arises from impaired functioning of the skeletal muscles involved in swallowing. This impairment results from reduced ability of the muscles to generate the optimum force needed to perform the required actions, the symptom known as muscle weakness. It is commonly believed that muscle weakness in dysphagia results from a decrease in muscle activity due to reduced voluntary motor unit recruitment. Therefore, strengthening exercises are predominantly chosen to rehabilitate dysphagia; which however may not be a universally applicable solution. This is because muscle overactivity characterized by increased involuntary motor unit recruitment is also a characteristic of stroke patients and can interfere with generation of required muscle force. Presently, muscle overactivity in dysphagia diagnosis and management remains unexplored in contrast to limb muscles that are also structurally classified as skeletal muscles (like swallowing muscles) but have been extensively investigated for both muscle overactivity and weakness following stroke. Here we analyse the previous research exploring the neuromuscular pathophysiology of dysphagia and make a corresponding analogy of swallowing muscles with the limb muscles to suggest possibilities of muscle overactivity as a potential pathophysiological factor underlying dysphagia so that target specific rehabilitations could be designed.

Keywords: Stroke, Dysphagia, Neuromuscular pathophysiology, Muscle over activity, Rehabilitation.

Introduction

Swallowing is a process vital for human survival [1]. The simple process requires a complex neuromuscular interaction for a food bolus to pass from the oral cavity to the oesophagus via the pharynx while keeping the airway protected [2]. Difficulty in the process of swallowing, known as dysphagia, may arise due to a wide range of neurological disorders and most commonly seen in patients following stroke. Systematic review of the epidemiological studies of dysphagia worldwide has found that 8.1–80 % of stroke patients, were diagnosed with dysphagia [3]. Dysphagia affects hydration and nutritional status of patients with stroke and delays their recovery [4] and can lead to serious consequences like aspiration pneumonia, choking, and even premature death [5, 6]. Looking at the severity that dysphagia poses, there is an emerging insufficiency in the corresponding treatment modalities. Studies have suggested that efficacy of treatment protocols currently used in dysphagia management are limited [3, 7-9].

One of the reasons for such limitation could be the inadequacy of pathophysiological understanding. The present review aims to bridge the gap between the treatment modalities adopted for dysphagia and its efficacy by reporting different evidences that can guide to appropriate understanding of the impaired neuromuscular physiology of muscles involved in swallowing.

Swallowing occurs in a highly coordinated sequence of events through the interaction of 8 cranial and 2 spinal nerves involving more than 30 muscles [10-12], and is divided into three phases [12]. The oral phase involves the
The central execution of motor command also known as paresis/weakness [22].

Following the lesion there occurs gradual adaptive changes in both higher neural centers, target muscles and soft tissue [23]. One consequence of these gradual changes is the progressive development of abnormal responses to muscle stretch in the paretic body part also known as spasticity [23]. Patients with spasticity present a syndrome of muscle overactivity that constitutes the second fundamental factor of motor impairment seen in stroke patients along with weakness [23].

Moreover, muscles in the larynx and muscles involved in speech which are in neighbourhood to the muscles involved in swallowing (i.e. sharing similar anatomical location and neural innervations) have been found to reflect spasticity and is known as spastic dysphonia [24] and dysarthria [25], respectively. In a recent study, it was concluded that on treatment for hyperresponsiveness and increased muscle tension in larynx, the accompanied functional dysphagia also got resolved [26]. However, to our knowledge similar categorization of flaccid and spastic dysphagia following stroke has neither been characterised nor investigated.

Irrespective of the aetiology behind neurogenic dysphagia (weakness/spasticity), the conventional procedure follows strengthening of swallowing muscles as the only treatment protocol [27]. Such exercises include effortful swallows, Mendelsohn manoeuvre, Masako manoeuvre, Head Lift exercise, and expiratory muscle strength training. The strength training exercises are aimed to increase the range of motion by increasing the activity of the muscles involved in swallowing [28]. This may be an optimum choice for treatment of dysphagia in which muscle weakness accounts for the impairment. However, application of this treatment protocol may be insufficient in the case of dysphagia with muscle spasticity as the underlying cause. In stroke patients, there could be an increase in motor unit discharge due to loss of inhibitory input from the central nervous system (CNS) that results in muscle overactivity [23].

Muscle overactivity characterised as increase in involuntary motor unit recruitment can interfere with normal voluntary motor unit recruitment ability [23]. Hence, strengthening an overactive muscle can interfere with restoration of normal muscle activity [23]. Bobath hypothesised that strengthening an overactive muscle might be harmful as it can distort the motor control to that muscle [29]; though contradictions have also been reported [30, 31].

However, strong advocacy for following the relaxing modalities in restoring the functionality of an overactive limb muscle have been suggested [23, 32-36]. The practice of relaxing modalities is based on the principle that that overactive muscle requires a decrease in muscle activity [35, 36]. However, this controversy regarding following of appropriate rehabilitative approach, that is either strengthening or relaxing (or both) of swallowing muscles in dysphagia following stroke needs to be explored. In this regard, it is important to characterize the neuromuscular pathophysiology of dysphagia on the perspectives of both weakness and spasticity before making any decisions regarding the appropriateness of rehabilitative protocol followed in dysphagia treatment.

The current review will investigate the neuromuscular pathophysiology of dysphagia from the perspectives of weakness and spasticity. Section 2 will provide an overview of the neurophysiology and the subsequent sections will address the pathophysiology of swallowing. In this context, due to lack of extensive research in discerning the neuromuscular pathophysiology of dysphagia from the perspectives of weakness and spasticity, a simultaneous crosslink to research in limb muscles will be carried out.
Such evaluations will propel future studies in adopting the strategies used for limb muscles research in swallowing muscles. Both swallowing and limb muscles belong to the same category of skeletal muscles and presents the symptoms of paresis following stroke. We acknowledge that there are limitations about the applicability of techniques involved in limb muscle research to swallowing muscle due to their potentially different anatomical locations, nerve innervations, and functionality; however, they could be used as a backbone to platform scopes for similar research in dysphagia.

Neurophysiology of Swallowing

Swallowing could be of two types i.e. volitional and reflexive. For volitional swallowing, the motor cortex in the frontal lobe programs voluntary muscular movements; the subcortical area and the basal ganglia controls and stabilizes motor functions; the brainstem contains nuclei for the cranial nerves that regulate buco-facial, pharynx, and larynx muscles [4,5]. On a different note, reflexive swallowing is preprogramed in the brain stem nuclei that constitutes the central pattern generator [37]; although it can be activated by some specific regions of cerebral cortex [38].

Although stroke could damage the neural system at any of these level and result into dysphagia, however the present perspectives are limited to the lesions of the volitional swallowing because spasticity is a known characteristic of voluntary skeletal muscles.

Neuromuscular Pathophysiology of Weakness Following Stroke

To understand muscle weakness as one of the neuromuscular patho-physiologies in stroke, it is important to understand the physiology of muscle action. The action of a muscle is described as the development of tension or force in the muscle due to voluntary motor action potentials from the nervous system [39].

Tension is the force exerted by the muscle on the musculoskeletal structures and load is the force exerted by the musculoskeletal structures on the muscle [40]. This tension may or may not result in the displacement of the musculoskeletal structures depending on the proportion of the load [41]. If the tension is more than the load, it results in movement, whereas, no displacement could be seen when tension is less than the load, even though the voluntary stimulus from the CNS is present [42]. The amount of tension generated in a muscle depends on neural input [43]. The total muscle activity because of the neuronal input to a muscle depends on two aspects of motor unit physiology: the number of motor units recruited per unit area and the rate of discharge per motor unit [44-47]. A decrease in recruitment of motor units during voluntary tasks can result in the decrease in muscle activity in pathological condition of stroke; some authors refer to this as "weakness" [48, 49].

Decreased voluntary motor unit recruitment in a muscle can cause a reduction in the amount of force generated in a muscle [50, 51]. SEMG can be a way to measure the amount muscle activity as a result of voluntary stimulus for a given task [52-54]. SEMG can be recorded by a pair of electrodes placed over the surface of the skin that displays the voltage potential difference between the two electrodes [55]. In order to characterise decreased voluntary motor unit recruitment in swallowing muscles by SEMG, it is necessary to know how it has been done in limb muscles of stroke.

Weakness in Limb Muscles Following Stroke

The symptom of muscle weakness following stroke is referred to as a quantitative lack of muscle activity to the agonist muscles [49] resulting in reduced generation of force in the concerned muscle [44, 56].

This phenomenon in stroke has been quantitatively characterised in limb muscles by a decrease in integrated electromyographic (iEMG) value [57-59]. iEMG is defined as the area under the curve of the rectified SEMG signal, i.e., the mathematical integral of the absolute value of the raw SEMG signal over a period of a maximum voluntary contraction [53, 60]. iEMG is a process of amplitude analysis of SEMG that can provide a quantitative estimation of the amount of voluntary muscle activity for a given action [53].

Weakness in Swallowing Muscles Following Stroke

To our knowledge, works discerning the neuromuscular physiology of swallowing muscles are limited in their claims regarding the decrease in muscle activity. Many researchers use SEMG of the suprahyoid muscles as biofeedback in rehabilitative
treatment [61-70]. In these studies, the maximum amplitude of moving average curve (MAV) of SEMG is the parameter of choice [53]. In general, the conventional reporting strategy is to measure the MAV before the start and after the completion of treatment protocols on a subject to quantify the effectiveness of the treatment [61-70].

A systematic approach will be to report group comparisons of MAV between the healthy and dysphagic patients to establish the primary cause before applying the treatment. To our knowledge, only one study evaluated the MAVs in orofacial muscles for dysphagia from brain stem stroke patients and found an increase in MAV compared to healthy individuals [71]. More studies are required at this stage for different categories of stroke resulting in dysphagia to confirm or negate the longstanding assumption muscle weakness as the only underlying pathophysiology. Moreover, a strong reliable measure would be to estimate IEMG in comparison to MAV [60, 72].

Duration of SEMG has been another parameter often utilised in dysphagia research. Studies have shown that the duration of suprahyoid muscle SEMG in dysphagic patients is prolonged compared to healthy controls for patients with stroke patients like suprabulbar palsy with lacunar infarct [73], whereas in middle cerebral artery stroke patients the same duration is reported to be shortened [74].

This creates a controversy in generalising this parameter for every category of stroke causing dysphagia. Hence, due to lack of systematic approaches made in dysphagia research none of the parameters can be utilised as a direct measure to quantify decrease or increase in muscle activity for the muscles involved in swallowing. The present review highlights some gaps that are continued in dysphagia research and emphasises on a structured approach following similar studies as done in limb muscles to establish a parameter that accounts for muscle weakness in various categories of stroke patents causing dysphagia.

Neuromuscular Pathophysiology of Muscle Over activity (MO) Following Stroke

Muscle overactivity is referred to as increased involuntary motor unit recruitment [23]. This is seen in stroke as difficulty in suppressing the muscle activity, even when the muscle is at rest and devoid of voluntary stimulus [32]. The phenomenon of increased involuntary neuronal input can be seen either in the primary muscle or any other secondary muscle involved in the biomechanical movement [23, 75]. Upper motor neuron relaxes a muscle when its contraction is complete and inhibits the extra neuronal discharge to a muscle during both action and rest [76]. This inhibitory upper motor neuron input originates from the cerebral cortex and projects to the brainstem pathways [77]. Loss of this inhibitory input from the cortex following stroke increases the brainstem pathway discharge to the limb muscles.

This is due to the fact that brainstem pathways innervate the spinal nerve lower motor neuron [78, 79]. However, a homologous neuronal pathway innervating the cranial nerve lower motor neuron has also been demonstrated [80-83] broadening the scopes for muscle overactivity in muscles that are innervated by both cranial and spinal nerves.

As cranial nerves execute the process of swallowing (volitional or reflexive), so likelihood of muscle overactivity in dysphagia following the loss of inhibitory upper motor neuron input is maximised. In order to investigate the presence/absence of muscle overactivity in swallowing muscles, it is important to discuss how it has been investigated in limb muscles.

Muscle over Activity in Limb Muscles Following Stroke

Muscle overactivity following stroke has been observed as abnormal activation patterns, spastic dystonia, and hypertonia [23, 32] in the limb muscles. These features are clinically important for diagnosis and subsequent treatment in order to regain the function of impaired limb muscle [84]. We will discuss them in the subsequent sections.

Abnormal Activation Patterns

To describe abnormal activation patterns seen in limb muscles following stroke, a discussion about normal physiology of muscle activation patterns will facilitate understanding of the scenario.

The central nervous system regulates two types of activation patterns in order to perform a movement: 1) reciprocal inhibition of antagonist and agonist muscles i.e., when an
agonist muscle responsible for a movement undergoes activation, the antagonist muscle responsible for that same movement is inhibited; and co-activation of synergist muscles i.e., when one muscle is activated the synergist muscle is also activated simultaneously [85].

Abnormal activation patterns of muscles are core features seen in spastic paresis of stroke and could be identified by measuring SEMG activity of both the agonist and antagonist muscles. This has been extensively investigated in limb muscles [36, 86-88]. As discussed, the abnormal muscle activation pattern could also result in reduced generation of force in the muscle and might look like weakness.

Two varieties of abnormal activation patterns are demonstrated in limb muscles of stroke: co-contraction and inverse muscle activity. When the normal reciprocal inhibition of agonist and antagonist muscles fails to ensue, co-contraction of agonist and antagonist muscles occurs which results in reduced ability of a muscle to generate optimum force [89, 90]. For example, when the biceps and triceps are activated simultaneously, the antagonistic action of the triceps muscle prevents the agonist biceps muscle from flexing the elbow in stroke patients [91].

Similarly, in normal physiology, when both of the muscles of a synergist pair are active simultaneously, the cumulative force leads to the desired movement. However, when one muscle is active and the other is not, this results in a reduced amount of force, which in turn leads to muscle fatigue as a result of extra work [92-94]. The inactivity in the synergistic muscle is due to an increase in inhibitory function of upper motor neuron on the synergistic muscle [95].

**Spastic Dystonia**

Spastic dystonia is seen in muscles at rest and is characterised by the spontaneous burst of activities. This is commonly seen in the hemiparetic posture of stroke patients [32, 96, 97]. This has been recorded electromyographically in shoulder abductors, elbow flexors, and muscles of the wrist [97]. This phenomenon was first explored in monkeys and was referred to as spastic dystonia by Denny-Brown [98, 99]. In limb muscles, spastic dystonia is seen as an inability to relax after a voluntary contraction and is reflected in the prolonged firing of motor units [23, 100, 101].

**Hypertonia**

Before describing the abnormal muscle tone, it is important to discuss the physiology of muscle tone. When there is no voluntary stimulus from the CNS and the muscle is at rest, background tension so created is known as the tone of the muscle [41].

Muscle tone is vital for maintaining the erect posture in humans. Muscle tone is maintained by a reflex at the spinal level called the stretch reflex, a muscle contraction that occurs in response to stretch detected within a muscle [51]. This reflex is regulated from higher centres of the CNS including the reticular formation of the brainstem, the cerebral cortex and the cerebellum [46, 51]. Both increase and decrease in muscle tone can result in restricted movement of the musculoskeletal structures [102]. This increase in tone seen in stroke patients is known as hypertonia [102].

Hypertonia results from motor neurone hyperactivity due to either hyperactive stretch reflex or loss of inhibition from higher centres that regulate the stretch reflex at spinal level [46]. Muscle hypertonicity is demonstrated clinically as an increase in resistance when a muscle is passively stretched [46].

**Muscle Over activity in Swallowing**

**Muscles Following Stroke**

Crary & Baldwin (1997) measured SEMG of the perioral muscles, masseter and infrahyoid muscles in brainstem stroke patients and compared these with healthy individuals [71].

They found that the SEMG amplitude measures (MAV, baseline, onset to peak duration) of perioral and infrahyoid muscles were higher in dysphagia patients compared to normal individuals, which however may indicate muscle overactivity. Nevertheless, a controversy in this measure could exist due to the forceful voluntary effort made by dysphagia patient in an attempt to swallow normally thereby increasing the amplitude of SEMG recordings. Therefore, measures that are more reliable are required to confirm the presence of muscle overactivity. Such parameters could be obtained by following the approach made in the research on limb muscles so that features of muscle overactivity.
(abnormal activation patterns, spastic dystonia, and hypertonia) could also be established in swallowing. In the next section, previous studies on swallowing muscles that hinted in parts to these muscle overactivity features will be discussed and possible extensions to those studies will be suggested.

**Abnormal Activation Patterns**

A normal activation pattern of reciprocal inhibition is seen between the masseter and suprahyoid muscles during the opening and closing of the jaw in healthy individuals [103]. If activation pattern of these muscles is investigated in dysphagic patients following stroke, then abnormalities are expected. Demonstrating such abnormal activation pattern of co-contraction could also act as a biomarker for muscle overactivity in stroke patients.

**Spastic Dystonia**

Crary, Carnaby, & Groher have demonstrated a biomechanical correlation (in terms of timing and SEMG measures) between the trajectory of hyoid movement and SEMG signals of suprahyoid muscles in normal individuals [104]. Similar research could be implemented on dysphagia patients and group comparisons could be made to establish the significance of the obtained measures to establish the presence or absence of spastic dystonia. In these experiments, demonstration of spontaneous activity of suprahyoid muscles at the resting stage and the inability of suprahyoid movement to relax after the offset of biomechanical movement of hyoid can be a suggestive measure of spastic dystonia.

**Hypertonia**

Two studies reported by Clark et al (2015,2014) demonstrated an altered muscle tone for orofacial muscles of spastic dysarthria [105, 106]. Their study used myotonometer for measuring hypertonicity in orofacial muscles that are a small probe that is placed over the muscle of interest, which measures tissue compliance by sending a pulse of perturbation.

These studies demonstrated uniqueness in their effort to explore muscle stiffness (resistance of a muscle to deformation by tensile force). Muscle stiffness forms the nonneural component of muscle tone that assesses the viscous and elastic properties of soft tissue [107]. Although, the same orofacial muscles are involved in both speech and oral phase of swallowing, but to our knowledge, a similar attempt of investigating the muscle tone of orofacial muscles in dysphagic patients has not been reported.

**Limitations**

We acknowledge the fact that the principles and techniques of characterising the neuromuscular pathophysiology in the limb muscles may not be directly employed in swallowing muscles due to many evident barriers of which two majors are (1) Physiological barrier: It is important to note that the swallowing muscles are supplied by corticobulbar system whereas limb muscles are supplied by the corticospinal system. Limb muscles are involved in only volitional function whereas swallowing muscles are involved in both volitional and reflexive function. Although the neuromuscular physiology reflected may vary, but nevertheless, the research on limb muscles could act as a baseline for its implementation in dysphagia because both belong to the category of skeletal muscles and present the symptoms of weakness muscles and present the symptoms of weakness following stroke.

Therefore, characterising dysphagia from the angles of muscle overactivity cannot be further neglected (2) Technological barriers: Anatomical location of the swallowing muscles also adds to its inaccessibility for non-invasive direct measures to be applied on them. For example, passive stretching needed for demonstration of muscle tone applied on limb muscle cannot be applied on swallowing muscles. Hence, care needs to be taken in the design of the experimental measures to be used for dysphagia.

**Summary**

The article discussed in detail the neuromuscular pathophysiology that can lead to reduced movement of swallowing structures. The review highlighted muscle weakness in dysphagia following stroke and the two probable pathophysiology muscle overactivity and weakness responsible for addressing the symptoms. Segregating the two pathophysiology before following the conventional treatment protocol of strengthening exercises would be an important step in dysphagia diagnosis and management as suggested from various viewpoints cited in the present review. Such segregation can have a direct translational impact as it will indicate remodelling of the rehabilitative approach
followed in dysphagia in three ways namely (1) The presence of muscle overactivity will indicate that the current emphasis on strengthening followed as rehabilitative protocols in dysphagia may not be always appropriate. Therefore, dysphagia will need to be categorised based on the pathophysiology of the swallowing muscles, and rehabilitation specific to the underlying cause needs to be advocated; (2) The absence of muscle overactivity and presence of weakness as a stand-alone factor will indicate that the current rehabilitative approach is appropriate and could be reinforced in patients for more efficient recovery; and (3) A combination of muscle overactivity and weakness would suggest that rehabilitative approach needs to be optimised targeting both causes. Categorising the treating protocol according to the above-mentioned factors will facilitate better recovery of swallowing impairments seen in dysphagia.

References


