University of Alberta

Respiratory, laryngeal, and articulatory adjustments to changes in vocal loudness in typically developing children and children with spastic-type cerebral palsy

by

Erin Dale Archibald

A thesis submitted to the Faculty of Graduate Studies and Research
in partial fulfillment of the requirements for the degree of

Master of Science
in
Speech-Language Pathology

Department of Speech Pathology and Audiology

Spring 2011
Edmonton, Alberta

EXAMINING COMMITTEE

Dr. Carol Boliek, Speech-Language Pathology

Dr. Megan Hodge, Speech-Language Pathology

Dr. Benjamin Tucker, Linguistics

ABSTRACT

This study explored the physiological adjustments made by the speech mechanism when sustained maximum phonations and sentences differing in vocal loudness were produced by typically developing children and children with cerebral palsy (CP). Respiratory adjustments (lung volume initiation, termination and excursions), chest wall muscular amplitude adjustments (intercostal, obliques), vocal fold adjustments (speed quotient), fundamental frequency of selected vowel nuclei and area of mouth opening were calculated. A total of eight children (4 typically developing children, 4 children with CP) were studied. Results indicated that overall typically developing children adjusted lung volume initiation, lung volume excursion, intercostal and oblique muscle activity, speed quotient, fundamental frequency, and area of mouth opening to meet vocal loudness targets. In contrast, children with CP primarily adjusted intercostal and oblique muscle activity, speed quotient, and fundamental frequency to meet vocal loudness targets.

ACKNOWLEDGMENT

It is a pleasure to thank those who made this thesis possible. I am thankful to my supervisor, Carol Boliek, for her guidance, support, and dedication to this study. I also want to thank my thesis committee members, Megan Hodge and Benjamin Tucker, for their expert advice and support. I am grateful to my parents, Dale and Arlyne Archibald, my brothers, Brett and Darrell Archibald, my partner Vincent Levesque, and my friends for their support and encouragement. I give special thanks to the children and families that participated in this study. This work was supported, in part, by the Women & Children's Health Research Institute in Edmonton AB, the Queen Elizabeth II Graduate Scholarship sponsored by Alberta Scholarship Programs, and the Edmonton Oilers Community Foundation.

Table of Contents

List of Tables

List of Figures

LIST OF ABBREVIATIONS

CP: Cerebral Palsy

MP: Maximum Sustained Phonation

VC: Vital Capacity

OP: Oral Pressure

LVI: Lung Volume Initiation

LVT: Lung Volume Termination

LVE: Lung Volume Excursion

IC: Intercostal Muscles

OB: Oblique Muscles

%MVE: Percent Maximum Voluntary Effort

SQ: Speed Quotient

F_0: Fundamental Frequency

MA: Area of Mouth Opening

INTRODUCTION

Variations in vocal loudness occur naturally in healthy speakers during conversation. The ability to adjust loudness is important for effective communication. Abnormalities of loudness control (e.g., soft voice, monoloudness, uncontrolled or variable loudness) are often associated with the speech of individuals who have neurogenic communication disorders (Yorkston, Beukelman, Strand, & Bell, 1999). Of particular interest are children with cerebral palsy (CP), who often exhibit difficulties managing vocal loudness in conversation. Voice and speech treatments for children with CP are limited and lack clinical effectiveness (Yorkston, 1996). As a consequence, this population continues to be under-served and marginalized. The overarching aim of the research study was to inform future therapy strategies designed to enhance oral communication in children who have motor speech disorders. The purpose of the study was to provide a more detailed understanding of the physiological adjustments made by the speech mechanism when producing sentences differing in vocal loudness (sound pressure level) in typically developing children and children with spastic-type CP.

The sound pressure level (SPL) of the speech signal is a measure of its physical magnitude. Sound pressure measurements are made using a decibel scale (dB SPL). Sound pressure level for running speech activities is conditioned by adjustments of the breathing apparatus, laryngeal apparatus, and pharyngeal-oral apparatus. The strongest perceptual correlate of SPL is loudness such that the higher the SPL, the greater the magnitude of perceived loudness (Baken &

Orlikoff, 2000). Loud speech often involves a deeper inhalation, a greater degree of vocal fold closure, and larger articulatory excursions compared to speech produced at typical conversational levels (Dromey & Ramig, 1998b). During loud speech production, vowels increase in dB SPL more than consonants. In addition, sonorant sounds tend to increase in duration, whereas stop consonants tend to decrease in duration during loud speech (Dromey & Ramig, 1998b). Variations in vocal loudness carry important suprasegmental information of spoken language (Dromey & Ramig, 1998b).

Changes in vocal loudness levels enhance prosody, emotional content, and intelligibility of speech (Baker, Ramig, Sapir, Luschei, & Smith, 2001). Inter and intra-individual differences have been observed in the way speech apparatus adjustments are made to achieve changes in vocal loudness. For example, Stathopoulos & Sapienza (1993) reported that some individuals rely on deeper inhalations before speaking to take advantage of the greater recoil forces of the lung and chest wall available at higher lung volumes. Others more fully adduct the vocal folds to raise SPL without significant increases in the lung volume initiations. Makiyama, Yoshihashi, Mogitate, & Kida (2005) reported that vocal loudness is controlled by respiratory effort when laryngeal adjustment in the form of increased laryngeal resistance, is poor. For example, people with vocal fold paralysis showed marked increase in respiratory effort to achieve a large positive lung pressure when producing a loud phonation. In contrast, if there is adequate ability to adjust the larynx, vocal loudness is controlled by making coordinated laryngeal and respiratory adjustments for achieving increased laryngeal airway

resistance. Children with CP are known to have difficulty making these coordinated laryngeal and respiratory adjustments (Netsell, Lotz, Peters, & Schulte, 1994).

Cerebral palsy is the most common movement disorder in children. It is "…an umbrella term for a group of non-progressive motor impairment syndromes secondary to lesions or anomalies of the brain arising in the early stages of its development" (Mutch, Alberman, Hagberg, Kodama, & Perat, 1992, p. 549). The prevalence of CP in industrialized countries is 2 to 2.5 cases per 1000 live births (Gupta, 2001). Spastic CP is the most common form of CP and most often correlates with a lesion in the direct motor pathway including the motor cortex and its descending white matter tracts. Spasticity is characterized by increased tone, hyperactive reflexes, weakness, and poor coordination (Goldstein, 2001). It includes decreased coordination through loss of selective muscle control as well as decreases in speed of movement, strength and endurance (Goldstein, 2001). Children with spastic CP often have compromised respiratory and laryngeal systems. As a consequence, their speech may be characterized as having one or more of the following qualities: (a) breathiness, (b) variable voice quality, (c) monopitch, (d) hypernasality, and (e) reduced or monoloudness (Workinger, 2005).

Lee Silverman Voice Treatment (LSVT®LOUD) is a highly structured intervention program that focuses on increasing vocal loudness in adults with Parkinson disease (PD) and has been shown to improve vocal loudness and speech intelligibility in adults with PD (Ramig, Countryman, Thompson, & Horii,

1995). Initial findings by Fox and Boliek, (submitted) indicated that *LSVT®LOUD* has a similar positive effect on children with cerebral palsy (CP) who have voice and speech disorders. A single therapy target, vocal loudness, reduces the cognitive demands placed on a client during therapy. Dromey & Ramig (1998b) suggested that vocal loudness has a widespread and consistent positive impact across the speech subsystems. That is, a dysarthric speaker may be able to improve both voice and articulation by simply focusing on vocal loudness (Dromey & Ramig, 1998b).

An understanding of the biomechanical and aerodynamic adjustments, made by healthy children in an effort to change vocal loudness, may provide a model that emphasizes effective strategies for increasing loudness in children with motor-speech disorders. It is important for clinicians to understand how loudness adjustments lead to greater stability of performance, making it easier to predict the outcome of therapy (Dromey & Ramig, 1998b). Understanding the underlying adjustments used to produce variations in vocal loudness will provide useful information to clinicians using interventions like *LSVT®LOUD*. Clinicians can then determine where in the speech mechanism adjustments are being made and whether or not those adjustments are appropriate for achieving healthy voice and speech outcomes.

There is little information about speech mechanism adjustments to changes in vocal loudness. Dromey & Ramig (1998 a;b) studied the effects of manipulating rate, sound pressure level, and lung volume across three subsystems: respiration, phonation, and articulation in healthy adults. Only one other study

investigated laryngeal and respiratory function in children during the production of syllable trains at different sound pressure levels (Stathopolus & Sapienza, 1997). Whereas these studies provided initial observations about the relationship between vocal loudness and related changes in the speech subsystems, they did not study: (a) children unencumbered (e.g., without a face mask, head/chin supports); (b) production of phrases; (c) children with neurogenic communication disorders; (d) chest wall muscle activity (EMG) related to the respiratory subsystem; or (e) impedance measurements [electroglottograph (EGG)] associated with vocal fold behaviour. The addition of EMG and EGG data expanded the current understanding of vocal loudness adjustments made by the respiratory and laryngeal subsystems; respectively. The measurement protocol allowed children to produce sentences while remaining unencumbered and in a more natural communication scenario.

Background

Part I of this section, includes a description of the respiratory, laryngeal, and oroarticulatory speech subsystems with emphasis on typical development and the role each subsystem plays in producing speech at different loudness levels. Part II of this section includes a review of voice and speech disorders of children with spastic-type CP. Information regarding how adults with dysarthria adjust their speech mechanism to achieve vocal loudness follows and served as a basis for making some predictions about how children with CP might respond to tasks designed to manipulate vocal loudness. Potential implications of findings from the current study are outlined with respect to using *LSVT®LOUD* for treating children

with CP who have voice or speech disorders. The research questions, predictions, methods, data analysis, results, and discussion follows.

Part I - Typical Development of the Speech Subsystems

Respiratory Subsystem

Running speech. The respiratory system provides the driving force necessary for phonation and speech production. The displacement of the abdomen and rib cage creates the necessary air pressure behind valves as well as airflow through cavities within the larynx and upper airway (Hixon, Goldman, & Mead, 1973). Subglottal pressure must be maintained between 5 and 7 cmH_2O for speech production at conversational loudness. During an utterance, the average resistance offered by the larynx and upper airway is constant, although there are slight variations based on individual phonemes produced. Flow and alveolar pressure increase sharply at the onset of speech, maintain constant values during the utterance, and abruptly decrease at the end of the utterance (Hixon et al. 1973). Speech is produced acoustically by the disturbances of air via the respiratory-laryngeal interface in combination with intricate and rapid movements of downstream articulators. Generally, conversational speech begins at twice the resting tidal breathing depth and continues to near resting expiratory level or end expiratory level (EEL) of the breathing apparatus. The respiratory subsystem of the speech mechanism is involved in the regulation of loudness, linguistic stress, and the division of speech into various linguistic units and breath group lengths (e.g., words or syllables produced on a single expiration).

Both muscular and non-muscular forces regulate the respiratory apparatus for conversational speech. The volume of the rib cage wall and abdominal wall decreases throughout the breath group in running speech produced in an upright body position. The rib cage wall volume decreases at a faster rate than the abdominal wall volume and contributes approximately 80 percent of the lung volume excursion (Hixon, Mead, & Goldman, 1976). With decreasing lung volume, expiratory muscles must exert greater muscular pressure to overcome the smaller recoil force of the respiratory apparatus (Hixon, 1973). During conversation, there are frequent demands for rapid changes (75 to 150ms) in muscular pressure (1 to 3 cm H_20), which are important for stress contrasts (Hixon, 1973).

Lung volume events during running speech activities typically occur in the midrange of the vital capacity (VC) (Hixon, 1973). The muscles most significant for maintaining steady alveolar pressure are the internal intercostals (Hixon, 1973). The internal intercostals fill the small spaces between the ribs and serve to briefly contract when the utterance requires rapid, small pressure variations. The internal intercostals influence loudness since they create rapid, small variations in driving pressure supplied to the larynx and upper airway. Large changes in pressure, such as those required for emphatic utterances, involve additional abdominal muscle activity (Hixon et al., 1976).

Mechanical factors are partially responsible for explaining why speech activities most often occur in the midrange of the vital capacity. At the upper and lower ends of the vital capacity, the respiratory apparatus is stiffer and requires

greater muscular forces to control, in comparison to the mid-volume range (Hixon, 1973). When utterances are produced above the resting expiratory level, a speaker can take advantage of the positive recoil of the respiratory apparatus to help control the speech mechanism.

Relaxation pressure is the pressure produced by the passive force of the breathing apparatus; it varies with lung volume. The most positive relaxation pressure is created at the largest lung volumes and the maximum negative relaxation pressure occurs at the lowest lung volumes. The relaxation pressure is positive for most running speech. As a result, little muscular energy is required against inspiratory recoil forces. Speech produced at volumes below the resting level would require more muscular energy. When speech infringes on the expiratory reserve volume, muscular pressure is exerted against a negative relaxation pressure (Hixon, 1973). Relaxation pressure and muscular pressure both contribute to conversational speech.

Respiratory control of vocal loudness. Compared to lung volumes used for speech produced at a typical loudness level (40 to 60% VC), loud speech demands higher alveolar pressures and is typically initiated from higher lung volumes (approximately 60 to 80% VC) to take advantage of the higher respiratory recoil forces available. Soft speech requires low alveolar pressures, often initiated at lower lung volumes (generally above end expiratory level) compared to speech of normal loudness. Expiratory phrases in soft speech end near the same lung volumes as speech of normal loudness (slightly above or at resting expiratory level; 35to 40% VC), whereas loud utterances are often terminated at lung

volumes above the resting expiratory level (Hixon, 1973). Stathopoulos & Sapienza (1993) reported that, for loud speech, there are significantly higher lung volume and rib cage initiations and excursions and a higher rib cage volume contribution. The rib cage accounts for a larger surface area than the abdomen; thus displacement of the rib cage to generate a particular lung volume for speech is more efficient than displacing the abdomen (Stathopoulos & Sapienza, 1997). The rib cage and abdomen play an important role in loudness modulation.

The key role of the respiratory system in loudness control is to modulate tracheal (subglottal) pressure. Finnegan, Lushei, & Hoffman (2000) suggested that a change in tracheal pressure is primarily due to a change in alveolar pressure. During speech, increases in muscular pressure are related to the difference between the alveolar pressure desired and the prevailing relaxation pressure. The alveolar pressure demanded for speech is the most important factor influencing the depth of inspiration during conversational utterances (Hixon, 1973). According to Hixon and colleagues (Hixon et al., 1976), greater inspiratory muscular pressures are used by the chest wall during inspiration and increased expiratory muscular pressures are applied during expiration of the same breathing cycle for loud conversational speech. A larger inspiration results in greater potential respiratory energy. The release of this energy, in conjunction with increased expiratory drive, raises alveolar pressure above its normal loudness level for speech (Hixon et al., 1976). The amount of inspiratory "braking" depends on the vocal loudness target, lung volume initiation relative to vital capacity, and length of the intended utterance.

Hoit, Plassman, Lansing, & Hixon (1988) studied the role of expiratory muscles of the abdomen in speech production via electromyography (EMG). They studied ten healthy 22-29 year-olds (5 men, 5 women) while performing maximal effort manoeuvres, resting tidal breathing, and four speech production tasks. These speech production tasks included: conversational speaking, reading at normal loudness, reading at twice-normal loudness, and counting throughout the vital capacity. During the aforementioned tasks, the lateral region of the abdomen was highly active in the upright position (obliques & transverse abdominis), whereas EMG activity was negligible in the middle region (rectus abdominis). Hoit et al. (1988) suggested that the lateral group of abdominal muscles plays an important role in configuring the chest wall during upright resting tidal breathing and speech production, whereas the rectus abdominis does not. Abdominal EMG activity was greatest during loud speech production and during speech produced at low lung volumes.

Development. Throughout childhood and adolescence, the breathing apparatus increases in size and its composition and configuration change (Hoit, Hixon, Watson, & Morgan, 1990). Boliek, Hixon, Watson, & Morgan (1996) reported that the amount of air children expired per breath group increases with age and that this is clearly linked to physical growth of the breathing apparatus. In addition, there are changes in mechanical properties. These changes include increases in the outward recoil of the chest wall, decreases in overall compliance, and increases in the pulmonary apparatus's recoil pressure (Hoit et al., 1990).

In order to vocalize, infants and children employ highly variable speech breathing strategies, which include different: (a) lung volume initiations, terminations and excursions; (b) chest wall shapes; and (c) relative contributions of the rib cage wall and abdominal wall to lung volume change (Boliek, Hixon, Watson, & Morgan, 1996, 1997). Stathopoulos & Sapienza (1997) reported that lung width, lung length, and total lung capacity increase relatively steadily until 14-16 years of age. However, speech breathing values become adult-like between 10 and 12 years of age (Hoit et al., 1990; Stathopoulos & Sapienza, 1997).

Hoit et al., (1990) found that speech breathing was similar for the 10-, 13-, and 16-year-old groups. Children ages 10 to 16 years of age tended to produce breath groups that were: (a) initiated in the midrange of both the vital capacity and rib cage capacity; (b) terminated in the lower portion of both the vital capacity and rib cage capacity; (c) initiated at relatively large abdominal volumes; (d) terminated at slightly smaller abdominal volumes; and (e) produced using primarily rib cage contribution to lung volume change. Speech breathing in the 7 year-olds was quite different from that of the older children and adolescents (Hoit et. al., 1990). The 7-year-old children began and ended their breath groups at larger lung volumes, used more air per breath group, and produced fewer syllables per breath group than the older age groups. Hoit et al. (1990) interpreted the larger lung volumes as a strategy for producing higher alveolar pressures by taking advantage of higher recoil pressure at larger lung volumes, since younger children use higher tracheal pressures for speech production. However, inconsistent with Hoit and colleagues, Stathopoulos & Sapienza (1997) suggested that children use

more effort and are thus less efficient than adults (e.g., they terminate lung volumes substantially below end-expiratory level). Thus, with respect to the respiratory system, it appears that the key difference between children and adults is that children exhibit a variable speech breathing pattern that becomes more systematic by 10 years of age.

Laryngeal Subsystem

Running speech. Laryngeal muscle control requirements in humans differ for various laryngeal functions. Voice is produced as the vocal folds are positioned in the midline of the glottis and air flow from the lungs causes increases in the subglottal pressure that overcomes the resistance of the vocal folds and blows them apart (Ludlow, 2005). As the vocal folds open, air flows between the folds reducing the pressure between them, and the myoelasticity of the folds returns them midline, allowing the cyclic process to continue. This passive vibration will continue as long as there is higher subglottal than supraglottal pressure (Ludlow, 2005). The speaker must maintain adequate air flow from the lungs during exhalation and use adequate muscle activity to keep the vocal folds in the midline for vibration to occur.

The laryngeal muscles are either intrinsic (confined to the larynx) or extrinsic (attaching the larynx to other structures within the head and neck). Vocal fold movements are described as adductory or abductory. Intrinsic muscles are usually described as adductors or abductors, while the cricothryroid muscle elongates the vocal folds (Ludlow, 2005). The extrinsic laryngeal muscles change

the position of the larynx in the neck by raising or lowering the thyroid cartilage (Ludlow, 2005).

Individual differences in the use of laryngeal muscles during speech occur naturally. Changes in voice loudness depend on an interaction between increases in subglottal pressure and vocal fold tension (Ludlow, 2005). Finnegan et al. (2000) found that speakers control laryngeal muscles independent of subglottal air pressure to produce changes in dBSPL and fundamental frequency (F_0). The most difficult aspect of laryngeal muscle control for speech involves rapid and precise changes in vocal fold adduction and abduction for voice onset and offset, respectively. For example, the speaker must be able to vary subglottal pressure and laryngeal muscle activity to change vocal fold opening within a few milliseconds in order to mark certain linguistic contrasts that are critical to meaning, such as the distinction between voiced and voiceless sounds (e.g. /b/ versus /p/) (Ludlow, 2005).

Laryngeal control of vocal loudness. Speech produced during conversation may vary between 25 to 30 dBSPL (Hixon, Weismer, & Hoit, 2008). The laryngeal system plays a critical role in the modulation of vocal loudness. For example, increases in vocal loudness are associated with a longer closed phase of the vocal folds accompanied by greater adductory forces, resulting in an increase in laryngeal opposing pressure (Stathopoulos & Sapienza, 1993). These forces facilitate the build-up of tracheal air pressure, which increases as speech becomes louder (Hirano, Ohala, & Vennard, 1969). The muscles primarily involved in increasing vocal fold adductory forces are the lateral cricoarytenoid and

interarytenoid muscles. Finnegan et al. (2000) reported that thyroarytenoid (TA) muscle activity increases with increases in dBSPL; they suggested that, when the TA muscle contracts, it decreases the stiffness of the vocal fold cover, reduces strain, and accordingly allows greater amplitude of vocal vibration. Furthermore, TA contraction increases resistance and hence increases subglottic pressure (Finnegan et al., 2000)

Activation of the laryngeal muscles affects the amplitude and frequency of vocal fold vibration (Finnegan et al., 2000). Two underlying features of laryngeal behaviour are important to how efficiently energy from the breathing apparatus is converted into acoustic energy. These features include: (1) abruptness with which the vocal folds return to midline and airflow declines (rate of decline is related to strength of the input pressure required to make the vocal folds vibrate) and (2) average glottis size during higher sound pressure level production (Tang & Stathopoulos, 1995). Optimal acoustic power is generated when the average glottal size is halfway between that for tight adduction of the vocal folds and that for loose adduction of the vocal folds (Tang & Stathopoulos, 1995).

Both laryngeal opposing pressure and laryngeal airway resistance increase with increases in SPL. In order to contain the increased tracheal air pressure and prevent it from escaping, the opposing pressure must increase by contractions of the lateral cricoarytenoid and interartyenoid muscles (Ludlow, 2005) resulting in increased laryngeal airway resistance. Increases in tracheal pressure may be related to increased speed of vocal fold closure, increased air particle velocity through the glottis, increased fundamental frequency (perceived as pitch change),

and increased amplitude of vocal fold vibration (Stathopoulos & Sapienza, 1997). Furthermore, there are significant decreases in laryngeal open quotient measures when higher intensity levels are produced (Stathopoulos & Sapienza, 1993).

Development. From birth to adulthood, the laryngeal apparatus undergoes significant change. The structures of the laryngeal framework are soft and pliable at the beginning of life and increase in stiffness with age (Kent, 1976). In addition, the larynx migrates from the third to fourth cervical vertebrae at birth to the seventh cervical vertebra between 10 and 20 years of age (Hixon et al., 2008).

Children have a smaller larynx than do adults. This means children have a smaller glottal area and smaller amplitude of vocal fold vibration; this increases laryngeal airway resistance and consequently affects how aerodynamic energy is converted into acoustic energy (Stathopoulos & Sapienza, 1997; Tang & Stathopoulos, 1995). The infant larynx triples in size from birth to adulthood (Hixon et al., 2008). From infancy through adulthood, the mass of the individual intrinsic muscles of the larynx increase with age (Kahane & Kahn, 1984). Furthermore, children have relatively short membranous vocal folds with fewer elastic fibres and a greater density of fibroblasts than adults (Hixon et al., 2008). This means that vocal fold vibration is more difficult to control during childhood (Tang & Stathopoulos, 1995). Moreover, the multilayered structure of the lamina propria of the adult vocal folds is not apparent until about sixteen years of age. The difference in the lamina propria between infancy (homogenous and undifferentiated) and adulthood prevents the infant from producing the subtle vocal fold adjustments like those observed in adults (Kent, 1997). Consequently,

children have a different set of challenges for making phonatory adjustments, which may be a result of less efficient laryngeal valving (Stathopoulos & Sapienza, 1997).

During and following puberty, fundamental frequency decreases in males and to a much lesser extent in females. This decrease in fundamental frequency is due to an increase in mass and stiffness of the vocal folds, likely a result of the development of collagen and elastic fibres in the vocal ligaments (Tang & Stathopoulos, 1995). In addition, male vocal folds significantly increase in length and thickness (Hollien, Green, & Massey, 1994). Vocal fold maturation is not complete until 16-20 years of age (Stathopoulos & Sapienza, 1997).

Respiratory and laryngeal coordination for vocal loudness. Several studies have been designed to investigate the individual contribution of each speech subsystem for varying SPL, but few have defined the interactive function of these subsystems. Additionally, little is known about the developmental trajectory of these coordinative behaviours, especially between the respiratory and laryngeal subsystems. A seminal study conducted by Stathopoulos & Sapienza (1997) provided an initial evaluation of the respiratory and laryngeal role and function for controlling vocal loudness. In addition, the study addressed cross-sectional changes in development from 4 to 14 years of age. That work, in part, formed the basis for the present study.

As described previously, increases in SPL require increases in tracheal pressure. Increased tracheal pressure results in increased force acting on the vocal folds. Consequently, the vibratory pattern of the vocal folds adjusts and changes

the shape of the glottal airflow waveform, as well as the resulting harmonic structure (Stathopoulos & Sapienza, 1997). Stathopoulous & Sapienza (1997) examined how laryngeal and respiratory function varied with manipulation of sound pressure level in adults and children. They studied 140 individuals (twenty 4-, 6-, 8-, 10-, 12-, and 14-year old children and 20 adults, with 10 females and 10 males in each group). Each participant produced three trials of a syllable train consisting of repetitions of /pa/ at low, medium, and high sound pressure levels.

In order to observe laryngeal behaviour, Stathopoulos & Sapienza (1997) measured maximum flow declination rate of the glottal airflow waveform, open quotient, fundamental frequency (F_0), tracheal pressure, and alternating glottal airflow (i.e. flow maximum minus flow minimum). To study respiratory behaviour, they measured lung, rib cage, and abdominal volume initiations, terminations, and excursions. Results indicated that, as the SPL of the voice increased, greater alternating glottal airflows were produced and "greater vocal fold movement away from the maximum medial point reached by the edge at the closed phase occurred" (Stathopoulos & Sapienza, 1997). The 14-year-old boys and men generally had longer glottal closure time with increases in sound pressure level compared to the women and children (young children's vocal fold function resembled that of women). These differences were thought to relate to laryngeal size and vocal fold configuration (Stathopoulos & Sapienza, 1997). Stathopoulos & Sapienza (1997) also found that translaryngeal airflow was greater during the low intensity production compared to the medium intensity production. The greater translaryngeal airflow during the soft production may be

related to the increased glottal opening associated with a soft voice. Children produced a higher tracheal pressure than adults at each intensity level. In addition, there was a tendency for laryngeal airway resistance to increase with intensity, since increased stiffness maintains adduction when the tracheal pressure is high (Finnegan et al., 2000). According to Stathopoulos & Sapienza (1997), children's increased laryngeal airway resistance could result in increased tracheal pressure, as a result of smaller airway structures.

According to Stathopoulos & Sapienza (1997) F_0 increased with loudness. F_0 was most affected by tracheal pressure when the vocal folds were smaller. In 4-year-old children, there was a significant increase in F_0 as tracheal pressure increased. F_0 did not increase substantially as tracheal pressure increased in 6-year-olds, who have longer vocal folds. Women, 14-year-old girls and all of the child groups younger than 14 years showed similar F_0 patterns to each other and different patterns from the 14-year-old boys and men. The men and 14 year-old boys produced lower fundamental frequencies than the women and children, due to anatomical differences in vocal fold length and mass. The authors explained that laryngeal function measures must relate to laryngeal size rather than body size.

As vocal intensity increased from medium to high, all participants in the Stathopoulos & Sapienza (1997) study used larger lung and rib cage volume excursions. Participants exhibited significantly higher lung and rib cage volume initiations as well as significantly higher rib cage volume terminations. The 12- and 14- year-old children terminated their utterances closer to end expiratory

level; their pattern for increasing SPL was similar to that of an adult. Younger children had greater chest wall compliance, and accordingly, their ribcage volume terminations extended well below end-expiratory level.

One of the primary differences between adults and children in the Stathopoulos & Sapienza (1997) study was that adults used laryngeal adjustments to increase loudness, whereas the children did not. There were functional differences between how adults and children adjusted for loudness. These differences appeared to depend on the size of the laryngeal structures, the length and mass of the vocal folds, tracheal pressure, and laryngeal airway resistance. Further, Stathopoulos & Sapienza (1997) suggested that differences between older children and adults versus younger children, with respect to ribcage volume terminations, were due to differences in chest wall compliance.

Oroarticulatory System

Running speech. Muscle activity of the head and neck is important to speech production. A variety of combinations of postures and movements of the soft palate, pharynx, mandible, tongue, and lips produce different sounds of speech. The place and degree of constriction in the pharyngeal-oral airway, in addition to the presence or absence of lip rounding, influences which vowel, diphthong, or consonant is produced. The mandible, tongue, velum, and lips undergo continuous movement throughout sound sequences. Of specific interest to vocal loudness are lip and jaw movement.

Movement of the lips is highly versatile. In one utterance, lips may protrude, retract, spread, compress, thin, or thicken. Each lip is able to move

independently as well as coordinate with the other lip. More than a dozen facial muscles surrounding the mouth opening enable adjustments of the lips. Lips can move along different dimensions: vertical, side-to-side, and front-to-back. The upper lip is fixed to the maxilla, whereas the lower lip moves with the mandible. The mandible is able to move upward and downward, forward and backward, and side to side. It relies on the hinge-like and gliding actions of the temporomandibular joints. Seven muscles are involved in movement of the mandible.

Oroarticulatory control of vocal loudness. The characteristics of mouth opening during speech depend on the phonetic requirements of the utterance. Tasko & McClean (2004) performed a correlation analysis relating average lower lip and jaw-muscle activity to lip and jaw movement, distance, speed, and duration. Recordings were obtained on orofacial movement, muscle activity (i.e. mentalis, depressor labii inferior, anterior belly of the digastric, and masseter muscles), and the acoustic signal in three typical speakers as they repeated a simple test utterance at varying rates and loudness levels. Across the loudness conditions, surface EMG levels were positively correlated with movement speed and distance in all participants. Vocal loudness (dB SPL) varied systematically with increases and decreases in EMG level, movement, speed, and movement distance (McClean & Tasko, 2003). Strong linear associations between EMG level, speed, and distance were found.

Wohlert & Hammen (2000) obtained perioral surface EMG signals from 20 adults who read a paragraph at variable rates and loudness levels. They found

higher average EMG signal amplitudes for fast, loud, and precise speech and lower average amplitudes for slow and soft speech. Schulman (1989) noted that, with increases in SPL, there is not only an increase in subglottal pressure, but there is also an increase in the velocity and displacement of the articulators, resulting in larger lip opening and tighter lip compression during bilabial stops.

Schulman (1989) suggested that tongue height and oral opening must be adjusted to maintain formant relationships for vowels when airflow varies. Thus, changes in amplitude of lip-muscle activity may result from controlled articulatory movements for specific sounds. With respect to EMG amplitude, clear speech was like loud or fast speech. According to Wohlert & Hammen (2000), this represents a reorganization of motor control. Recordings from the lower lip had greater amplitudes than those from the upper lip. The lower lip showed activity from muscles responsible for opening and closing, and the upper lip showed activity from muscles responsible for lip closure or rounding.

Tasko & McClean (2004) evaluated how orofacial kinematic behaviour changed as a function of speaking task in a group of 15 healthy male speakers. Orofacial articulator motion was recorded using a Carstens AG100 Articulograph which tracks the motion of sensor coils attached to the skin using biomedical tape. Moving from soft to habitual to loud speech created systematic increases in stroke (movement) distance. Stroke distance is the distance traveled between stroke onset and offset. There were also increases in the standard distance for the upper lip, lower lip, tongue blade, and mandible. A similar pattern was observed for peak stroke speed; it was statistically significant for the gum line (between the

mandibular incisors) and the lower lip. This loudness-related scaling of kinematic events was more pronounced for nonsense phrases with greater kinematic demands on the oral articulators, compared to test utterances containing a broader repertoire of speech sounds, such as "combine all the ingredients in a large bowl." The gum line between the mandibular incisors and lower lip consistently shows significant increases/decreases in peak stroke speed, distance traveled between stroke onset and offset, and standard distance, with increases/decreases in loudness (Tasko & McClean, 2004).

Loud phonation has been associated with increased articulatory displacement and increased velocity for the upper and lower lips in typical adults as well as adults with Parkinson disease (Dromey & Ramig, 1998a). A more open vocal tract allows for more efficient radiation of acoustic energy. Accordingly, larger articulatory excursions can directly contribute to higher SPL. Dromey & Ramig (1998a) suggested that increased loudness may improve articulation, as larger lip displacements allow spatial targets to be met more easily. In addition, they noted that a decreased rate of speech increased lip displacement slightly, though the effects of rate on articulation were more variable and less predictable, compared to the effect of loudness on articulation.

Dromey & Ramig (1998a) studied the effects of manipulating lung volume on phonatory and articulatory kinematic behaviour during sentence production in healthy adults. Five men and five women repeated the sentence "I sell a sapapple again" under five lung volume conditions. These included: (1) speaking normally, (2) speaking after exhaling most of the air from the lungs, (3) speaking at end

expiratory level (EEL), (4) speaking after a maximal inhalation, and (5) speaking after a maximal inhalation while attempting to maintain normal speech. A head-mounted strain gauge cantilever system was used to track upper and lower lip movement during speech. Cantilever beams were guided through small beads attached to the speaker's lips with an adhesive tab. Upper lip displacements generally decreased for lung volume conditions that were higher or lower than normal lung volume conditions. There were no significant changes in lower lip displacements across lung volume conditions.

Dromey & Ramig (1998b) compared the effects of changing sound pressure level (SPL) on respiratory, phonatory, and articulatory behavior during sentence production. Five men and 5 women repeated the sentence, "I sell a sapapple again," under 5 SPL conditions. Loud speech led to increases in lung volume initiation, lung volume termination, fundamental frequency, semitone standard deviation, and upper and lower lip displacements. Again, they used a strain-gauge cantilever system to track upper and lower lip movement during speech.

To reiterate, upper and lower lip displacements increased in sentences spoken at louder than normal levels; lower lip displacement increased more than upper lip displacement. Dromey & Ramig (1998b) suggested that a more open vocal tract allows for more efficient radiation of acoustic energy. Accordingly, larger articulatory excursions can directly contribute to higher SPL.

Development. The pharyngeal-oral apparatus undergoes many changes from birth to adulthood. For example, the mandible increases in size and changes

in shape to accommodate the addition of permanent teeth (Zemlin, 1998). Also, the angle between the body and the ramus becomes less obtuse (Zemlin, 1998). Further, the junction between the pharyngeal and oral parts of the pharyngeal-oral apparatus develops from a rounded shape, found in the newborn infant, to a sharp right angle shape observed in the adult (Vorperian, Kent, Lindstrom, Kalina, Gentry, & Yandell, 2005). Moreover, the vocal tract doubles in length from birth to adulthood (Vorperian et al., 2005). As well, the tongue descends within the neck from the first year of life to about 6 years of age; growth of the tongue is continual throughout childhood and into puberty (Vorperian et al. 2005). In addition, the lips transition from a near circular sphincter (observed during infancy), to an elliptical shaped sphincter observed in adults (Burke, 1980). During this time, the muscle synergies for the lips develop and the diversity of movement offered by the lips changes.

Green, Moore, & Reilly (2002) compared adult upper lip, lower lip, and jaw movement patterns to those of children. They found that adult-like speech movement patterns emerge earlier in the mandible than in the lips. Specifically, they found that one-year-olds produced highly variable lip movements and their movements did not resemble adult movements. However, jaw movements produced by one-year-olds were highly similar to those of adults. Articulators showed highly stable movements within and across adult speakers but not within or across young children. “Speech development involves integrating lip movement into a relatively well-established mandibular movement pattern” (Green et al., 2002, p.75).

PART II - Voice and Speech Disorders of Children with Spastic-type CP

Children with mild to moderate quadriplegia spastic CP can exhibit voice or speech disorders (or both) which can be secondary to disorders of speech breathing. Children with severe quadriplegia spastic CP may show involvement of all speech subsystems (Workinger, 2005). As children with spastic CP grow, they spend increasing amounts of time in fixed positions, which cause contractures and can cause physical deformities. These contractures and deformities result in a regression in voice and speech production, especially in the areas of loudness, voice quality, and resonance (Workinger, 2005).

Respiratory muscle weakness and lack of muscle control during speech is common amongst children with CP. Children with spastic CP have weak expiratory musculature and generate expiratory air pressures significantly lower than typical children. They have reduced expiratory reserves, reduced inspiratory capacities, and reduced vital capacities, relative to typical children (Workinger, 2005). In addition, children with spastic CP show an inability to maintain a constant subglottal pressure across an utterance. Speech production in an individual with CP may be judged as adequate, if they have adequate valving at the level of the larynx, velopharynx, and/or orofacial structures. If, however, they do not have adequate valving, the respiratory system may have to work harder to maintain appropriate driving pressures for speech. Conversely, children with CP with compromised respiratory systems may adapt using compensatory adjustments of the laryngeal and oral-pharyngeal systems (Workinger, 2005).

Problems with laryngeal-respiratory coordination can cause dysphonia. Children with CP often demonstrate inefficient laryngeal valving of the airstream during speech production. Voice quality deviations in children with CP are related to difficulties making appropriate adductions and abductions of the vocal folds, as well as problems maintaining adequate vocal fold tension. Inefficient thryoarytenoid and lateral cricoartyenoid muscles could interfere with proper valving of the expiratory airstream and thus the regulation of vocal intensity (Baker, Ramig, Sapir, Luschei, & Smith, 2001). When the vocal folds are not adducted appropriately, or if there is inadequate tension, a breathy weak intensity voice results. When the vocal folds are hyper-adducted and very tense, a strained voice quality can result, potentially resulting in vocal nodules. Individuals with CP generally speak with lower vocal intensity, likely resulting from an inability to achieve and sustain adequate subglottal pressure (Workinger, 2005).

Children with spastic CP possess some abnormal oral movement patterns and postures that interfere with speech production. Specifically, they have difficulty with antagonist co-contraction of the depressor labii inferior and the orbicularis oris superior (Workinger, 2005). The depressor labii inferior muscle pulls the lower lip downward and toward the side; the orbicularis oris muscle moves the lips toward one another and forward. Furthermore, children with CP have difficulty coordinating articulatory movements. They reduce velocity of movement to achieve the appropriate range of movement needed to meet a particular target (Workinger, 2005). Factors affecting the speed, range of movement, force, timing, and accuracy of movement in their speech articulators

include abnormalities in: (1) muscle tone, (2) coordination, (3) strength, and (4) endurance of speech musculature. Primarily, individuals with CP have slower distorted speech, which is characterized by omissions, substitutions, and nasalization errors. In addition, children with spastic CP have relatively small vowel areas, which may interfere with speech intelligibility.

Speech mechanism adjustments for loudness have been studied in adults with typical speech and to a much lesser extent in children with typical speech development and production. The outcome of manipulating loudness in adults with hypokinetic dysarthria, secondary to Parkinson Disease, also has been studied. From this research, it may be possible to make inferences regarding how the speech mechanism adjusts when manipulating loudness in children with dysarthria, secondary to CP. Both of these groups have difficulty adjusting vocal loudness, which affects their speech intelligibility. The following section outlines the research regarding speech production in adults with dysarthria.

Speech Production in Hypokinetic Dysarthria

Individuals with hypokinetic dysarthria have reduced vocal loudness; a breathy, harsh, or hoarse voice quality; imprecise and reduced range of articulatory movements; reduced prosodic pitch inflection; and overall reduction in speech intelligibility (Sapir, Spielman, Ramig, Story, & Fox, 2007). For example, vowels tend to centralize due to limited movement of the speech articulators in dysarthric speech (Sapir et al., 2007), which has a negative impact on speech intelligibility. Both increased loudness and rate reduction have been reported to be associated with an increase in the size of the articulatory-acoustic

working space and better acoustic distinctiveness for speakers with dysarthria (Tjaden & Wilding, 2004).

Tjaden & Wilding (2004) studied fifteen speakers with dysarthria secondary to multiple sclerosis, twelve speakers with dysarthria secondary to Parkinson disease, and fifteen healthy controls. They read a passage in habitual, loud, and slow conditions. Vowel acoustic distinctiveness was maximized in the slow condition, but stop acoustic distinctiveness was maximized in the loud condition. Treatment aimed at increasing loudness would be preferred if the treatment goal was maximizing the acoustic distinctiveness of stops. Overall intelligibility (obtained using magnitude estimation) for speakers with Parkinson disease improved in the loud condition, relative to their habitual and slow conditions.

Kleinow, Smith, & Ramig (2001) reported improved measures of spatial and temporal movement stability for the lower lip, when speakers with hypokinetic dysarthria voluntarily increased vocal loudness. Other reports suggest an expanded vowel space area when speakers with hypokinetic or ataxic dysarthria increased vocal loudness after LSVT®*LOUD*. Ramig & Fox (2000) also reported improved intelligibility with the increased vowel space area for a speaker with ataxic dysarthria who received *LSVT®LOUD*. Articulatory displacements and speech intelligibility increase when speakers with dysarthria increase loudness (Tjaden & Wilding, 2004).

More recently, Sapir, Spielman, Ramig, Story, & Fox, 2007 found that individuals treated with *LSVT®LOUD* showed significant changes in vocal sound

pressure level, F2 of the vowel /u/, the ratio F2i/F2u, and "vowel goodness" ratings (how well an uttered vowel is judged to be an appropriate exemplar of an intended vowel). This change indicated improvement in vocal and articulatory functions. Sapir et al. (2007) suggested that enhancement in acoustic measures may be related to improved lip rounding, tongue movement, and/or laryngeal movement. *LSVT®LOUD* appeared to be an efficient way to boost many aspects of speech production in individuals with Parkinson's disease, who have an accompanying hypokinetic dysarthria.

Lee Silverman Voice Treatment

Lee Silverman Voice Treatment *(LSVT®LOUD)* is a highly structured intervention program that focuses on increasing vocal loudness by increasing phonatory effort, vocal fold adduction, and self-monitoring of vocal loudness (Baumgartner, Sapir, & Ramig, 2001). *LSVT®LOUD* requires intensive, high effort speech exercise combined with a simple, redundant, and salient treatment target to encourage loudness in daily living, across simple to more complex tasks (Fox, Ramig, Ciucci, Sapir, McFarland, & Farley, 2006). *LSVT®LOUD* targets inadequate muscle activation, which underlies hypokinesia and bradykinesia. Whereas *LSVT®LOUD* was originally developed and tested on people with Parkinson disease, the treatment has been shown to improve vocal loudness and speech intelligibility in children (Fox & Boliek, submitted).

Baumgartner et al. (2001) noted that *LSVT®LOUD* should improve voice quality and intensity, prosodic inflections, articulatory precision, resonance, and speech intelligibility. For example, they found significant pre- to post-treatment

improvement in the perceived hoarseness and breathiness of the voice of patients with Parkinson Disease. Kleinow et al. (2001) observed improvements in vocal fold adduction, maximum flow declination rate, subglottal pressure, lung volume excursion, maximum phonation duration, fundamental frequency range, and SPL following *LSVT®LOUD*. The success of this treatment may be attributed to its pervasive effect of training vocal loudness across the entire speech production mechanism and/or the intensive mode of delivery, which is consistent with principles of motor learning and activity-dependent neuroplasticity.

STUDY AIMS

To fully evaluate the potential usefulness of intensive voice treatment (*LSVT®LOUD*) with children who have neurogenic communication disorders (e.g., cerebral palsy), it is important to understand the physiological adjustments individuals use to alter loudness. Understanding biomechanical and aerodynamic adjustments made by healthy children to alter loudness may position clinicians to provide better treatment strategies to individuals with neurogenic communication disorders. Clinicians can then determine where in the speech mechanism adjustments are being made and if these are appropriate for achieving healthy voice and speech outcomes.

PURPOSE

The purpose of the current study was to provide a comprehensive understanding of the physiological adjustments made in the speech mechanism when typically developing children and children with spastic-type cerebral palsy

produced sustained maximum phonation and sentences differing in vocal loudness.

PRIMARY RESEARCH QUESTIONS

The primary research questions were:

1. How do respiratory behaviours, chest wall muscle activation patterns, laryngeal adjustments and mouth opening change in response to producing a normal or loud maximum phonation?
2. How do respiratory behaviours, chest wall muscle activation patterns, laryngeal adjustments and mouth opening change in response to producing soft, normal, or loud productions of a prescribed spoken sentence?
3. How do children with spastic-type cerebral palsy compare to matched typically developing control children on respiratory behaviours, chest wall muscle activation patterns, laryngeal adjustments and mouth opening change in response to producing soft, normal, or loud productions for maximum phonation and for prescribed spoken sentences?

RESEARCH DESIGN

This descriptive study used a one-way within-subjects experimental design, with eight dependent variables, replicated across two groups: (1) typical children (8-12 years) and (2) children with CP (8-12 years). There were two types of tasks: (1) sustained maximum phonation and (2) sentence repetition ("I sell a sapapple again."). The independent variable was the Loudness Condition which had two levels in the sustained maximum phonation task (Normal, 2X loud) and four levels (Soft, Normal, 2X loud, and 4X loud) in the sentence task. The

dependent variables were: (1) lung volume initiation (LVI, measured in cm^3), (2) lung volume termination (LVT, measured in cm^3), (3) lung volume excursion (LVE measured in %VC), (4) intercostal muscle average amplitude (percent maximum voluntary effort, measured in %MVE), (5) oblique muscle average amplitude (percent maximum voluntary effort, measured in %MVE, (6) laryngeal speed quotient (SQ, a ratio measure), (7) average fundamental frequency ($F_{0,}$ measured in Hz and semitones), and (8) maximum mouth area (MA, measured as a mean difference from normal in mm^2).

RESEARCH HYPOTHESES

Maximum Sustained Phonation

Typical Children

1. LVI will increase from Normal to 2X Loud productions.
2. LVT will decrease from Normal to 2X Loud productions.
3. LVE will increase from Normal to 2X Loud productions.
4. %MVE will increase in the intercostal muscles from Normal to 2X Loud productions.
5. %MVE will increase in the oblique muscles from Normal to 2X Loud productions.
6. There will be no change in SQ from Normal to 2X Loud productions.
7. F_0 will increase from Normal to 2X Loud productions.
8. MA will increase from Normal to 2X Loud productions.

Children with Cerebral Palsy

1. LVI will not change from Normal to 2X Loud productions.
2. LVT will not change from Normal to 2X Loud productions.
3. LVE will not change from Normal to 2X Loud productions.
4. %MVE will increase in the intercostal muscles from Normal to 2X Loud productions.
5. %MVE will increase in the oblique muscles from Normal to 2X Loud productions.
6. There will be an increase in SQ from Normal to 2X Loud productions.
7. F_0 will increase from Normal to 2X Loud productions.
8. MA will not change from Normal to 2X Loud productions.

Sentence Production

Typical Children

1. LVI will increase from Soft to 4X loud productions.
2. LVT will decrease from Soft to 4X loud productions.
3. LVE will increase from Soft to 4X loud productions.
4. %MVE will increase in the intercostal muscles from Soft to 4X loud productions.
5. %MVE will increase in the oblique muscles from Soft to 4X loud productions.
6. There will be no change in SQ from Soft to 4X loud productions.
7. F_0 will increase from Soft to 4X loud productions.
8. MA will increase from Soft to 4X loud productions.

Children with Cerebral Palsy

1. LVI will not change from Soft to 4X loud productions.
2. LVT will not change from Soft to 4X loud productions.
3. LVE will not change from Soft to 4X loud productions.
4. %MVE will increase in the intercostal muscles from Soft to 4X loud productions.
5. %MVE will increase in the oblique muscles from Soft to 4X loud productions.
6. There will be an increase in SQ from Soft to 4X loud productions.
7. F_0 will increase from Soft to 4X loud productions.
8. MA will not change from Soft to 4X loud productions.

RESEARCH METHODS

To gain a comprehensive picture of the manipulations required to change loudness, five measures were used: (a) respiratory kinematics, (b) chest wall surface electromyography (EMG), (c) Electroglottography (EGG), (d) acoustics (F_0), and (e) area of mouth opening. The addition of EMG and EGG data, in particular, expanded current understanding of changes in loudness. EMG provided information regarding muscle activity of two muscle groups involved in speech (intercostals and obliques) and the EGG provided information about laryngeal activity. In addition, subjects were unencumbered while speaking and phonating, such that natural speech and maximum phonation was possible.

Participants

A total of 8 children, ages 8 to 12 years, participated in the study. Four children had CP and were matched for age and sex to four typically developing children. All control participants were: (a) native speakers of English, (b) from non-smoking households, (c) free from upper or lower respiratory infection at the time of testing, and (d) had normal hearing and vision (or corrected to within normal limits), and (e) no speech or language difficulties as determined by parent and teacher reports. Table 2 shows the subject code, age and sex for the control participants matched to participants with CP.

Table 1. Subject code, age and sex for typically developing children matched to participants with cerebral palsy.

Subject	Age	Sex	Height (cm)	Weight (kg)	Subject	Age	Sex	Height (cm)	Weight (kg)
F1001CL.1	10	Female	149.50	36.00	F1001EL.1	10	Female	136.50	35.00
F1201CL.1	12	Female	153.00	40.00	F1201EL.1	12	Female	145.50	31.00
M0801CL.1	8	Male	126.00	26.80	M0801EL.1	8	Male	110.50	18.60
M1201CL.1	12	Male	147.50	48.00	M1201EL.1	12	Male	173.00	56.75

All participants with cerebral palsy were: (a) from English speaking homes (or bilingual where one of the languages spoken was English), (b) from non-smoking households, (c) free from respiratory infection at the time of testing, (d) had hearing and vision within normal limits or corrected to normal (determined by medical records), (e) diagnosed by a physician with spastic or mixed-ataxic-quadriplegia (described using the Gross Motor Function Scale (Palisano, Cameron, Rosenbaum, Walter, & Russel, 2006) and clinical observations), (f) ¾ children were impaired at all levels of the speech mechanism (respiratory, laryngeal, and oro-articulatory subsystems) and ¼ children were impaired at the oro-articulatory level of the speech mechanism, as assessed

according to Workinger (2005), (g) without severe velopharyngeal incompetence, and (h) able to repeat the target phrase.

Children with CP were diagnosed with spastic or mixed spastic-ataxic dysarthria (diagnosis was based on the latest evaluation by a registered speech language pathologist), and assigned a level of function (group) according to Yorkston, et al. (1999) and modified by Hodge (2009). Children with CP were also assessed for overall intelligibility, average maximum phonation duration (MP), average maximum fricative duration (MFD), average maximum monosyllable repetition rate (MRR-Mono), and average maximum tri-syllable repetition rate (MRR-Tri) using the TOCS+ protocol (Hodge & Daniels, 2004). Table 2 provides descriptive information for the participants with CP. All children with CP were given an oral mechanism examination which was adapted from Workinger (2005). Results from the oral mechanism examination are presented for each child with CP in Table 3.

Table 2. Description of children with cerebral palsy who participated in the study with respect to their level of function (Group) (lower values = greater communication impairment, See Appendix H for Group descriptions); level on the Gross Motor Function Scale (GMFS) (lower values = least gross motor function impairment, See Appendix G for the GMFS); type of cerebral palsy; type of dysarthria; overall sentence intelligibility score (in %); average maximum phonation duration (MP); average maximum fricative duration (MFD); average maximum monosyllable repetition rate (MRR-Mono); and average maximum tri-syllable repetition rate (MRR-Tri).

Subject	Level of Function	GMF Scale	Type of Cerebral Palsy	Type of Dysarthria	Intelligibility (%)	MP (s)	MFD (s)	MRR-Mono (syll/s)	MRR-Tri (syll/s)
F1001EL.1	Group 5	Level II	Spastic	NR	95.00[1]	15.35	NA	NA	NA
F1201EL.1	Group 3	Level III	Mixed/Ataxic	Mixed Ataxic	9.80	7.15	2.84	3.62	3.14
M0801EL.1	Group 4	Level V	Spastic	Spastic	32.60	8.35	5.26	2.94	2.10
M1201EL.1	Group 4	Level V	Spastic	Mixed Spastic	7.80	1.15	1.65	2.37	2.37

* NR = Non Remarkable

* NA = Not Available

[1] = Intelligibility measure was based on non-standarized sentence repetition

Table 3. Results obtained from the oral mechanism exam. Descriptions are for structural/functional deviations and abnormalities in the velopharyngeal, laryngeal, and respiratory systems, as well as subjective observations of muscle tone.

Subject	Structural/ Functional Deviations	Velopharynx	Larynx	Respiratory System
F1001EL.1	Asymmetry in lips.	Velum deviation left.	NR	NR
		Mild hypernasality.		
F1201EL.1	Labored tongue lateralization.	NR	Restricted pitch range.	Reduced breath support.
	Disordered tongue elevation.			
M0801EL.1	Not able to pucker/spread lips.	Velum deviation right.	Strained, strangled voice.	Reduced breath support.
	Disordered tongue elevation, lateralization, and resting posture.	Mild hypernasality.	Restricted pitch range.	Variable loudness range.
			Ab/adductor blocks. Delayed voice onset.	Variable subglottal pressure for speech.
M1201EL.1	Forward resting tongue posture.	Velum deviation right.	Restricted pitch range.	Reduced breath support.
		Mild hypernasality.		Variable loudness range.
				Variable subglottal pressure for speech.

* NR = Non Remarkable
* NA = Not Available

Tasks

Subjects were asked to repeat the sentence; *I sell a sapapple again*, at each of the 4 loudness levels (.5X Loud, Normal Loud, 2X Loud, and 4X Loud). Sentences were repeated immediately following the model given for loudness, provided by the researcher. This sentence token was chosen to facilitate comparison with the Dromey & Ramig studies (1998a, 1998b). A bar graph was

used to provide a visual display of the targeted loudness increases and decreases across the various loudness conditions. This procedure was commensurate with procedures used in past studies showing that, when internally calibrated changes in loudness were used, more natural approximation targets were achieved compared to targets that were externally driven via a SPL meter (Dromey & Ramig, 1998, a;b).

The control participants were able to say the phrase on one breath group. However, children with CP sometimes needed more than one breath to complete the sentence. Before recording, children were given the opportunity to practice each condition to ensure they understood the task, successfully modelled the examiner and received feedback about loudness adjustments produced. After the practice trials, five trials of each sentence for each condition were produced by each subject. The order of conditions was not randomized and were completed by each subject in the following order: (1) Normal loudness, (2) 2X normal loudness, (3) 4X normal loudness and (4) .5X normal loudness. Before the .5X normal loudness condition, participants produced five trials of the target sentence at normal loudness for the purpose of recalibration.

After the sentence tasks, subjects were asked to phonate as long as possible at two modeled loudness levels (Normal Loud and 2X Loud). Participants were given the opportunity to practice before recording each condition, again to ensure that they understood the task and could successfully model the examiner. Feedback was provided and children were encouraged to *work hard* to phonate for as long as possible. Three sustained maximum

phonations were performed at each loudness level in the following order: (1) Normal Loudness and (2) 2X normal loudness. Again, a bar graph was used to provide a visual display of the loudness increase participants were expected to make, in order to help children calibrate the targeted increase in vocal loudness.

General Procedures

Video recordings during the testing sessions were obtained through a camera on a tripod placed in front of the subject. All physiological signals, except for the 2D capture of mouth movement, were time-locked and acquired using an 8-channel FM digital recorders and simultaneously recorded on a 16-channel data acquisition system (Powerlab, AD Instruments, Inc., Colorado Springs, CO). All signals, except acoustic data, were sampled at 10kHz. Acoustic data was acquired at a sampling rate of 44 kHz and simultaneously recorded onto a digital audio tape (DAT) for acoustic analysis. A digital sound pressure level meter was placed 30 cm from each subject's mouth to obtain dB SPL readings at one second intervals during the experimental procedure. All subjects were fitted with a head-mounted microphone with a constant mouth-to-microphone distance and calibrated for dB SPL. An additional small condenser microphone was placed at approximately shoulder level for use during kinematic analysis. Figure 1 provides an example of the simultaneous acquisition of all physiological signals using Powerlab for three repetitions of the target utterance

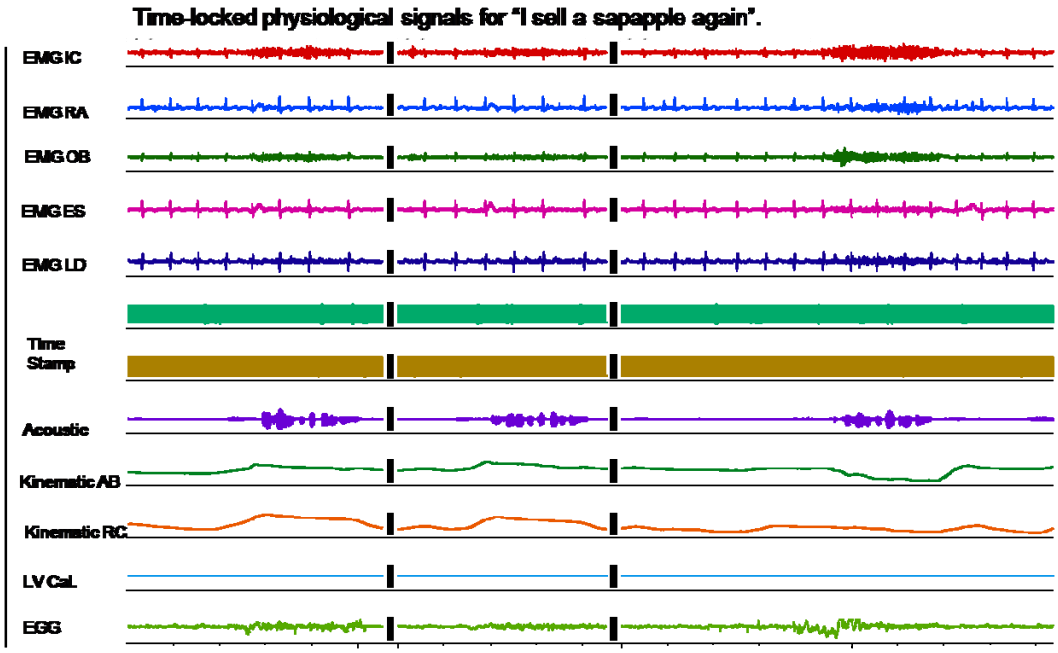

Figure 1. Time-locked physiological signals for the sentence, "I sell a sapapple again" produced by a typical child speaker. Of specific interest are channels 1 (EMG IC), 3 (EMG OB), 8 (acoustic), 9 (kinematic signal from the abdomen), 10 (kinematic signal from the rib cage), and 12 (EGG). These channels show muscle activation for the intercostal muscles and oblique muscles, as well as movement of the abdomen and rib cage, in addition to EGG signals, respectively. Signals acquired at 10kHz.

dB SPL

In order to be sure that participants were adjusting dB SPL for the loudness conditions, a sound pressure meter measured dB SPL at one second intervals. The dB SPL for each sentence and for each phonation was averaged across multiple trials (5 and 3, respectively), in order to determine the average dB SPL of each condition for each type of task.

Estimated Subglottal Pressure

To document each participant's ability to adjust subglottal pressure across loudness conditions, estimates of subglottal pressure were derived from pressure measurements taken at the airway opening. Oral pressure was measured using a differential pressure transducer calibrated against a u-tube manometer. A small

tube was placed just behind the child's lips and the nasal airway was occluded via nose clips. The child was asked to produce the syllable train /pi/ at a rate of approximately one syllable/second and on one breath group. Syllable trains were produced at the four levels of loudness (.5X Loud, Normal Loud, 2X Loud, and 4X Loud). The oral pressure was averaged across multiple trials within a loudness condition, in order to determine the average oral pressure (cmH_20) of each loudness condition. The average oral pressure was then used as an estimate of subglottal pressure produced during speaking tasks.

Respiratory Kinematics

Calibrated volumes displaced by the body surface are equal to those displaced by pulmonary structures (Hixon et al., 1973). The total change in lung volume is represented by the combined displacements of the rib cage wall and the abdominal wall (Hixon et al., 1973). Changes in lung volume result when volume exchange is permitted with the environment through the open larynx and upper airway. These can be achieved with either part of the chest wall or through any combination of relative displacements of the rib cage and abdomen (Hixon et al., 1973). Relative motion relationships during isovolume manoeuvres and relative motion during speech can be graphically displayed. From that graph, it is possible to estimate the individual volume contributions of the rib cage and abdomen to changes in lung volume (Hixon et al., 1973).

Equipment and Procedures

All subjects were seated upright in a chair. Variable inductance plethysomograph bands (Respitrace system, Ambulatory Monitoring Company,

Ardsley, NY) were placed around the chest wall. Each band encircled its respective chest wall part and sensed changes in size, expressed as an average cross-sectional area through the height of the transduction band (Stradling, Chadwick, Quirk, & Phillips, 1985). The band for the rib cage was positioned with its upper edge slightly below the axillae and its lower edge slightly below the nipples. The band for the abdomen was positioned with its upper edge slightly below the costal margin and its lower edge slightly above the iliac crests (Boliek et al., 1996). Water-soluble ink was used to mark the placement of the bands to aid in the detection of any shifts in their positions. After calibration, output signals from the transduction bands were used as estimates of the volume displacements of the rib cage and the abdomen, and their sum was used as an estimate of the volume displacement of the lung (Watson, 1979). At the beginning of the experiment, a standardized 100 cm^3 syringe was used to administer air to a pneumotachometer coupled to a differential pressure transducer and integrated to create a reference volume for the calibration of lung volume.

Subjects were asked to breathe through a face-mask connected to the pneumotachometer-pressure transducer, integrated for volume, while simultaneous kinematic signals also were collected. This procedure allowed for the conversion of motion sensed at the chest wall to volume (Boliek et al., 1996; 1997). The face-mask was only applied to the airway opening for the calibration procedure. The child was unencumbered for the remainder of the data collection session. Isovolume manoeuvres were used to calibrate the movements of the rib cage and abdomen (Hixon et al, 1973). The isovolume manoeuvre was

accomplished by having the child hold his or her breath (with nose clips applied) while pulling their stomach in and allowing it to relax at end expiratory level. This measured rib cage and abdomen movements at EEL and thus allowed calculation of their relative contributions to lung volume, which was calculated post hoc. To establish measures of tidal (rest breathing), children were asked to sit quietly while researchers appeared to be checking equipment. Attempts were made to obtain reliable vital capacities from all children. However, only one child (control) was able to reach the criteria of producing stable vital capacities with 5% of predicted values. Therefore, predicted vital capacities (ranging from 1703 – 3237 cm^3) were established for each child based on his or her height. These values were then used for converting absolute lung volume excursions to relative excursions in percent predicted vital capacity (%VC).

Measurements

The analysis protocol used was exactly like that used in previous studies (Boliek, Hixon, Watson, & Jones, 2009; Boliek, et al., 1996; 1997). Isovolumes were used to adjust the kinematic signals for rib cage and abdomen based on their coupling to the lung. This process allowed for the summation of rib cage and abdomen kinematic signals. The summed signal was converted to lung volume (in cm^3) based on the known volume collected at the airway opening and calibrated against a 100 cm^3 syringe (Boliek, et al., 1996; 1997; 2009). Measures of lung volume initiation (LVI) and lung volume termination (LVT) were obtained relative to end expiratory level (EEL). LVE was measured relative to percent vital

capacity (%VC), in order to account for variations in lung volume related to the height differences in the subject pool.

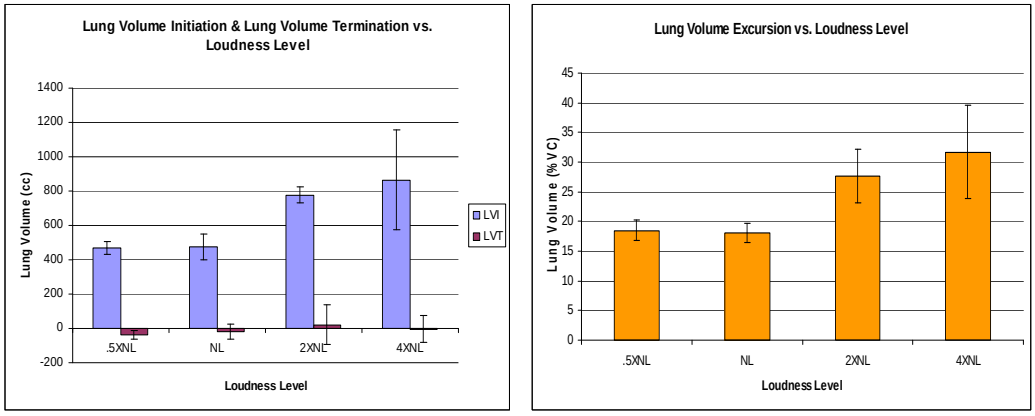

Figure 2. Measures of lung volume initiation (cc), lung volume termination (cc), and lung volume excursion (%VC), relative to EEL, in a typical child speaker, at various levels of loudness produced during sentence tasks.

Electromyography (EMG)

Surface EMG is a non-invasive technique used to detect the activation of muscles groups (Loeb & Gans, 1995). The relative amplitude of the EMG signal can indicate how strongly particular muscles are being used during a variety of tasks. Major changes in voltage and current occur when muscle fibres are activated by motor neurons. Electrical currents are generated by action potentials in the muscle fibres. Changes in the amplitude of the EMG signal specifies when muscle activity starts and stops relative to a predetermined baseline or tonic activity. The EMG signal can provide additional information regarding the number of active motor units and the frequency at which they fire (Loeb & Gans, 1995).

Equipment and Procedures

Electromyography (EMG) data were obtained using bipolar surface electrodes. Electrode pairs were used to record muscle activity from 2 muscle groups: intercostals (IC) and obliques (OB). Signals were collected from the right side of the body with standardized electrode placement lateral of mid-line (12-15 cm) and between the 6^{th} and 7^{th} intercostals. A ground electrode was placed over the clavicle. To standardize the EMG signal, maximum voluntary effort (MVE) tasks were performed for each muscle group. The MVE task for obliques required resistance by the opposite shoulder (left) to force applied by the examiner's hand. The MVE task for the intercostals was a breath-hold at the top of VC.

Measurements

Movement artifacts were removed from the data. EMG signals were filtered at 100 Hz and rectified. Amplitude was compared against the MVE produced for each participant for the purpose of amplitude normalization and the ability to compare within subjects across conditions. Percent MVE was calculated for each muscle by task for each participant. Figure 3 shows an example of the raw EMG and associated muscle amplitudes in % MVE acquired from a child. Whereas other muscle groups are depicted in this Figure, only IC and OB were of interest in the current study.

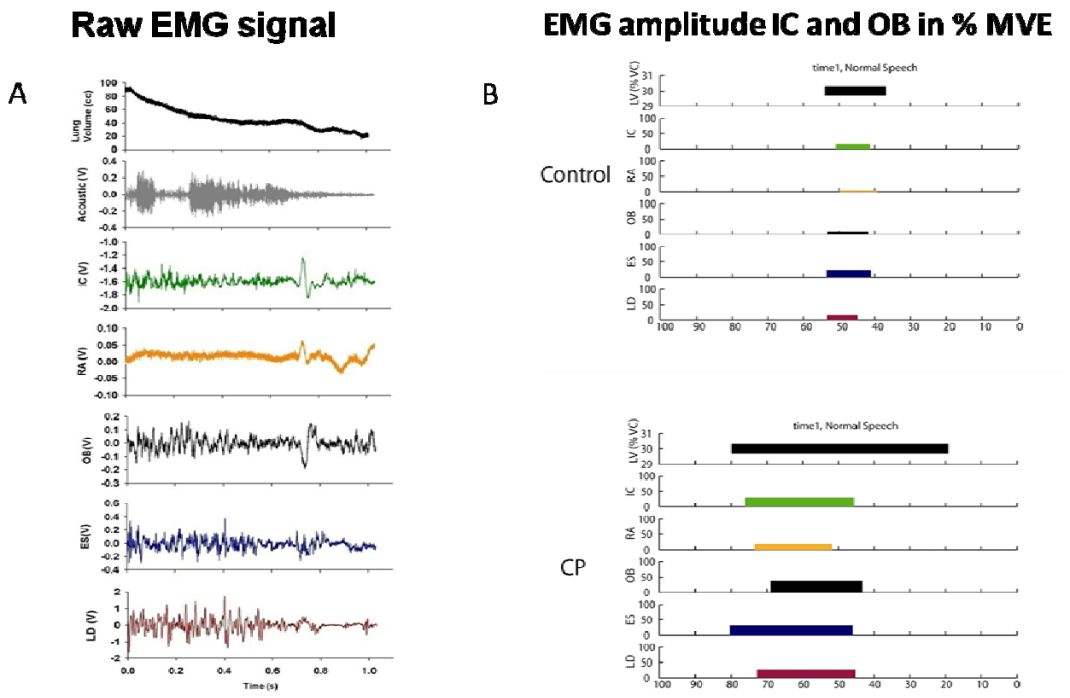

Figure 3. Vital capacity signal, acoustic and raw EMG signals for speech (A). Percent maximum voluntary effort and EMG onset (B), for various muscle groups for a typical child speaker and a child with cerebral palsy. The intercostal and oblique muscles are of specific interest (B).

Electroglottography (EGG)

EGG is a simple non-invasive method to estimate contact area of the vocal folds. Electrodes are placed on both sides of the neck over the left and right alae of the thyroid cartilage. Weak high frequency electric current flows between the two electrodes. The impedance to flow by the laryngeal muscles is measured. Vocal fold tissue is a good conductor of electricity, whereas the air between the vocal folds is a poor conductor of electricity. When the laryngeal airway is open, electrical impedance increases; when the vocal folds approximate each other, the electrical impedance decreases (Baken & Orlikoff, 2000). Electroglottograms represent the changes in vocal fold contact over time during voiced productions.

Equipment and Procedures

Electroglottograph surface electrodes were placed on the subjects' skin on either side of the thyroid cartilage and held in place with a Velcro band. A small, high frequency current was passed between the electrodes. The signal was low-pass filtered (approx. 700 to 1000 Hz) to remove slower articulatory frequencies and electrical drift in the direct current signal. The resulting wave form gave a representation of vocal fold dynamics including contact patterns. The signal provided a measure of speed quotient. Speed quotient (SQ) is defined as the duration of the open phase divided by the duration of the closed phase between 20-80% of the impedance rise and fall amplitude. Of the numerous measurements one can derive from the EGG wave form, SQ appears to be the most sensitive to changes in dB SPL (Sapienza, Stathopoulos, & Dromey, 1998).

Measurements

The SQ was derived from the time taken for impedance to rise from 20% to 80% of the peak-to-peak amplitude divided by the time for the signal to decrease from 80% to 20% of the downward phase of the same duty cycle. Figure 4 represents EGG signals and a simulated derived waveform showing the calculation of SQ. EGG waveforms were selected from the vowel tokens "I" and s "a" p produced for each sentence and from the sustained maximum phonations. The SQ was averaged across multiple trials for each condition (i.e. five trials of sentences and three trials of maximum phonations). The middle 60 ms of each sample (30 ms to each side of the midpoint) was analyzed provided the sample

could be filtered into a smooth signal without compromising the amplitude of the signal.

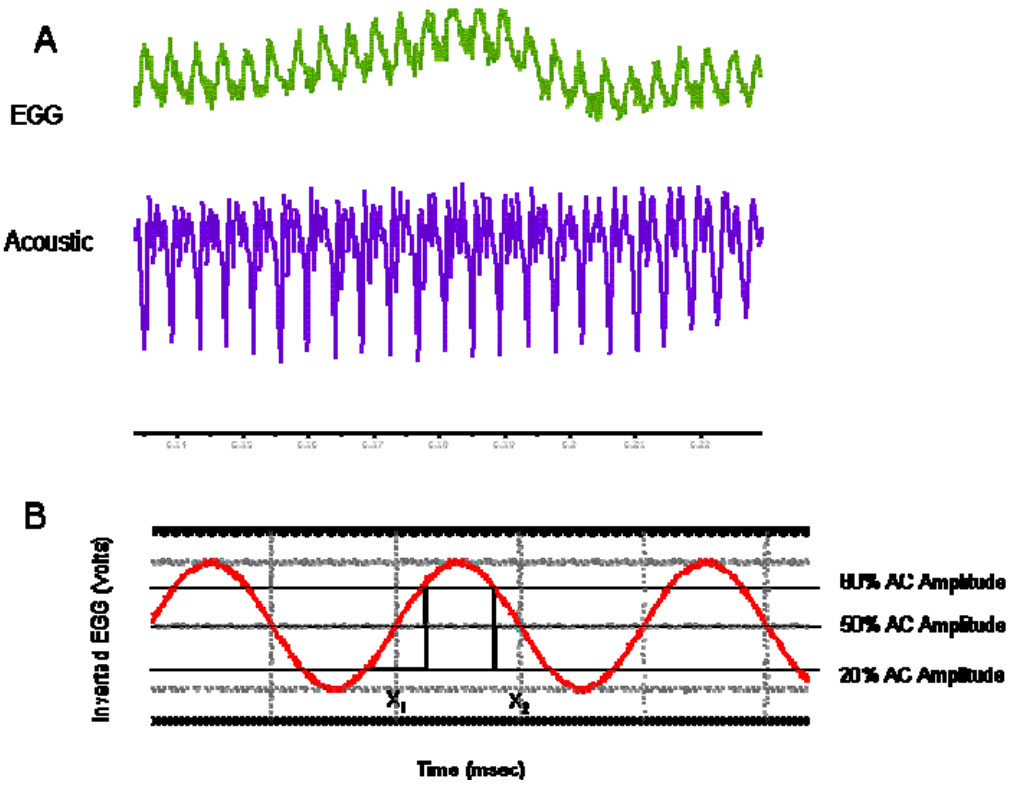

Figure 4. Acoustic signal and resulting EGG signal in typical child speaker, during production of the "ah" vowel (A). Signal is not rectified. Simulated signal and speech quotient measurement (X_1/X_2) (B).

Acoustic Measures of Mean Fundamental Frequency

The fundamental frequency (F_0) of vocal fold movement is related to the average rate of vocal fold vibration. It can be expressed in cycles per second (e.g., Hertz) or semitones (Baken & Orlikoff, 2000). Pitch is the auditory-perceptual correlate of F_0. Fundamental frequency is controlled by adjustments of the vocal folds. Primarily, it is influenced by vocal fold stiffness, the vibrating mass of the vocal folds, and the tension of the vocal fold covers (Hixon et al., 2008). Cricothyroid muscles tend to stretch the vocal folds and increase the tension

(force per unit length) along them. This thins the vocal folds and stiffens them, causing them to vibrate faster. Stiffness can also be increased by contraction of the thyroarytenoid muscles (Ludlow, 2005). The vibrating length, and thus mass, of the vocal folds can be changed through actions of the lateral cricoarytenoid muscles (Ludlow, 2005).

Equipment and Procedures

The microphone attached to the forehead was used to capture the audio signal and it was used for all acoustic analyses. An acoustic signal was recorded and stored on DAT medium, edited using *Praat* (Boersma & Weenick, 2010), and stored on a computer hard drive.

Measurements

Mean F_0 was measured using *Praat* software (Boersma & Weenick, 2010). The average F_0 (middle 60 ms (30 ms to each side of the midpoint)), for "I" and s "a" p in the sentence task was calculated for each condition. The average F_0 was also calculated for the maximum phonation tasks. Average F_0 was converted to semitones for normalization purposes (Heylen, Wuyts, Mertens, De Bodt, & Van de Heyning, 2002).

The following formula was used to convert Hz to semitones:

$ST = \log(f/fb)/0.0251$, where f is the frequency (Hz) to be converted and f_b (Hz) is the frequency that corresponds with the semitone just below the average F_0 (Hz) produced at normal loudness. The number 0.0251 was the constant used to convert Hz to semitones because an octave containing 12 semitones corresponds to a doubling of the frequency, or $\log(2)/12=0.0251$ (Heylen et al., 2002). The

Normal Loud condition in both maximum sustained phonation tasks and sentences tasks equalled 1 semitone. Semitone measures for derived for the other conditions were calculated using the normal value (1) obtained from the Normal Loud condition, as the reference.

Motion Tracker 2D

In previous literature, Dromey & Ramig (1998a) used a head-mounted, strain gauge cantilever system to track the movements of the upper and lower lips during speech. The cantilever beams were guided through small beads secured with adhesive tape to the speaker's lips. They found that lip displacement increased during loud speech. To leave the participants unencumbered, this study employed a Motion Tracker 2D protocol (Dr. D. Webber, University of Pittsburgh, Pittsburgh, PA), involving a MATLAB (2010a, The MathWorks, Natick, MA) application capable of tracking up to 18 markers in a Cartesian coordinate system. The motion tracker system was used to analyze each video clip (video format of 60 fps (DV): resolution, 720 x 480). Motion Tracker 2D (Dr. D. Webber, University of Pittsburgh, Pittsburgh, PA) is more feasible for use with children, as it requires no adhesives, can take accurate measurements during sentence production, controls for movement artifact, and allows mouth area to be calculated.

Equipment and Procedures

Circular markers, approximately 0.5 cm in diameter, were drawn at 8 reproducible anatomical landmarks onto the face, using a water soluble eyeliner pencil. The following points were drawn: (1) on each side of the upper lip

(between the philtrum and lip corner along the upper lip border), (2) on each side of the lower lip (between the edge of the chin and the corner of the lip), (3) on the lip corners, and (4) on the forehead. The central nose was marked as a static point. This point served as a reference point for measuring the excursion of the other facial markers. The distance between the nose and forehead was recorded for calibration purposes. The subjects were positioned in front of a white screen to maximize the contrast between the markers and skin. In addition, a spotlight was directed onto the face, while subjects performed speech tasks. The digital camera framed the face. Figure 5 shows an example of the 2-D marking system.

Measurements

Maximum area of mouth opening for sentences and maximum phonations was calculated for each condition. Area was defined as the two-dimensional space (in mm^2) that was derived from the maximum displacement of lip and lip corner markers. Area was calculated by using a function in MATLAB (2010a, The MathWorks, Natick, MA) called convhull. Convhull measures the indices of the points of a 2D convex hull and the area of the convex hull.

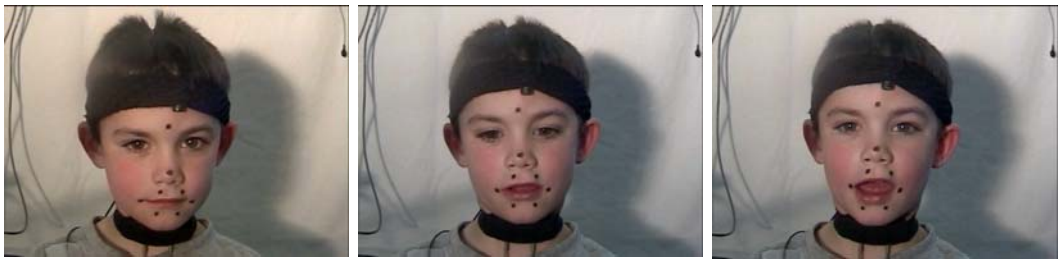

Figure 5. Mouth displacement: small to large opening (left to right) in typical child. Tracking dots were drawn on the child's face to measure area of mouth opening. Nose and forehead dots served as reference points. Used with permission.

DATA ANALYSIS

Respiratory Kinematics

The kinematic signals were acquired, post hoc, using Lab View (National Instruments, Austin, TX) and customized software. Rib cage, abdomen, and lung volume were displayed in a y-t mode and volume displacements were read in relation to EEL. EEL was selected as zero for rib cage, abdomen, and lung volume displacements. Volumes larger or smaller than EEL were expressed as positive or negative values, respectively. Lung volume initiations and terminations were measured for each breath group for all conditions (Boliek et al., 1996; 1997; 2009).

Electromyography

Measurements of relative amplitude (%MVE) were acquired for IC and OB muscles for each task, trial and condition. Average amplitude was calculated for each participant based on the number of useable trials within a condition.

Electroglottography

EGG waveforms were filtered and rectified prior to measurement. A customized MATLAB (2010a, The MathWorks, Natick, MA) program (Dr. L. S. Gan, University of Alberta, Edmonton, AB) was used to calculate SQ for “I” and s “a” p and for the maximum phonation.

Acoustic Measure of Fundamental Frequency

Acoustic signals were analyzed using *Praat* (Boersma & Weenick, 2010). Vowel nuclei “I” and s “a” p were selected for analysis in the sentence tasks. The maximum phonation tasks were also analyzed using *Praat*. A customized *Praat*

script was used to analyze the tokens (Dr. B. Tucker, University of Alberta, Edmonton, AB). The beginning and end of the periodic signal for each vowel served as the start-end point for editing each token. Average F_0 was calculated for "I" and s "a" p in the sentence task and for "ah" in the maximum phonation task.

Motion Tracker 2D

Motion Tracker 2D (Dr. D. Webber, University of Pittsburgh, Pittsburgh, PA) calculates the position of each marker in relation to the static point in pixels. The program uses the distance between the forehead and nose points as a reference to determine the number of pixels in one millimeter. This distance is used to convert pixels into distance (in mm). All coordinates were saved in coded files. Customized MATLAB (2010a, The MathWorks, Natick, MA; Dr. D. Webber, University of Pittsburgh, Pittsburgh, PA; Fraser, R., & Chow, D., BSc) software was used to determine the maximum area of mouth opening for each vowel token within the sentence and across all conditions. Difference scores (e.g., area derived from normal loudness condition – area derived from experimental conditions) were calculated for the purpose of normalizing area across subjects. Mean difference scores were used in the statistical treatment of the data.

STATISTICAL ANALYSIS

Individual means and standard deviations were calculated for all physiological variables for each task, condition, and subject. Group data, for each variable, were displayed in box and whisker plots, where the lower fence indicated the 5^{th} percentile and the upper fence indicated the 95^{th} percentile. Averaged individual data along with standard deviations were displayed for each

variable. Visual trend analyses of individual and group data were used to describe the results. Exploratory between-groups statistical comparisons of typical children and children with CP were carried out using a Multiple Analysis of Variance (MANOVA). In addition, exploratory one-way, within subjects, Analysis of Variance (ANOVA) statistics were conducted for each dependent variable by each group, across 4 levels of loudness.

RESULTS

The results section will be presented as follows: (1) reliability analysis, (2) data on the average dBSPL produced for sustained maximum phonation tasks and sentence tasks, (3) data on the average estimated subglottal pressure achieved for syllable trains of /pi/ produced at each loudness level, (4) descriptive data (mean, median, standard deviation) for each dependent variable for both sustained maximum phonation tasks and sentence tasks, and (5) visual trend analyses for each dependent variable for both sustained maximum phonation tasks and sentence tasks.

Reliability Analysis

Ten percent of the total data collected from the following measures was randomly selected, using a random numbers generator, for re-analysis: lung volume events (i.e. LVI, LVT, and LVE), speed quotient, fundamental frequency, mouth opening, and estimated subglottal pressure. Sound pressure level (dB SPL) was not selected for re-analysis because it was collected online as the participant performed the tasks. Muscle activity was not selected for re-analysis because it was analyzed using a highly automatized customized MATLAB program (2010a,

The MathWorks, Natick, MA; Dr. L.S Gan, University of Alberta, Edmonton, AB). The researcher re-analyzed ten percent of the data in order to calculate intra-rater reliability and an independent rater analyzed the same ten percent of the data in order to calculate inter-rater reliability. Intra- and inter-rater reliability results are presented in Table 4.

Table 4. Intra-rater reliability and inter- rater reliability, reported as Pearson correlations for each variable. All correlations were significant at the 0.01 level (two-tailed).

Variable	Intra-rater Reliability	Inter-rater Reliability
Lung Volume Events	0.958	0.909
Speed Quotient	0.640	0.474
Fundamental Frequency	0.932	0.879
Mouth Opening Area	1.000	0.999
Estimated Subglottal Pressure (Oral Pressure)	0.999	0.999

Confirmation of Loudness Production

dBSPL for Maximum Sustained Phonation

Table 5 and Figure 6 present the results for dB SPL on maximum phonation tasks produced at normal and twice normal loudness. As can be seen by examining the average values shown in Table 5, all participants increased their dB SPL when asked to produce maximum phonations at twice the normal level.

Table 5. Mean, standard deviation, and median values for sound pressure (dB SPL derived from the sound level meter with a 30cm mouth-to-mic distance) for two loudness conditions (NL = Normal Loudness and 2X NL = 2X Normal Loudness). Data from typically developing children (control subjects) and children with CP (experimental subjects) are depicted in the two left and two right columns; respectively.

		Control Subjects		Experimental Subjects	
		Conditions		Conditions	
Variables:		NL	2X NL	NL	2X NL
dB SPL	Mean	62.16	74.10	69.25	78.20
	(sd)	(3.63)	(4.83)	(6.58)	(4.46)
	Median	61.65	71.835	67.155	76.765

*NL = Normal Loudness

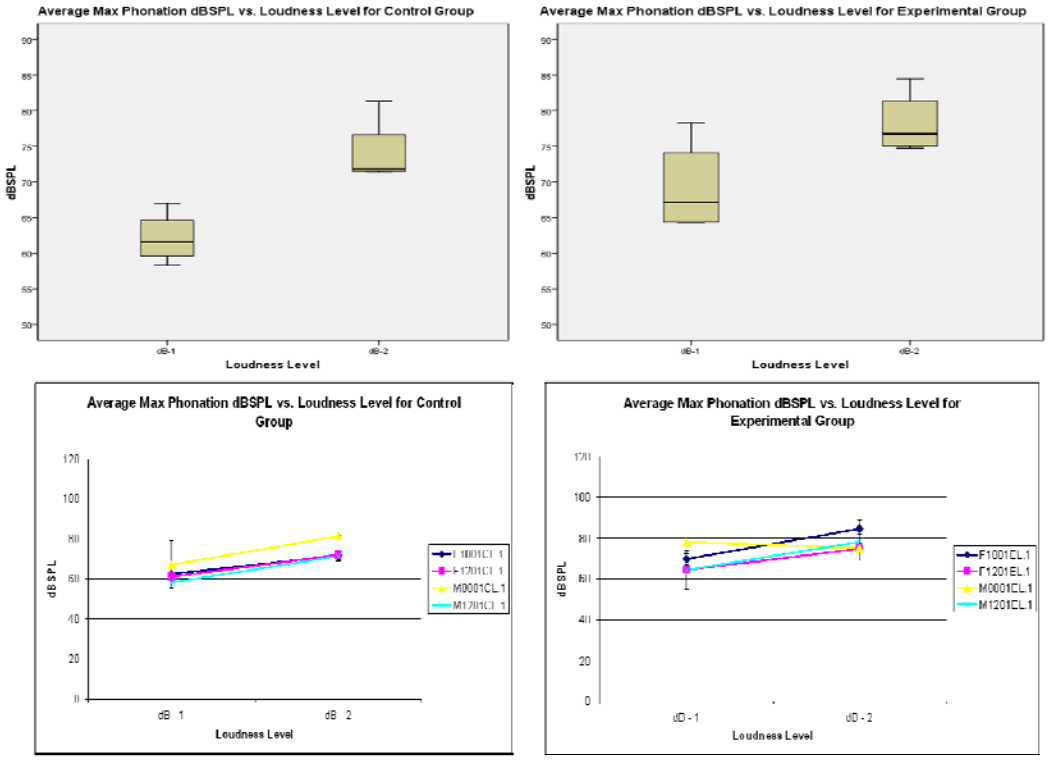

Figure 6. Group and individual data for sound pressure level, measured using a sound level meter with a 30cm mouth-to-mic distance, for maximum phonation tasks. Four panels represent group and individual data. Data from the control group (typically developing children) are shown in the two panels on the left and data for the experimental group (children with CP) are shown in the two panels on the right. In all four panels, sound pressure is depicted on the *y*-axis in dB SPL. In all four panels, loudness condition (dB1 = Normal Loudness and dB2 = 2X Normal Loudness) is depicted on the *x*-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation dB SPL values for individual participants. The matched participants are displayed in the same color.

The box plots presented in Figure 6, show that there are no outliers in either group, but the overall range of dB SPL is slightly larger for children with CP for phonations produced at normal loudness. The box plots also show that in general, children with CP produced sustained maximum phonation at slightly higher dB SPL levels for both conditions compared to their control counterparts. As can be seen in the lower right panel of Figure 6, one participant with CP (M0801EL.1) did not change dB SPL when asked to phonate at twice normal loudness.

dBSPL for Sentence Tasks

Study participants completed the sentence task in the following order: Normal Loudness, 2X Normal Loudness, 4X Normal Loudness, Normal Loudness, and .5X Normal Loudness. There were two Normal Loudness conditions for the sentence tasks. The second set of Normal Loudness sentences were used for re-calibration purposes. In other words, the participants recalibrated to Normal Loudness after the 4X Normal Loudness task before completing the .5X Normal Loudness task. Paired sample *t*-tests were used to determine if the first set of Normal Loudness values were significantly different from the second set of Normal Loudness values for each variable for all eight participants. The results of the *t*-tests revealed that the first set of Normal Loudness values were not significantly different from the second set of Normal Loudness values on any dependent measure. In addition, dB SPL values derived from the first Normal Loudness condition were not significantly different from dB SPL values derived from the second Normal Loudness condition.

The results of the t-tests are as follows (1) lung volume initiation, $t(7) = -1.167$, $p = 0.282$; (2) lung volume termination, $t(7) = 1.174$, $p = 0.279$; (3) lung volume excursion, $t(7) = -1.741$, $p = 0.125$; (4) intercostals muscle activity, $t(7) = 0.548$, $p = 0.601$; (5) oblique muscle activity, $t(7) = -1.089$, $p = 0.312$, (6) speed quotient for "I"; $t(7) = 0.111$, $p = 0.915$; (7) speed quotient for s "a"p, $t(7) = -0.480$, $p = 0.646$; (8) fundamental frequency for "I", $t(7) = -1.895$, $p = 0.100$; (9) fundamental frequency for s "a"p, $t(7) = -0.245$, $p = 0.841$; (10) dB SPL, $t(7) = -0.012$, $p = 0.345$; and (11) area of mouth opening, $t(7) = -1.080$, $p = 0.316$. The

results of this study are therefore based on the comparisons to the first set of Normal Loudness values.

Table 6 and Figure 7 present the results for dB SPL on sentence tasks produced at .5X Normal Loudness, Normal Loudness, 2X Normal Loudness, and 4X Normal Loudness. As can be seen by examining the average values shown in Table 6, all participants increased their dB SPL when asked to produce sentence tasks at increasing levels of loudness.

Table 6. Mean, standard deviation, and median values for sound pressure (dB SPL) derived from the sound level meter with a 30cm mouth-to-mic distance for four loudness conditions (.5X NL = Half Normal Loudness; NL = Normal Loudness; 2X NL = 2X Normal Loudness; and, 4X NL = 4X Normal Loudness). Data from typically developing children (control subjects) and children with CP (experimental subjects) are depicted in the two left and two right columns; respectively.

		Control Group				Experimental Group			
		Conditions				Conditions			
Variables:		**.5X NL**	**NL**	**2X NL**	**4X NL**	**.5X NL**	**NL**	**2X NL**	**4X NL**
dB SPL	Mean	56.29	60.64	74.36	81.73	56.78	62.35	70.26	79.04
	(sd)	(2.29)	(2.20)	(5.18)	(5.84)	(2.46)	(6.18)	(7.57)	(12.05)
	Median	56.53	60.425	73.8	81.2	55.85	64.68	69.84	82.89

NL = Normal Loudness

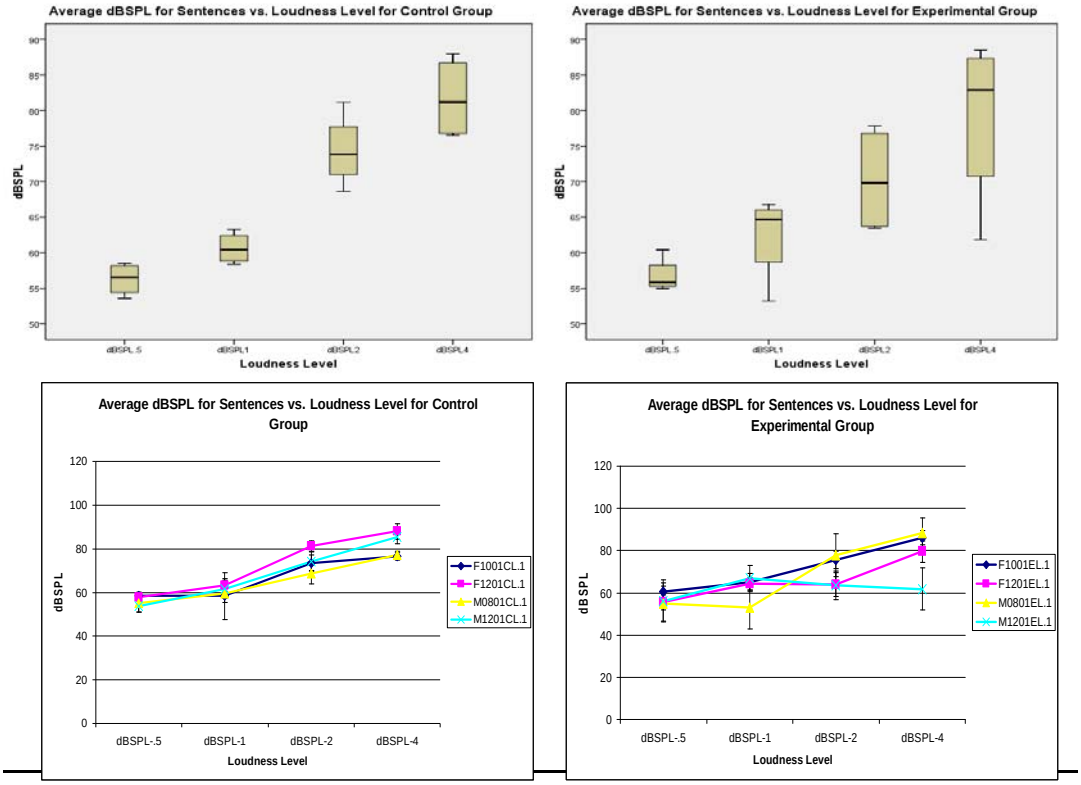

Figure 7. Group and individual data for sound pressure level, measured using a sound level meter with a 30cm mouth-to-mic distance, for sentence tasks. Four panels represent group and individual data. Data from the control group (typically developing children) are shown in the two panels on the left and data for the experimental group (children with CP) are shown in the two panels on the right. In all four panels, sound pressure is depicted on the *y*-axis in dB SPL. In all four panels, loudness condition (dB.5 = Half Normal Loudness; dB1 = Normal Loudness; dB2 = 2X Normal Loudness; and, dB4 = 4X Normal Loudness) is depicted on the *x*-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation dB SPL values for individual participants. The matched participants are displayed in the same color.

The box plots presented in Figure 7, show that there are no outliers in either group, but the overall range of dB SPL is slightly larger for children with CP for sentences produced at normal loudness, twice normal loudness, and four times normal loudness. The box plots also show that in general, both groups produced sentence tasks at similar dB SPL levels for all conditions. As can be seen in the lower right panel of Figure 7, one participant with CP (M0801EL.1) did not change dB SPL from half normal loudness to normal loudness. Another participant with CP (F1201EL.1) did not change dB SPL from normal loudness to

twice normal loudness, and finally one other participant with CP (M1201EL.1) exhibited only marginal changes in dB SPL across the loudness conditions.

Estimated Subglottal Pressure

Study participants completed the syllable train task for /pi/ in the following order: Normal Loudness, 2X Normal Loudness, 4X Normal Loudness, Normal Loudness, and .5X Normal Loudness. Table 7 and Figure 8 present the results for oral pressure, from which subglottal pressure was estimated, on syllable train tasks produced. As can be seen by examining the average values shown in Table 7, all participants increased their oral pressure when asked to produce syllable trains at increasing levels of loudness.

Table 7. Mean, standard deviation, and median values for oral pressure (oral pressure measured using a differential pressure transducer calibrated against a u-tube manometer) for four loudness conditions (.5X NL = Half Normal Loudness; NL = Normal Loudness; 2X NL = 2X Normal Loudness; and, 4X NL = 4X Normal Loudness). Data from typically developing children (control subjects) and children with CP (experimental subjects) are depicted in the two left and two right columns; respectively.

		Control Group				Experimental Group			
		Conditions				Conditions			
Variables:		**.5X NL**	**NL**	**2X NL**	**4X NL**	**.5X NL**	**NL**	**2X NL**	**4X NL**
Oral Pressure	Mean	7.12	9.73	11.24	12.38	7.63	8.82	10.43	11.26
(cmH_20)	(sd)	(2.00)	(1.71)	(2.65)	(3.53)	(0.50)	(1.76)	(0.43)	(0.84)
	Median	6.765	9.805	10.24	11.05	7.38	9.35	10.49	11.68

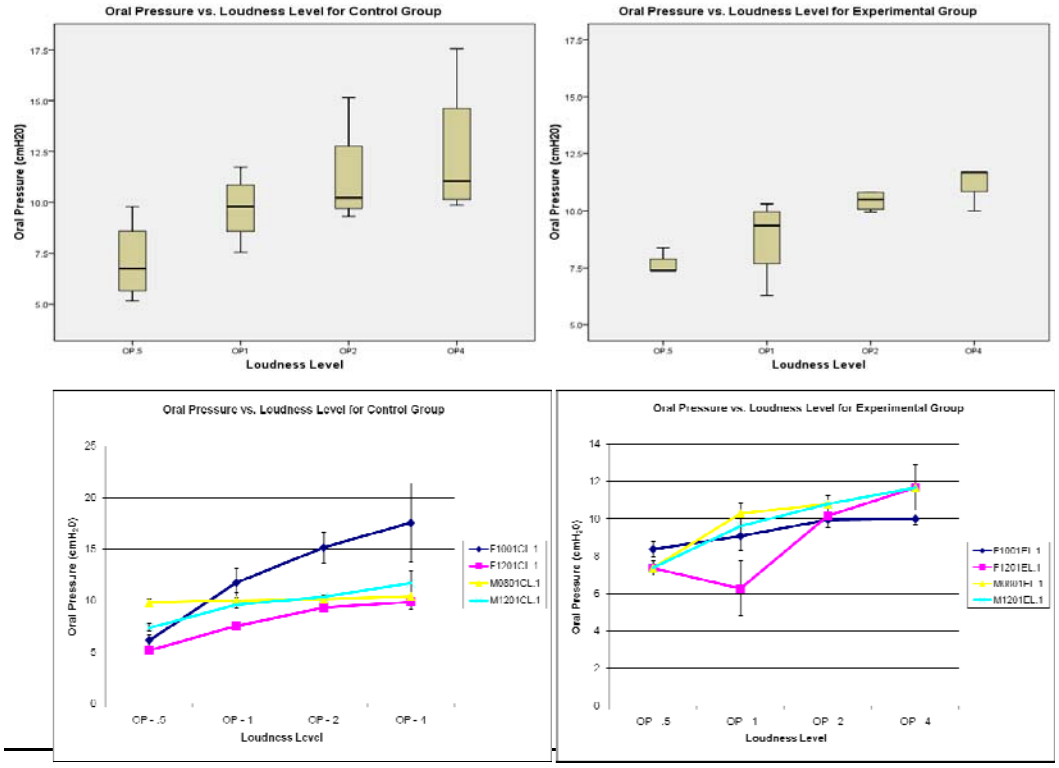

Figure 8. Group and individual data for oral pressure (cmH_20), measured using a differential pressure transducer calibrated against a u-tube manometer, for syllable train tasks. Four panels represent group and individual data. Data from the control group (typically developing children) are shown in the two panels on the left and data for the experimental group (children with CP) are shown in the two panels on the right. In all four panels, oral pressure is depicted on the *y*-axis in cmH_20. In all four panels, loudness condition (OP.5 = Half Normal Loudness; OP1 = Normal Loudness; OP2 = 2X Normal Loudness; and, OP4 = 4X Normal Loudness) is depicted on the *x*-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation oral pressure values for individual participants. The matched participants are displayed in the same color.

The box plots presented in Figure 8, show that there are no outliers in either group, but the overall range of oral pressure is larger for typically developing children for sentences produced at half normal loudness, twice normal loudness, and four times normal loudness. The box plots also show that in general, typically developing children produced syllable trains at slightly higher oral pressure levels for all conditions, except half normal loudness, compared to their counterparts with CP. As can be seen in the lower left panel of Figure 8, one

control participant (M0801CL.1) did not change oral pressure from half normal loudness to four times normal loudness. As can be seen in the lower right panel of Figure 8, a participant with CP (F1001EL.1) did not change oral pressure from twice normal loudness to four times normal loudness, and another participant with CP (F1201EL.1) decreased oral pressure from half normal loudness to normal loudness.

Descriptive Statistics for Dependent Variables

Maximum Sustained Phonation

Table 8 presents the descriptive statistical results for the dependent variables on maximum phonation tasks produced at normal and twice normal loudness.

Table 8. Mean, standard deviation, and median values for the dependent variables for two loudness conditions (NL = Normal Loudness and 2X NL = 2X Normal Loudness). Data from typically developing children (control subjects) and children with CP (experimental subjects) are depicted in the two left and two right columns; respectively.

		Control Subjects		Experimental Subjects	
		Conditions		Conditions	
Variables:		**NL**	**2X NL**	**NL**	**2X NL**
Lung Volume Initiation (cm^3)	Mean	873.53	962.26	393.54	436.30
	(sd)	(926.10)	(1012.82)	(409.34)	(376.47)
	Median	506.99	601.56	202.13	316.99
Lung Volume Termination (cm^3)	Mean	-333.74	-339.19	-61.55	-98.43
	(sd)	(266.84)	(217.94)	(116.79)	(126.61)
	Median	-204.98	-274.50	-9.46	-79.06
Lung Volume Excursion (% VC)	Mean	45.93	49.41	19.20	21.91
	(sd)	(42.03)	(43.27)	(16.00)	(12.01)
	Median	29.44	33.02	13.62	16.31
Intercostal Muscle Activity (% MVE)	Mean	46.74	53.58	48.37	49.40
	(sd)	(3.98)	(6.85)	(26.99)	(9.95)
	Median	47.83	55.68	38.06	46.99
Obliques Muscle Activity (% MVE)	Mean	37.61	43.82	27.16	40.37
	(sd)	(12.76)	(12.47)	(17.86)	(11.70)
	Median	36.23	45.61	30.50	39.92
Speed Quotient	Mean	0.59	1.36	0.79	0.66
	(sd)	(0.37)	(1.41)	(0.76)	(0.31)
	Median	0.51	0.78	0.47	0.57
Fundamental Frequency (Hz)	Mean	221.61	269.89	280.00	334.46
	(sd)	(22.55)	(43.29)	(37.90)	(51.31)
	Median	227.79	277.65	278.71	341.55
Fundamental Frequency	Mean	1.00	3.46	1.00	4.05
(semitones)	(sd)	(0.00)	(1.51)	(0.00)	(1.73)
	Median	1	2.935	1	4.11
Area of Mouth Opening	Mean	0.00	69.69	0.00	-342.62
(Mean Difference in mm^2)	(sd)	(0.00)	(307.13)	(0.00)	(577.32)
	Median	0.00	73.70	0.00	-233.40

*NL = Normal Loudness

Sentences

Table 9 presents the descriptive statistical results for the dependent variables on sentence tasks produced at half normal loudness, normal loudness, twice normal loudness, and four times normal loudness.

Table 9. Mean, standard deviation, and median values for the dependent variables for four loudness conditions (.5X NL = Half Normal Loudness; NL = Normal Loudness; 2X NL = 2X Normal Loudness; and, 4X NL = 4X Normal Loudness). Data from typically developing children (control subjects) and children with CP (experimental subjects) are depicted in the two left and two right columns; respectively.

		Control Group				Experimental Group			
		Conditions				Conditions			
Variables:		.5X NL	NL	2X NL	4X NL	.5X NL	NL	2X NL	4X NL
Lung	Mean	240.90	278.44	388.54	450.50	147.58	179.39	253.66	140.02
Volume	(sd)	(161.03)	(144.38)	(265.64)	(294.51)	(79.40)	(150.79)	(224.03)	(209.27)
Initiation (cm^3)	Median	193.97	247.41	290.40	367.62	130.93	130.63	188.55	83.70
Lung	Mean	-43.02	-14.14	-44.52	-28.69	-75.42	-20.13	-27.52	-95.11
Volume	(sd)	(37.33)	(27.49)	(71.31)	(30.78)	(97.33)	(54.92)	(76.79)	(118.89)
Termination (cm^3)	Median	-52.88	-18.21	-29.72	-20.59	-45.52	-27.84	-13.64	-105.92
Lung	Mean	11.05	13.67	16.94	18.69	9.01	8.27	11.90	10.00
Volume	(sd)	(5.52)	(7.49)	(8.38)	(9.98)	(4.46)	(4.90)	(7.30)	(4.59)
Excursion (%VC)	Median	10.12	14.51	16.40	17.67	8.54	7.45	10.71	8.71
Intercostal	Mean	13.26	17.39	16.21	26.99	32.76	43.78	41.49	58.73
Muscle Activity	(sd)	(6.60)	(7.82)	(9.25)	(13.97)	(13.53)	(21.02)	(7.10)	(13.34)
(% MVE)	Median	15.57	13.85	15.81	30.87	34.25	35.06	44.20	54.72
Obliques	Mean	20.76	11.56	15.49	24.01	13.71	17.92	20.87	34.15
Muscle Activity	(sd)	(3.12)	(14.46)	(5.54)	(13.31)	(8.41)	(10.32)	(16.42)	(28.07)
(% MVE)	Median	20.34	12.33	15.43	27.02	13.06	18.02	19.36	33.16
Speed	Mean	0.77	0.62	1.19	0.92	0.71	0.84	0.55	0.72
Quotient	(sd)	(0.18)	(0.19)	(0.62)	(0.48)	(0.31)	(0.30)	(0.19)	(0.30)
for "I"	Median	0.78	0.62	1.28	0.89	0.65	0.90	0.62	0.69
Speed	Mean	0.72	0.54	0.88	1.11	0.67	0.73	0.55	1.10
Quotient	(sd)	(0.38)	(0.39)	(0.45)	(0.57)	(0.22)	(0.37)	(0.18)	(0.61)
for s"a"p	Median	0.63	0.41	0.88	1.24	0.66	0.63	0.52	1.09
Fundamental	Mean	219.53	232.35	261.11	335.31	242.75	274.34	305.53	338.98
Frequency	(sd)	(22.62)	(12.95)	(42.77)	(46.99)	(30.20)	(18.12)	(40.77)	(38.88)
for "I" (Hz)	Median	223.76	235.02	277.46	350.81	235.00	272.95	289.71	340.09
Fundamental	Mean	212.34	206.09	243.46	300.08	256.36	281.04	276.29	354.99
Frequency	(sd)	(18.99)	(19.68)	(27.84)	(49.39)	(50.47)	(43.62)	(61.86)	(71.89)
for s"a"p (Hz)	Median	207.79	201.92	243.37	307.86	264.71	270.04	262.14	345.22
Semitones	Mean	0.02	1.00	2.85	5.35	0.13	1.00	2.18	6.90
for "I"	(sd)	(1.60)	(0.00)	(1.51)	(3.08)	(1.48)	(0.00)	(2.21)	(2.78)
	Median	-0.04	1.00	2.57	4.79	0.06	1.00	2.04	7.62
Semitones	Mean	0.87	1.00	3.88	7.19	1.18	1.00	2.83	7.54
for s"a"p	(sd)	(2.84)	(0.00)	(3.32)	(3.23)	(3.01)	(0.00)	(3.53)	(2.93)
	Median	1.62	1.00	4.14	6.29	2.24	1.00	2.03	6.99
Area of	Mean	88.96	0.00	-104.80	-276.74	176.31	0.00	59.82	80.76
Mouth Opening	(sd)	(75.62)	(0.00)	(180.87)	(88.65)	(612.70)	(0.00)	(331.77)	(723.89)
(MD, mm^2)	Median	86.00	0.00	-127.21	-258.43	136.91	0.00	76.05	72.84

*NL = Normal Loudness;
*MD= Mean Difference

Describing Results for Dependent Variables

The hypotheses for each dependent variable will be restated in the context of the current findings. The values for each dependent variable have been displayed as group data in the form of box and whisker plots and as individual means and standard deviations. For each variable, data will first be presented for the sustained maximum performance tasks followed by data from the sentence repetition tasks. Results will be presented based on the visual inspection of the group data (e.g., means, standard deviations and box plots) and also in terms of comparing the means and standard deviations from matched individuals (e.g., CP and matched control). Appendices I through N present means and standard deviations derived from each individual by task and condition. Exploratory statistical analysis will be presented at the end of the results section in an attempt to corroborate visual trend analyses.

Lung Volume Initiation – Maximum Sustained Phonation

Hypotheses:

1. a) In typical children, LVI will increase from Normal to 2X loud maximum phonation productions.

   b) In children with cerebral palsy, LVI will not change from Normal to 2X loud maximum phonation productions.

As can be seen in the top left panel of Figure 9, children in the control group initiated maximum phonation tasks at normal and two times normal loudness, across a wide range of lung volumes. The box plot indicates a slightly higher median value and slightly larger range for lung volume initiations when children were instructed to produce maximum phonations at twice normal

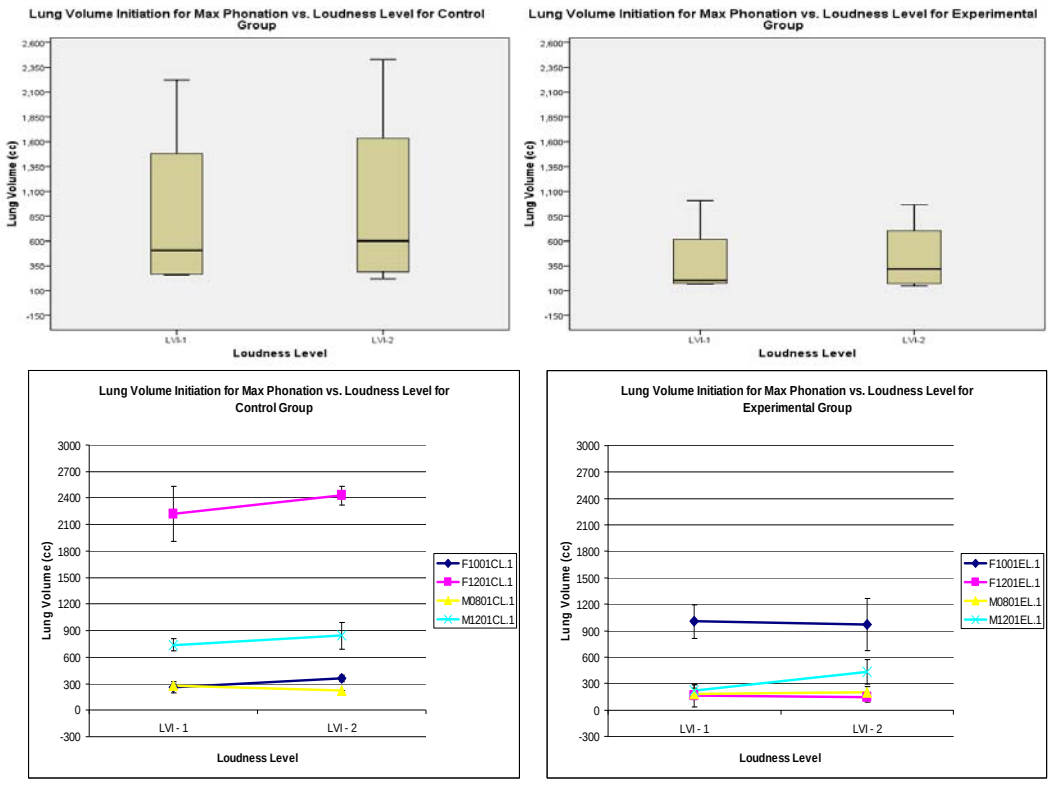

Figure 9. Group and individual data for lung volume initiation (in cm^3) relative to end-expiratory level (0 cm^3), for maximum phonation tasks. Four panels represent group and individual data. Data from the control group (typically developing children) are shown in the two panels on the left and data for the experimental group (children with CP) are shown in the two panels on the right. In all four panels, lung volume is depicted on the *y*-axis in cm^3. Positive values represent lung volumes initiated above end expiratory level (0 cm^3). In all four panels, loudness condition (LVI1 = Normal Loudness and LVI2 = 2X Normal Loudness) is depicted on the *x*-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation lung volume initiation values for individual participants. The matched participants are displayed in the same color.

loudness. Control group averages for lung volume initiation (see Table 8) show an average increase in lung volume initiation. A visual trend towards increasing lung volume initiation with loudness was noted (M = 873.53 cm^3, Normal Loud; M = 962.26 cm^3, 2X Loud).

As can be seen in the top right panel of Figure 9, children with CP initiated maximum phonation tasks at normal and two times normal loudness, across a small range of lung volumes. The box plot indicates a slightly higher

median value and a slightly larger range of lung volume initiations when children with CP were instructed to produce maximum phonations at twice normal loudness. Experimental group averages for lung volume initiation (see Table 8) showed only a marginal increase in lung volume initiation for the louder condition (M = 393.54 cm^3, Normal Loud; M = 436.30 cm^3, 2X Loud). Overall, the control group used a wider range of lung volume initiations and on average, produced maximum phonation tasks at larger lung volumes than children with CP, for both loudness conditions.

Comparisons of matched participants can be made from examining the lower two panels in Figure 4. F1001CL.1 initiated phonations at a lower lung volume (M = 258.2 cm^3, Normal Loud; M = 361.47 cm^3, 2X Loud) than her match, F1001EL.1 (M = 1006.45 cm^3, Normal Loud; M = 967.79 cm^3, 2X Loud). F1201CL.1 initiated phonations at much higher lung volumes (M = 2221.92 cm^3, Normal Loud; M = 2427.89 cm^3, 2X Loud) than her match, F1201EL.1 (M = 163.46 cm^3, Normal Loud; M = 143.45 cm^3, 2X Loud). M1201CL.1 initiated phonations at higher lung volumes (M = 738.8 cm^3, Normal Loud; M = 841.64 cm^3, 2X Loud) than his match M1201EL.1 (M = 222.13 cm^3, Normal Loud, M = 436.3 cm^3, 2X Loud). The two eight-year-old participants performed similarly (See Appendix J for individual means and standard deviations).

Lung Volume Initiation – Sentences

Hypotheses:

2. a) In typical children, LVI will increase from Soft to 4X loud sentence productions.

b) In children with cerebral palsy, LVI will not change from Soft to 4X loud sentence productions.

Figure 10 shows the results for lung volume initiations measured during sentence tasks. As can be seen in the top left panel of Figure 10, children in the control group initiated sentences at soft, normal, two times normal loudness and four times normal loudness, across a small range of lung volumes between 100 and 600 cm^3 above EEL. The box plot indicates a higher median value and larger

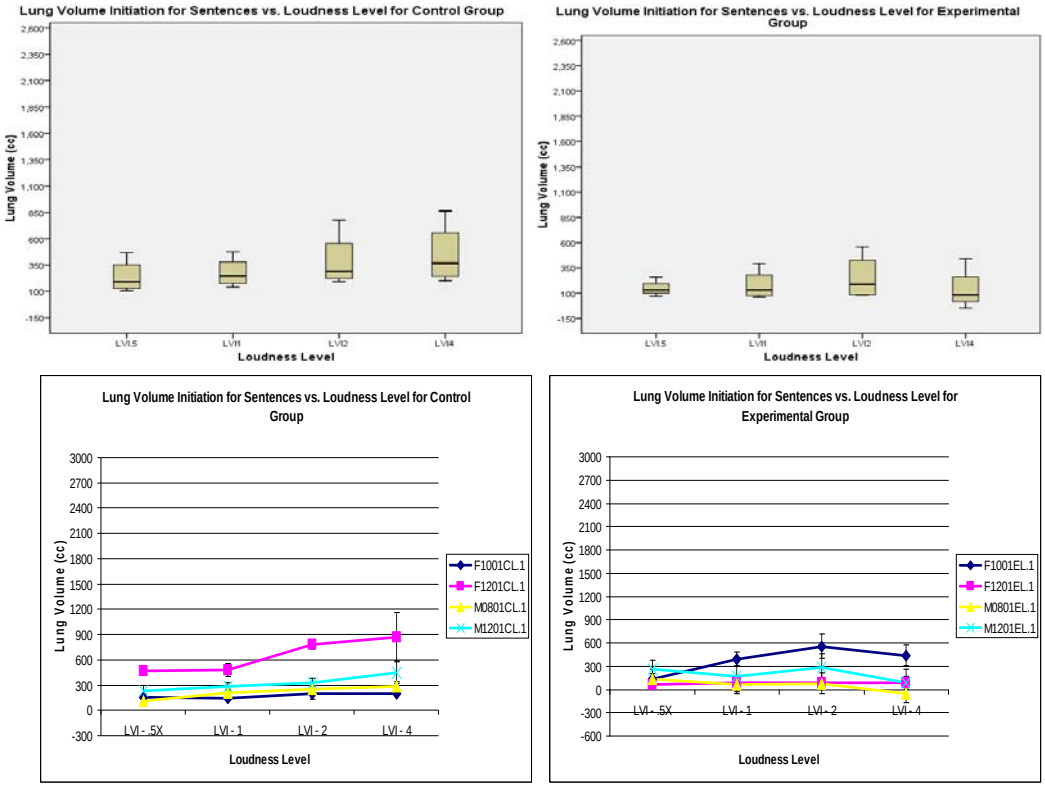

Figure 10. Group and individual data for lung volume initiation (in cm^3) relative to end-expiratory level (0 cm^3) for sentence tasks. Four panels represent group and individual data. Data from the control group (typically developing children) are shown in the two panels on the left and data for the experimental group (children with CP) are shown in the two panels on the right. In all four panels, lung volume is depicted on the *y*-axis in cm^3. Positive values represent lung volumes initiated above end expiratory level (0 cm^3). In all four panels, loudness condition (LVI.5 = Soft, LVI1 = Normal Loudness, LVI2 = 2X Normal Loudness, and LVI4 = 4X Normal Loudness) is depicted on the *x*-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation lung volume initiation values for individual participants. The matched pairs are displayed in the same color.

range for lung volume initiations when children were instructed to produce the sentence at twice normal loudness and four times normal loudness. Control group averages for lung volume initiation (see Table 9) show an average increase in lung volume initiation when participants were asked to produce sentences at increasing levels of loudness (M = 240.90 cm^3, .5X Loud; M = 278.44 cm^3, Normal Loud; M = 388.54 cm^3, 2X Loud; M = 450.50 cm^3, 4X Loud). A visual trend towards increasing lung volume initiation with loudness was noted.

As can be seen in the top right panel of Figure 10, children with CP initiated sentences at soft, normal, two times normal loudness and four times normal loudness, across a small range of lung volumes between 100 and 450 cm^3 above EEL. The box plot indicates a higher median value and larger range of lung volume initiations when children with CP were instructed to produce sentences at twice normal loudness and four times normal loudness. Experimental group averages for lung volume initiation (see Table 9) show an increase in lung volume initiation for normal loudness and two times normal loudness (M = 147.58 cm^3, Soft Loud; M = 179.39 cm^3, Normal Loud; M = 253.66 cm^3 2X Loud). Children with cerebral palsy showed a decrease in lung volume initiation at four times normal loudness (M = 140.02 cm^3; 4X Loud). Overall, the control group, on average, produced sentences at higher lung volumes than children with CP. Control children started from slightly higher lung volumes to produce sentences in the louder conditions. In contrast, children with CP initiated sentences at lower lung volumes but made similar relative adjustments for vocal loudness as observed in the controls with the exception of the loudest condition.

Comparisons of matched participants can be made from examining the lower two panels in Figure 10. All control participants started sentences at higher lung volumes than their matched CP partners across all conditions, which the exception of F1001EL.1 who initiated sentences at higher lung volumes (M = 129.79 cm^3, Soft Loud; M = 394.58 cm^3, Normal Loud; M = 557.95 cm^3, 2X Loud; M = 439.95 cm^3, 4X Loud) than her matched control (M = 153.87 cm^3, Soft Loud; M = 142.05 cm^3, Normal Loud; M = 195.35 cm^3, 2X Loud; M = 202.00 cm^3, 4X Loud) (See Appendix J for individual means and standard deviations). Three participants initiated soft sentences at higher lung volumes than normal loud sentences; namely, F1001CL.1, M0801EL.1, and M1201EL.1.

Lung Volume Termination – Maximum Sustained Phonation

Hypotheses:

3. a) In typical children, LVT will decrease from Normal to 2X loud maximum phonation productions.

   b) In children with cerebral palsy, LVT will not change from Normal to 2X loud maximum phonation productions.

As can be seen in the top left panel of Figure 11, children in the control group terminated maximum phonation tasks at normal and two times normal loudness, across a wide range of lung volumes.

As can be seen in the top left panel of Figure 11, children in the control group terminated maximum phonation tasks at normal and two times normal loudness, across a wide range of lung volumes extending below EEL, as expected. The box plot indicates a lower median value and similar range for lung volume terminations when children were instructed to produce maximum phonations at

twice normal loudness. Control group averages for lung volume termination (see Table 8) show a slight decrease in lung volume termination when children were asked to produce sustained maximum phonations at twice normal loudness.

As can be seen in the top right panel of Figure 11, children with CP terminated maximum phonation tasks at normal and two times normal loudness, across a small range of lung volumes below EEL. The box plot indicates a lower median value and slightly larger range of lung volume terminations when children with CP were instructed to produce maximum phonations at twice normal loudness. Experimental group averages for lung volume termination (see Table 8) show lower lung volume terminations for the louder condition (M = -61.55 cm^3, Normal Loud; -98.43 cm^3, 2X Loud).

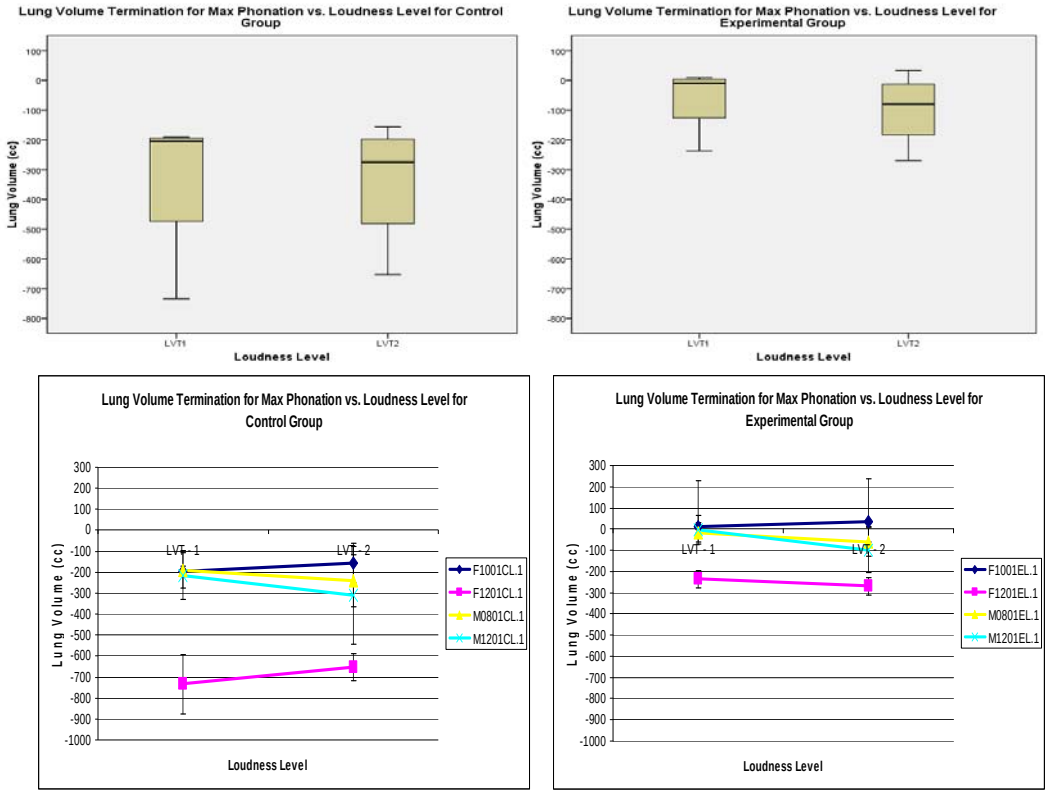

Figure 11. Group and individual data for lung volume termination (in cm^3) relative to end-expiratory level (0 cm^3), for maximum phonation tasks. Four panels represent group and individual data. Data from the control group (typically developing children) are shown in the two panels on the left and data for the experimental group (children with CP) are shown in the two panels on the right. In all four panels, lung volume is depicted on the *y*-axis in cm^3. Positive values represent lung volumes terminated above end expiratory level (0 cm^3). In all four panels, loudness condition (LVT1 = Normal Loudness and LVT2 = 2X Normal Loudness) is depicted on the *x*-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation lung volume termination values for individual participants based on three trials produced in each condition. The control group participants and experimental group participants are age and gender matched. These matched pairs are displayed in the same color.

Overall, the control group used a wider range of lung volume terminations and on average, appeared to terminate maximum phonations at lower lung volumes than children with CP for both loudness conditions. The control group exhibited lung volumes well below EEL, as expected, whereas the children with CP terminated at or only slightly below EEL.

Comparisons of matched participants can be made from examining the lower two panels in Figure 11. The typically developing children terminated their

sentences at much lower lung volumes than their matches with cerebral palsy. Two control participants had higher lung volume terminations for the louder condition (F1001CL.1: *M* = -196.13 cm^3, Normal Loud; *M* = -155.60 cm^3, 2X Loud and F1201CL.1: *M* = -733.33 cm^3, Normal Loud; *M* = -652.17 cm^3, 2X Loud). One child with CP also had higher lung volume terminations for the louder condition (F1001EL.1: *M* = 8.76 cm^3, Normal Loud; *M* = 33.58 cm^3, 2X Loud). Two pairs (M0801CL.1 and EL .l) and (M1201CL.1 and EL.1) showed similar changes in lung volume terminations by ending at lower volumes when producing phonations at twice normal loudness levels. One pair (F1001CL.1CL and EL.1) both terminated at slightly higher lung volumes when producing phonations at twice normal loudness levels. F1201CL.1 and her counterpart F1201EL.1 exhibited opposite lung volume termination patterns (See Appendix I for individual means and standard deviations).

Lung Volume Termination – Sentences

Hypotheses:

4. a) In typical children, LVT will decrease from Soft to 4X loud sentence productions.

   b) In children with cerebral palsy, LVT will not change from Soft to 4X loud sentence productions.

As can be seen in the top left panel of Figure 12, children in the control group terminated sentences at soft, normal, two times normal loudness and four times normal loudness, across a small range of lung volumes at, or slightly below EEL. The box plot indicates a lower median value at soft loudness but a consistent median value for the other loudness conditions. A larger range for lung volume

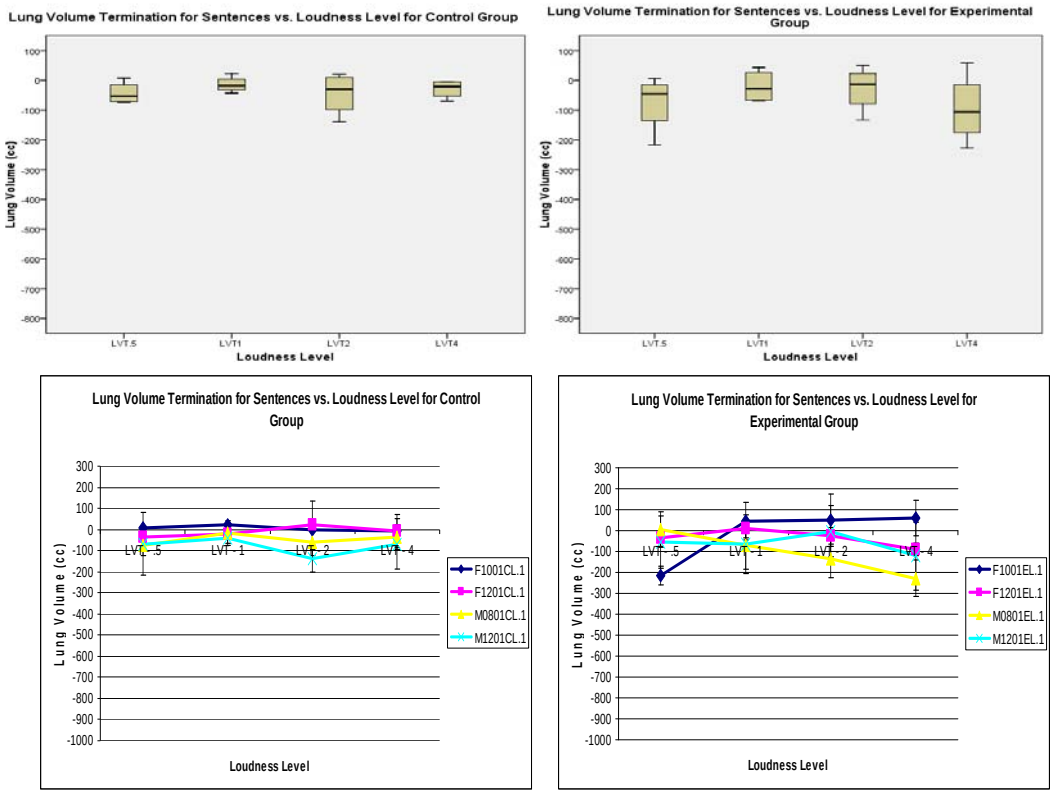

Figure 12. Group and individual data for lung volume termination (in cm^3) relative to end-expiratory level (0 cm^3), for sentence tasks. Four panels represent group and individual data. Data from the control group (typically developing children) are shown in the two panels on the left and data for the experimental group (children with CP) are shown in the two panels on the right. In all four panels, lung volume is depicted on the *y*-axis in cm^3. Positive values represent lung volumes terminated above end expiratory level (0 cm^3). In all four panels, loudness condition (LVT.5 = Soft, LVT1 = Normal Loudness, LVT2 = 2X Normal Loudness, and LVT4 = 4X Normal Loudness) is depicted on the *x*-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation lung volume termination values for individual participants. These matched pairs are displayed in the same color.

terminations were noted when typical children were instructed to produce the sentence at twice normal loudness. Control group averages for lung volume termination (see Table 9) show a fairly stable lung volume termination across loudness conditions. A visual trend towards decreasing lung volume termination with loudness was not noted (M = -43.02 cm^3, Soft Loud; M = -14.14 cm^3, Normal Loud; M = -44.52 cm^3, 2X Loud; M = -28.69 cm^3, 4X Loud).

As can be seen in the top right panel of Figure 12, children with CP terminated sentence tasks at soft, normal, two times normal loudness and four times loudness, across a larger range of lung volumes at or below EEL. The box plot indicates a lower median value and larger range of lung volume terminations when children with CP were instructed to produce sentences at soft loudness and four times normal loudness. Experimental group averages for lung volume termination (see Table 9) show lower lung volume terminations for soft loudness and four times normal loudness (M = -75.42 cm^3, Soft Loud; M = -20.13 cm^3, Normal Loud; M = -27.52 cm^3, 2X Loud; M = -95.11 cm^3, 4X Loud).

Overall, the control group, on average, terminated sentences at relatively consistent lung volumes at or near EEL, whereas children with CP tended to terminate sentences at lower lung volumes and were more variable than controls. However, both groups exhibited similar lung volume termination values when asked to produce sentences at twice normal loudness levels.

Visual examination of individual patterns showed two discrepant patterns. First, F1001EL.1 terminated well below EEL compared to her control counterpart for sentences produced at half normal loudness levels. However, she approximated terminations at or near the same levels as her control counterpart for all other loudness conditions. The second discrepant pattern was shown by M0801EL.1. Relative to his control counterpart, M0801.EL1 terminated sentences at increasingly lower lung volumes with increasing loudness, going well below EEL. In contrast, his control counterpart consistently terminated his breath

groups at lung volumes slightly below EEL for all loudness conditions (See Appendix J for individual means and standard deviations).

Lung Volume Excursion – Maximum Sustained Phonation

Hypotheses:

5. a) In typical children, LVE will increase from Normal to 2X loud maximum phonation productions.

   b) In children with cerebral palsy, LVE will not change from Normal to 2X loud maximum phonation productions.

As can be seen in the top left panel of Figure 13, children in the control group produced maximum phonation tasks at normal and two times normal loudness, using a wide range of their vital capacity ranging from approximately 20 to 80 percent. The box plot indicates a slightly higher median value and a

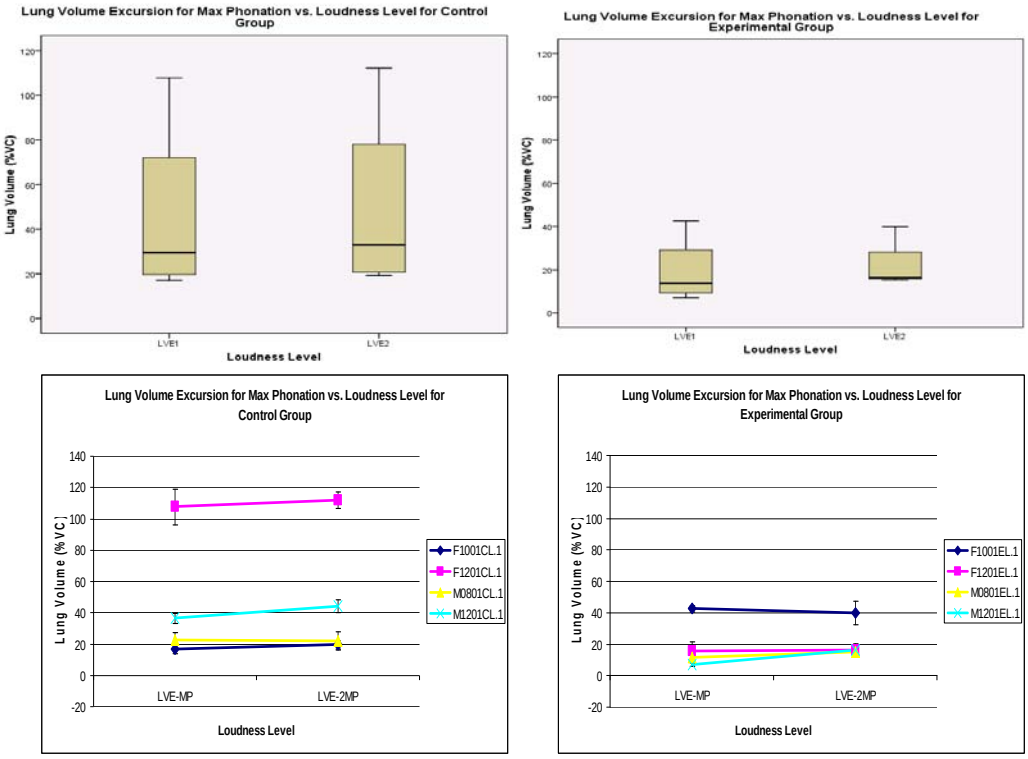

Figure 13. Group and individual data for lung volume excursion (in percent vital capacity), relative to predicted vital capacity, for maximum phonation tasks. Four panels represent group and individual data. Data from the control group (typically developing children) are shown in the two panels on the left and data for the experimental group (children with CP) are shown in the two panels on the right. In all four panels, lung volume is depicted on the *y*-axis in percent vital capacity. In all four panels, loudness condition (LVE1 (LVEMP) = Normal Loudness and LVE2 (LVE2MP) = 2X Normal Loudness) is depicted on the *x*-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation lung volume excursion values for individual participants. These matched pairs are displayed in the same color.

somewhat larger range for lung volume excursions when children were instructed to produce maximum phonations at twice normal loudness. Control group averages for lung volume excursion (see Table 8) show a slight average increase in lung volume excursion. A marginal visual trend towards increasing lung volume excursion with loudness was noted (M = 45.93 % VC, Normal Loud; M = 49.41 % VC, 2X Loud).

As can be seen in the top right panel of Figure 13, children with CP produced maximum phonation tasks at normal and two times normal loudness,

using a small range of their vital capacity approximately between 10 and 30 percent. The box plot indicates a slightly higher median value and a slightly smaller range of lung volume excursions when children with CP were instructed to produce maximum phonations at twice normal loudness. Experimental group averages for lung volume excursion (see Table 8) show only a marginal increase in lung volume excursion for the louder condition (*M* = 19.20 % VC, Normal Loud; 21.91 %VC at 2X Loud).

Overall, the control group used a wider range of lung volume excursions and on average, produced maximum phonations with larger relative excursions (%VC) than children with CP. Whereas control children appeared to use more air when producing louder phonations, children with CP did not. Absolute lung volumes derived from the vital capacity maneuvers revealed that on average, children with CP maximally expired about half the volume of air than the typically developing children (*M* = 799 cc and 1472 cc, respectively).

Comparisons of matched participants can be made from examining the lower two panels in Figure 13. It appeared that only one child (F1201CL.1) in the entire sample phonated throughout most of her vital capacity. All other children used a much smaller range for sustained maximum phonations produced at either loudness level. In one case (F1001EL.1), lung volume excursions were larger for phonations produced at normal and twice normal loudness than her control counterpart (See Appendix I for individual means and standard deviations).

Lung Volume Excursion – Sentences

Hypotheses:

6. a) In typical children, LVE will increase from Soft to 4X loud sentence productions.

   b) In children with cerebral palsy, LVE will not change from Soft to 4X loud sentence productions.

As can be seen in the top left panel of Figure 14, children in the control group produced sentences at soft, normal, two times normal loudness and four times normal loudness, across a small range of lung volume excursions ranging

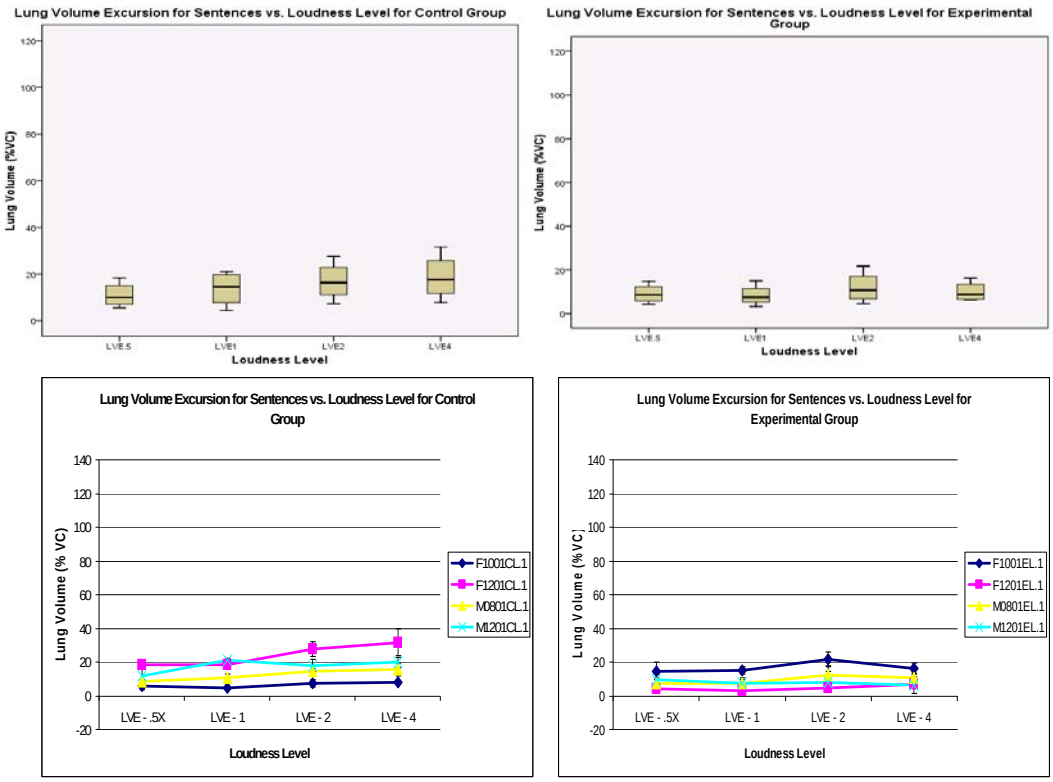

Figure 14. Group and individual data for lung volume excursion (in percent vital capacity), relative to predicted vital capacity, for sentence tasks. Four panels represent group and individual data. Data from the control group (typically developing children) are shown in the two panels on the left and data for the experimental group (children with CP) are shown in the two panels on the right. In all four panels, lung volume is depicted on the y-axis in percent vital capacity. In all four panels the loudness condition (LVE.5 = Soft, LVE1 = Normal Loudness, LVE2 = 2X Normal Loudness, and LVE4 = 4X Normal Loudness) is depicted on the x-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation lung volume excursionvalues for individual participants. These matched pairs are displayed in the same color.

from approximately 20 to 30 percent VC. The box plot indicates a higher median value and larger range for lung volume excursions when children were instructed

to produce the sentence at twice normal loudness and four times normal loudness. Further, the box plot indicates a lower median value and smaller range for lung volume excursions when children were instructed to produce the sentence at half normal loudness. Control group averages for lung volume excursion (see Table 9) show an average increase in lung volume excursion (M = 11.05 %VC, Soft Loud; M = 13.67 %VC, Normal Loud; M = 16.94 %VC, 2X Loud; M = 18.69 %VC, 4X Loud). A visual trend towards increasing lung volume excursion with loudness was noted.

As can be seen in the top right panel of Figure 14, children with CP produced sentences at soft, normal, two times normal loudness and four times normal loudness, across a smaller range of lung volumes (5 to 20 %VC) compared to the typical children. The box plot indicates a higher median value and larger range of lung volume excursions when children with CP were instructed to produce sentences at twice normal loudness. Experimental group averages for lung volume excursion (see Table 9) show a marginal increase in lung volume excursion for two times normal loudness, followed by a slight decrease in lung volume excursion for four times normal loudness (M = 9.01 %VC, Soft Loud; M = 8.27 %VC, Normal Loud; M = 11.90 %VC, 2X Loud; M = 10.00 %VC, 4X Loud). A visual trend for increasing lung volume excursion with loudness was not noted.

Overall, the control group used a wider range of lung volume excursions and on average, produced sentences with greater lung volume excursions than children with CP, for all loudness conditions. In addition, control children

increased lung volume excursion commensurate with increases in the loudness condition. Children with CP did not markedly adjust lung volume excursions with changes in vocal loudness.

Comparisons of matched participants can be made from examining the lower two panels in Figure 14. The control group produced sentences with larger lung volume excursions than the experimental participants, with the exception of F1001EL.1. She produced sentences with greater lung volume excursions (*M* = 14.79 %VC, Soft Loud; *M* = 14.99 %VC, Normal Loud; *M* = 21.7 %VC, 2X Loud; *M* = 16.24 %VC, 4X Loud) than her matched control partner, F1001CL.1 (*M* = 5.5 %VC, .5X Loud; *M* = 4.47%VC, Normal Loud; *M* = 7.38 %VC, 2X Loud; *M* = 7.77%VC, 4X Loud) (See Appendix J for individual means and standard deviations).

Intercostal Muscle Activity – Maximum Sustained Phonation

Hypotheses:

7. a) In typical children, %MVE will increase in the intercostal muscles from Normal to 2X loud maximum phonation productions.

   b) In children with cerebral palsy, %MVE will increase in the intercostals muscles from Normal to 2X loud maximum phonation productions.

As can be seen in the top left panel of Figure 15, children in the control group produced maximum phonation tasks at normal and two times normal loudness, across a small range of their %MVE but generally using approximately 50% of their MVE. The box plot indicates a higher median value and a larger range for %MVE when children were instructed to produce maximum phonations

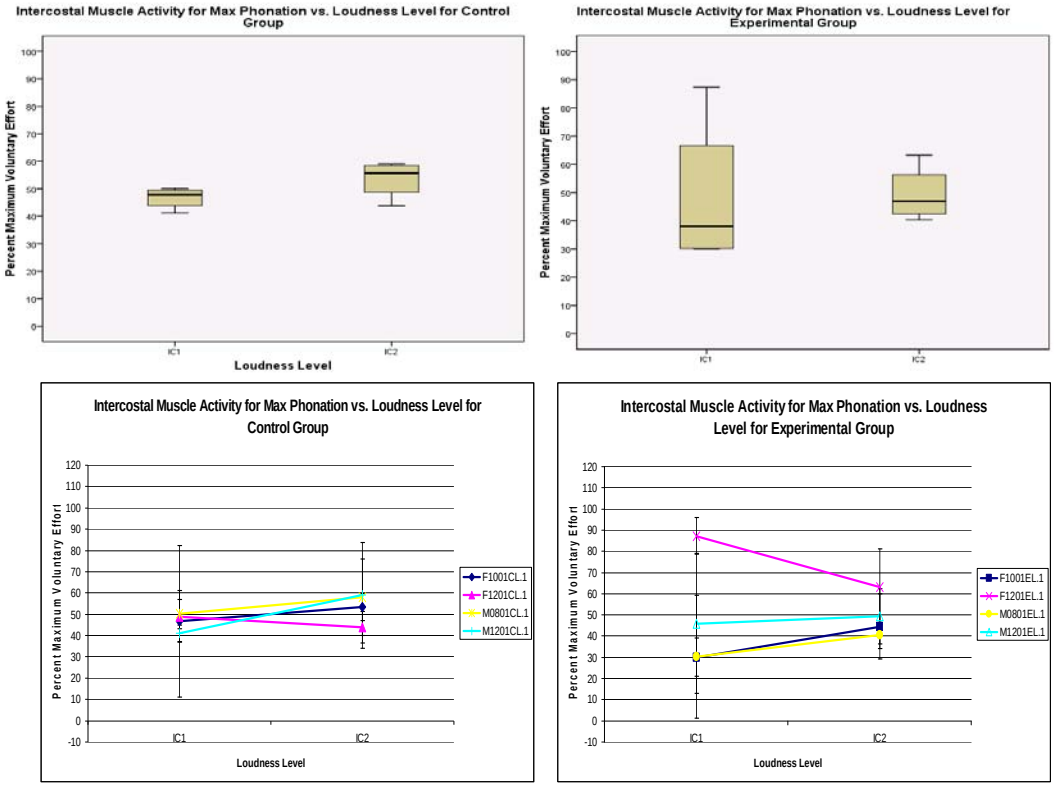

Figure 15. Group and individual data for intercostal muscle activity (in percent maximum voluntary effort, %MVE), relative to each participant's maximum intercostal voluntary effort, for maximum phonation tasks. Four panels represent group and individual data. Data from the control group (typically developing children) is shown in the two panels on the left and data for the experimental group (children with CP) is shown in the two panels on the right. In all four panels, intercostal muscle activity is depicted on the y-axis in %MVE. In all four panels, loudness condition (IC1 = Normal Loudness and IC2 = 2X Normal Loudness) is depicted on the x-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation intercostals muscle activity values (in %MVE) for individual participants. These matched pairs are displayed in the same color**.**

at twice normal loudness. Control group averages for %MVE for intercostal muscles (see Table 8) show a small average increase in %MVE (*M* = 46.74 % MVE, Normal Loud; *M* = 53.58% MVE, 2X Loud). A visual trend towards increasing %MVE for intercostal muscles with loudness was noted.

As can be seen in the top right panel of Figure 15, children with CP produced maximum phonation tasks at normal and two times normal loudness, using a large range of their %MVE, particularly at normal loudness. The box plot

indicates a higher median value and a smaller range of %MVE when children with CP were instructed to produce maximum phonations at twice normal loudness. Experimental group averages for %MVE (see Table 8) show only a marginal increase in %MVE for the louder condition (*M* = 48.37 % MVE, Normal Loud; 49.40 % MVE at 2X Loud).

Overall, the experimental group used a wider range of %MVE, predominantly at normal loudness. The experimental group and control group, on average, produced maximum phonations using similar %MVE, for both loudness conditions.

Comparisons of matched participants can be made from examining the lower two panels in Figure 15. The typically developing children produced phonations with greater %MVE than the children with CP, with the exception of F1201CL.1. She produced phonations with less %MVE (*M* = 48.92 % MVE, Normal Loud; *M* = 43.94 % MVE, 2X Loud) than her match, F1201EL.1 (*M* = 87.29 % MVE, Normal Loud; *M* = 63.26 % MVE, 2X Loud) (See Appendix I for individual means and standard deviations). These participants also were the only participants to show a decrease in %MVE from normal loudness to twice normal loudness.

Intercostal Muscle Activity – Sentences

Hypotheses:

8. a) In typical children, % MVE for intercostal muscles will increase from .5X Loud to 4X loud sentence productions.

b) In children with cerebral palsy, % MVE for intercostal muscles will increase from .5X Loud to 4X loud sentence productions.

As can be seen in the top left panel of Figure 16, the box plot indicates a higher median value and larger range for %MVE when children in the control group were instructed to produce the sentence at four times normal loudness.

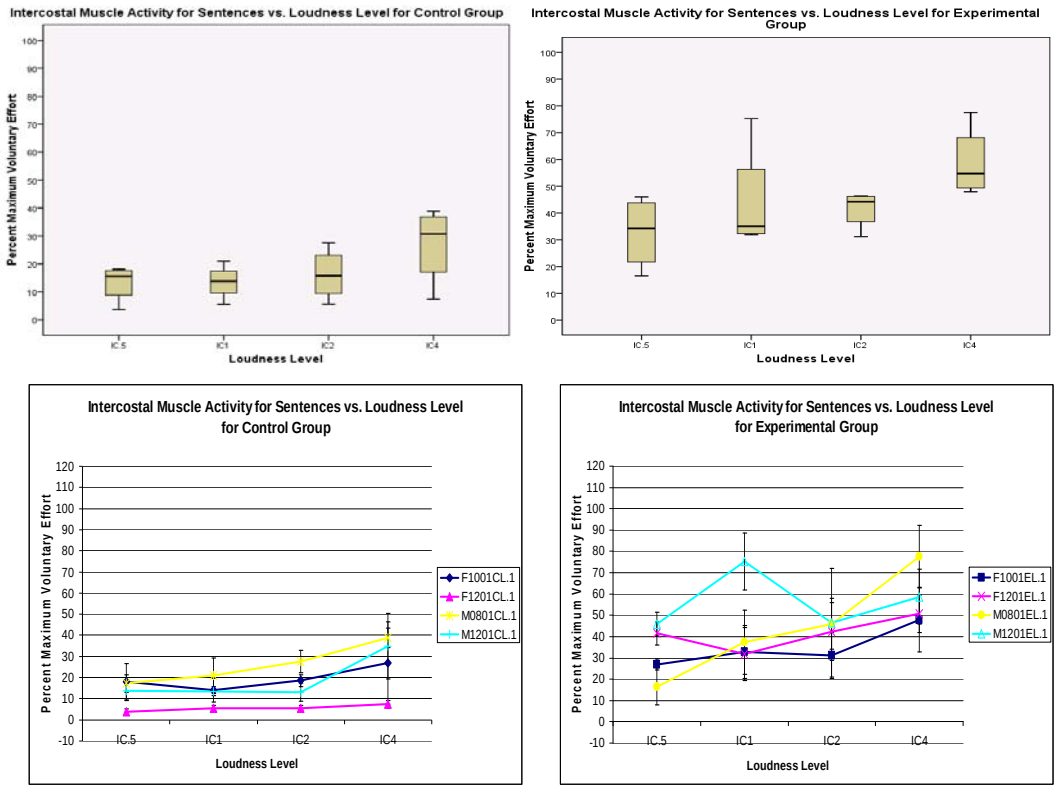

Figure 16. Group and individual data for intercostal muscle activity (in %MVE), relative to each participant's maximum intercostal voluntary effort, for sentence tasks. Four panels represent group and individual data. Data from the control group (typically developing children) is shown in the two panels on the left and data for the experimental group (children with cerebral palsy) is shown in the two panels on the right. In all four panels, intercostals muscle activity is depicted on the y-axis in %MVE effort. In all four panels the loudness condition (IC.5 = .5X Loud, IC1 = Normal

Loudness, IC2 = 2X Normal Loudness, and IC4 = 4X Normal Loudness) is depicted on the x-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation %MVE values for individual participants. These matched pairs are displayed in the same color.

Control group averages for percent %MVE (see Table 9) show an average increase in %MVE at four times loudness and an average decrease in %MVE at half normal loudness (*M* = 13.26 % MVE, .5X Loud; *M* = 17.39 % MVE, Normal Loud; *M* = 16.21 % MVE, 2X Loud; *M* = 26.99 % MVE, 4X Loud). A visual trend towards increasing intercostal muscle activity with loudness, predominately at four times normal loudness was noted.

As can be seen in the top right panel of Figure 16, children with CP produced sentences at half normal loudness, normal, two times normal loudness and four times loudness, across a larger range of %MVE compared to the typical children. The box plot indicates a higher median value for %MVE when children with CP were instructed to produce sentences at twice normal loudness and four times normal loudness. Further, the box plot indicates a smaller median value for %MVE when children with CP were instructed to produce sentences at half normal loudness. The smallest range in values for %MVE occurred at twice normal loudness. Experimental group averages for %MVE (see Table 9) show an increase in %MVE for four times normal loudness and a decrease in %MVE for half normal loudness (*M* = 32.76 % MVE, .5X Loud; *M* = 43.78 % MVE, Normal Loud; *M* = 41.49 % MVE, 2X Loud; *M* = 58.73 % MVE, 4X Loud). A visual trend for increasing %MVE with loudness was noted.

Overall, the experimental group used a larger range of %MVE and on average, produced sentences with greater %MVE for intercostal muscles than

typically developing children, for all loudness conditions. Both groups appeared to increase the %MVE of intercostal muscles when asked to produce sentences at louder than normal levels. However, sentences produced at softer than normal levels did not appear to change intercostal %MVE values from those observed when children spoke at normal loudness levels.

Comparisons of matched participants can be made from examining the lower two panels in Figure 16. Children with cerebral palsy produced sentences with greater %MVE for the intercostal muscles compared to their matched control partners. Overall, participants indicated a linear increase in intercostal muscle activity as loudness increased, with the exception of F1201EL.1 and M1201EL.1. F1201EL.1 used greater %MVE at half normal loudness compared to normal loudness (*M* = 41.52 % MVE, .5X Loud; *M* = 31.9 % MVE, Normal Loud; *M* = 42.38 % MVE, 2X Loud; *M* = 50.71 % MVE, 4X Loud) and M1201EL.1 used the greatest %MVE for the intercostal muscles at normal loudness (*M* = 46.00 % MVE, .5X Loud; *M* = 75.22 % MVE, Normal Loud; *M* = 46.4 % MVE, 2X Loud; *M* = 58.73 % MVE, 4X Loud) (See Appendix J for individual means and standard deviations). Generally, CP children were more variable than their matched counterparts.

Oblique Muscle Activity – Maximum Sustained Phonation

Hypotheses:

9. a) In typical children, %MVE will increase in the oblique muscles from Normal to 2X loud maximum phonation productions.

   b) In children with cerebral palsy, %MVE will increase in the oblique muscles from Normal to 2X loud maximum phonation productions.

As can be seen in the top left panel of Figure 17, the box plot indicates a higher median value and a similar range for %MVE when children were instructed to produce maximum phonations at twice normal loudness. Control group averages for %MVE for oblique muscles (see Table 8) show a small

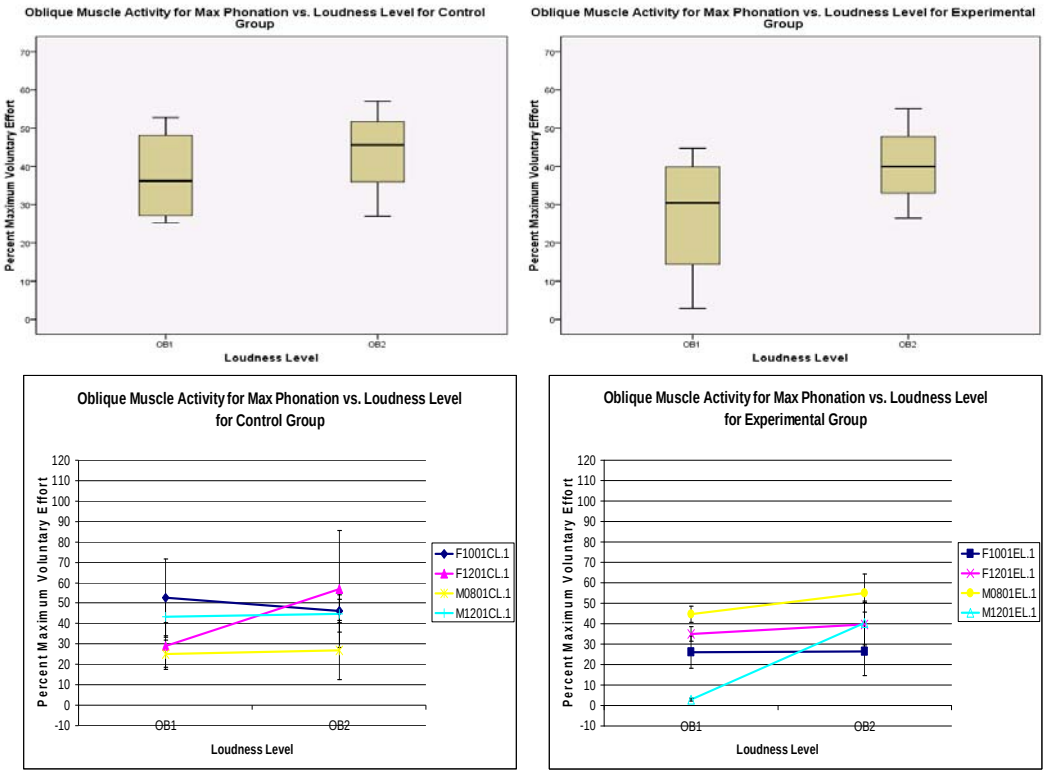

Figure 17. Group and individual data for oblique muscle activity (in %MVE), relative to each participant's %MVE, for maximum phonation tasks. Four panels represent group and individual data. Data from the control group (typically developing children) is shown in the two panels on the left and data for the experimental group (children with cerebral palsy) is shown in the two panels on the right. In all four panels, oblique muscle activity is depicted on the y-axis in %MVE. In all four panels, loudness condition (OB1 = Normal Loudness and OB2 = 2X Normal Loudness) is depicted on the x-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation obliques muscle activity values (in %MVE) for individual participants. These matched pairs are displayed in the same color.

average increase in %MVE (*M* = 37.61 % MVE, Normal Loud; *M* = 43.82% MVE, 2X Loud). A visual trend towards increasing %MVE effort for oblique muscles with loudness was noted.

As can be seen in the top right panel of Figure 17, children with CP produced maximum phonation tasks at normal and two times normal loudness. The box plot indicates a higher median value and a smaller range of %MVE when children with CP were instructed to produce maximum phonations at twice normal loudness. Experimental group averages for %MVE (see Table 8) show an increase in %MVE for the louder condition (*M* = 27.16 % MVE, Normal Loud; 40.37 % MVE at 2X Loud).

Overall, the experimental group exhibited a wider range of %MVE values at normal loudness but showed similar ranges and median %MVE values as controls for maximum phonations produced at twice normal loudness levels. Whereas the increase in %MVE from normal to twice normal loudness was apparent for control children, children with CP appeared to have a larger relative increase in %MVE when producing phonations at louder levels.

Comparisons of matched participants can be made from examining the lower two panels in Figure 17. The typically developing children produced phonations with greater %MVE for the oblique muscles than the children with CP, with the exception of M0801CL.1. He produced phonations with less %MVE (*M* = 25.23 % MVE, Normal Loud; *M* = 26.98 % MVE, 2X Loud) than his counterpart with CP, M0801EL.1 (*M* = 44.72 % MVE, Normal Loud; *M* = 55.13 % MVE, 2X Loud). In addition, all participants showed at least a slight increase in %MVE for oblique muscles from normal loudness to twice normal loudness, except F1001CL.1. She decreased maximum voluntary for oblique muscles from normal loudness to twice normal loudness (*M* = 52.77 % MVE, Normal Loud; *M*

= 46.29 % MVE, 2X Loud) (See Appendix I for individual means and standard deviations).

Oblique Muscle Activity – Sentences

Hypotheses:

10. a) In typical children, % MVE for oblique muscles will increase from .5X Loud to 4X loud sentence productions.

    b) In children with cerebral palsy, % MVE for oblique muscles will increase from .5X Loud to 4X loud sentence productions.

As can be seen in the top left panel of Figure 18, the box plot reveals the highest median value and the largest range for %MVE values obtained for oblique muscles when children were instructed to produce the sentence at four

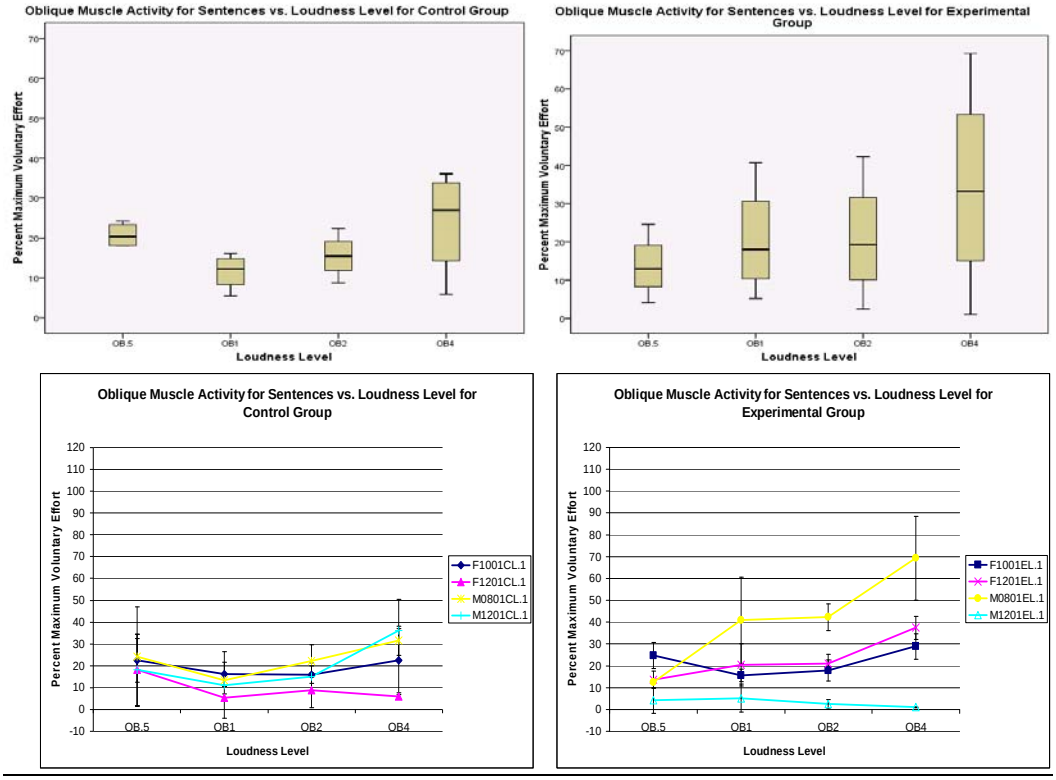

Figure 18. Group and individual data for oblique muscle activity (in %MVE), relative to each participant's %MVE, for sentence tasks. Four panels represent group and individual data. Data from the control group (typically developing children) is shown in the two panels on the left and data for the experimental group (children with CP) is shown in the two panels on the right. In all four panels, oblique muscle activity is depicted on the y-axis in %MVE. In all four panels the loudness condition (OB.5 = .5X Loud, OB1 = Normal Loudness, OB2 = 2X Normal Loudness, and OB4 = 4X Normal Loudness) is depicted on the x-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation %MVE values for individual participants. These matched pairs are displayed in the same color.

times normal loudness. In addition, the box plot indicates a higher median value for half normal loudness and two times normal loudness when compared to normal loudness. Control group averages for %MVE effort (see Table 9) show an average increase in %MVE from normal loudness to half normal loudness, two times normal loudness and four times loudness (*M* = 20.76 % MVE, .5X Loud; *M* = 11.56 % MVE, Normal Loud; *M* = 15.49 % MVE, 2X Loud; *M* = 24.01 % MVE, 4X Loud). The greatest %MVE occurred at four times normal loudness followed by half normal loudness. A visual trend towards increasing oblique

muscle activity from normal loudness to half normal loudness and from normal loudness to four times normal loudness was noted.

As can be seen in the top right panel of Figure 18, children with CP produced sentences at half normal loudness, normal, two times normal loudness and four times normal loudness, across a large range of %MVE. The box plot indicates a higher median value and larger range for %MVE when children were instructed to produce sentences at four times normal loudness. Further, the box plot indicates a smaller median value and a smaller range for %MVE when children with CP were instructed to produce sentences at half normal loudness. Experimental group averages for %MVE (see Table 9) show an increase in %MVE with an increase in loudness (M = 13.71 % MVE, Soft Loud; M = 17.92 % MVE, Normal Loud; M = 20.87 % MVE, 2X Loud; M = 34.15 % MVE, 4X Loud). A visual trend for increasing percent %MVE with loudness was noted.

Overall, the experimental group used a larger range of %MVE and on average, produced sentences with lower %MVE for oblique muscles at half normal loudness but produced sentences with greater percent %MVE for oblique muscles at the other levels of loudness, compared to typically developing children.

Comparisons of matched participants can be made from examining the lower two panels in Figure 18. F1001CL.1 and F1001EL.1 produced sentences with similar %MVE for the oblique muscles. F1201CL.1 and M0801CL.1 produced sentences with lower %MVE for the oblique muscles, except at half normal loudness, compared to their matches with CP, F1201EL.1 and

M0801EL.1, respectively. M1201CL.1 produced sentences with more %MVE for the oblique muscles compared to his counterpart with CP, M1201EL.1 (See Appendix J for individual means and standard deviations).

Speed Quotient – Maximum Sustained Phonation

Hypotheses:

11. a) In typical children, there will be no change in speed quotient from Normal to 2X loud maximum phonation productions.

    b) In children with cerebral palsy, speed quotient will increase from Normal to 2X loud maximum phonation productions.

As can be seen in the top left panel of Figure 19, the box plot indicates a higher median value and a larger range for speed quotient when children were

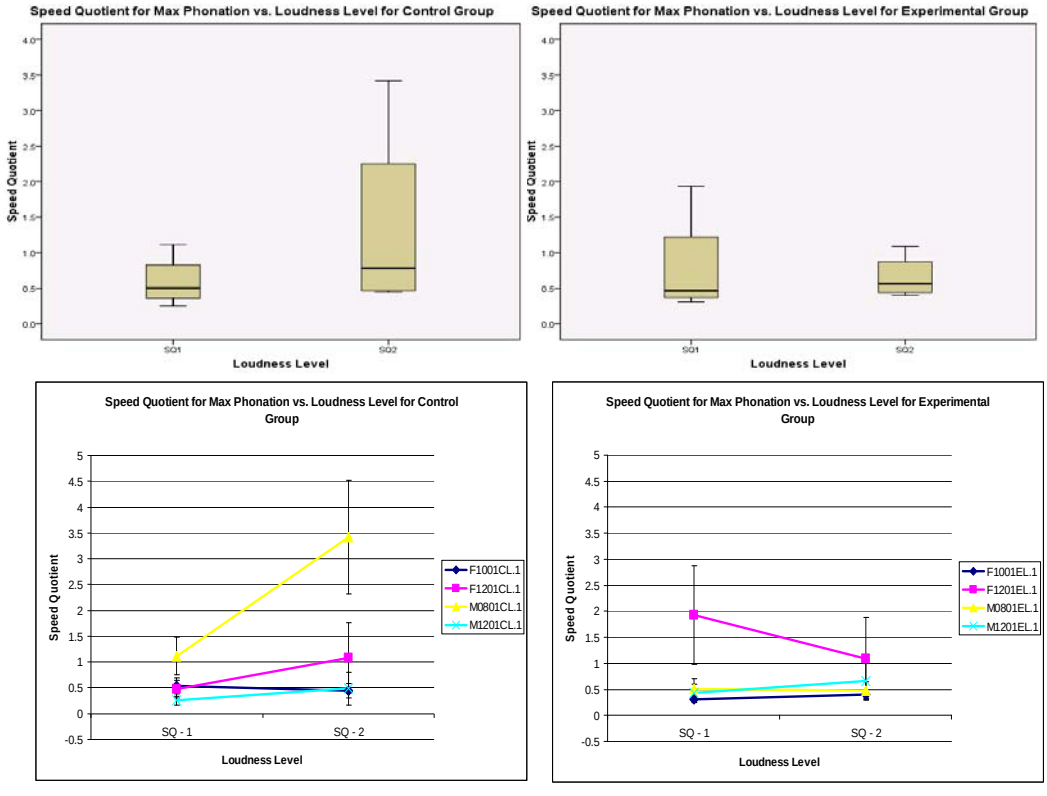

Figure 19. Group and individual data for speed quotient (SQ) (the time taken for impedance to rise from 20% to 80% divided by the time taken for it to decrease from 80% to 20%) for maximum phonation tasks. Four panels represent group and individual data. Data from the control group (typically developing children) are shown in the two panels on the left and data for the experimental group (children with cerebral palsy) are shown in the two panels on the right. In all

four panels, SQ is depicted on the y-axis. In all four panels the loudness condition (SQ1 = Normal Loudness and SQ2 = 2X Normal Loudness) is depicted on the x-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation SQ values for individual participants. These matched pairs are displayed in the same color.

instructed to produce maximum phonations at twice normal loudness. Control group averages for speed quotient (see Table 8) show an average increase in speed quotient (*M* = 0.59, Normal Loud; *M* = 1.36, 2X Loud). A visual trend towards increasing speed quotient with loudness was noted.

As can be seen in the top right panel of Figure 19, the box plot indicates a higher median value and slightly smaller range of speed quotients when children with CP were instructed to produce maximum phonations at twice normal loudness. Experimental group averages for speed quotient (see Table 8) show a decrease in speed quotient for the louder condition (*M* = 0.79, Normal Loud; *M* = 0.66, 2X Loud).

Overall, the control group used a wider range of speed quotients at two times normal loudness compared to the experimental group, but the experimental group used a wider range of speed quotients at normal loudness. The control group had a higher average speed quotient than the experimental group at two times normal loudness. In contrast, the experimental group had a higher average speed quotient than the control group at normal loudness.

Comparisons of matched participants can be made from examining the lower two panels in Figure 19. All participants showed an increase in speed quotient from normal loudness to twice normal loudness, with the exception of F1001CL.1 (*M* = 0.54, Normal Loud; *M* = 0.44, 2X Loud) and F1201EL.1 (*M* = 1.93, Normal Loud; *M* = 1.09, 2X Loud). These two participants showed a

decrease in speed quotient from normal loudness to twice normal loudness. In addition, M0801EL.1 (*M* = 0.5, Normal Loud; *M* = 0.48, 2X Loud) did not change speed quotient across conditions (See Appendix K for individual means and standard deviations).

Speed Quotient for "I" – Sentences

Hypotheses:

12. a) In typical children, speed quotient for "I" will not change from Soft to 4X loud sentence productions.

    b) In children with cerebral palsy, speed quotient for "I" will increase from Soft to 4X loud sentence productions.

As can be seen in the top left panel of Figure 20, the box plot reveals the highest median value and the largest range for speed quotient when children in the

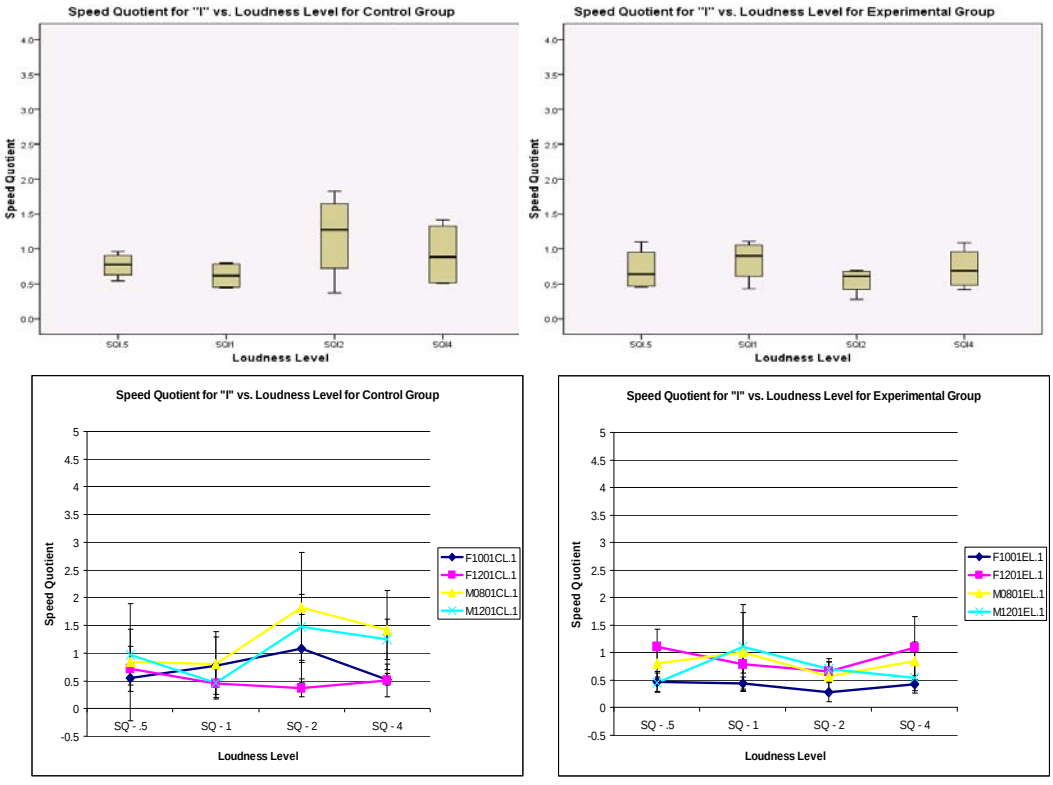

Figure 20. Group and individual data for SQ (the time taken for impedance to rise from 20% to 80% divided by the time taken for it to decrease from 80% to 20%) for the vowel "I" produced during the sentence tasks. Four panels represent group and individual data. Data from the control

group (typically developing children) are shown in the two panels on the left and data for the experimental group (children with cerebral palsy) are shown in the two panels on the right. In all four panels, speed quotient is depicted on the y-axis. In all four panels the loudness condition (SQ.5 = Soft, SQ1 = Normal Loudness, SQ2 = 2X Normal Loudness, and SQ4 = 4X Normal Loudness) is depicted on the x-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation SQ values for individual participants. These matched pairs are displayed in the same color.

control group were instructed to produce the sentence at two times normal loudness. Further, the box plot indicates the second highest median value and range for speed quotient occurred at four times normal loudness followed by half normal loudness. Control group averages for speed quotient (see Table 9) show an average increase in speed quotient from normal loudness to half normal loudness, two times normal loudness and four times loudness. The greatest speed quotient occurred at two times normal loudness followed by four times normal loudness and then half normal loudness (M = 0.77, Soft Loud; M = 0.62, Normal Loud; M = 1.19, 2X Loud; M = 0.92, 4X Loud). A visual trend towards increasing speed quotient from normal loudness to half normal loudness and from normal loudness to two times normal loudness and four times normal loudness was noted.

As can be seen in the top right panel of Figure 20, the box plot indicates minimal adjustment in speed quotient median and a small speed quotient range across loudness conditions for children in the experimental group. The highest median occurred at normal loudness. Experimental group averages for speed quotient (see Table 9) do not show a trend (M = 0.71, .5X Loud; M = 0.84, Normal Loud; M = 0.55, 2X Loud; M = 0.72, 4X Loud). No visual trend for SQ across loudness conditions is readily apparent.

Overall, the control group used a larger range of speed quotients at two times normal loudness and four times normal loudness and on average, produced

sentences with a larger speed quotient at two times normal loudness and four times normal loudness compared to children with cerebral palsy. The children preformed similarly at half normal loudness. At normal loudness, the experimental group produced sentences with a higher speed quotient than the control group.

Comparisons of matched participants can be made from examining the lower two panels in Figure 20. Control participants used higher speed quotients than the experimental participants, excluding the normal loudness condition where M0801CL.1 and M1201CL.1 both used lower speed quotients than their experimental partners. F1201CL.1 was an exception, as she produced sentences with lower speed quotients (*M* = 0.72, .5X Loud; *M* = 0.44, Normal Loud; *M* = 0.37, 2X Loud; *M* = 0.51, 4X Loud) than her match, F1201EL.1 (*M* = 1.10, .5X Loud; *M* = 0.79, Normal Loud; *M* = 1.83, 2X Loud; *M* = 1.42, 4X Loud) (See Appendix L-1 for individual means and standard deviations).

Speed Quotient for s"a"p – Sentences

Hypotheses:

13. a) In typical children, speed quotient for s "a"p will not change from .5X Loud to 4X loud sentence productions.

    b) In children with cerebral palsy, speed quotient for s "a"p will increase from .5X Loud to 4X loud sentence productions.

As can be seen in the top left panel of Figure 21, the box plot indicates a higher median value and a larger range for speed quotient when children in the control group were instructed to produce the sentence at two times and four times normal loudness. Further, the box plot shows a higher median value for speed

quotient at half normal loudness. Control group averages for speed quotient (see Table 9) show an average increase in speed quotient from normal loudness to half normal loudness, two times normal loudness and four times loudness. The greatest speed quotient occurred at four times normal loudness followed by two times normal loudness and then half normal loudness (*M* = 0.72, .5X Loud; *M* = 0.54, Normal Loud; *M* = 0.88, 2X Loud; *M* = 1.11, 4X Loud). A visual trend towards increasing speed quotient from normal loudness to half normal loudness and from normal loudness to two times normal loudness and four times normal loudness was noted.

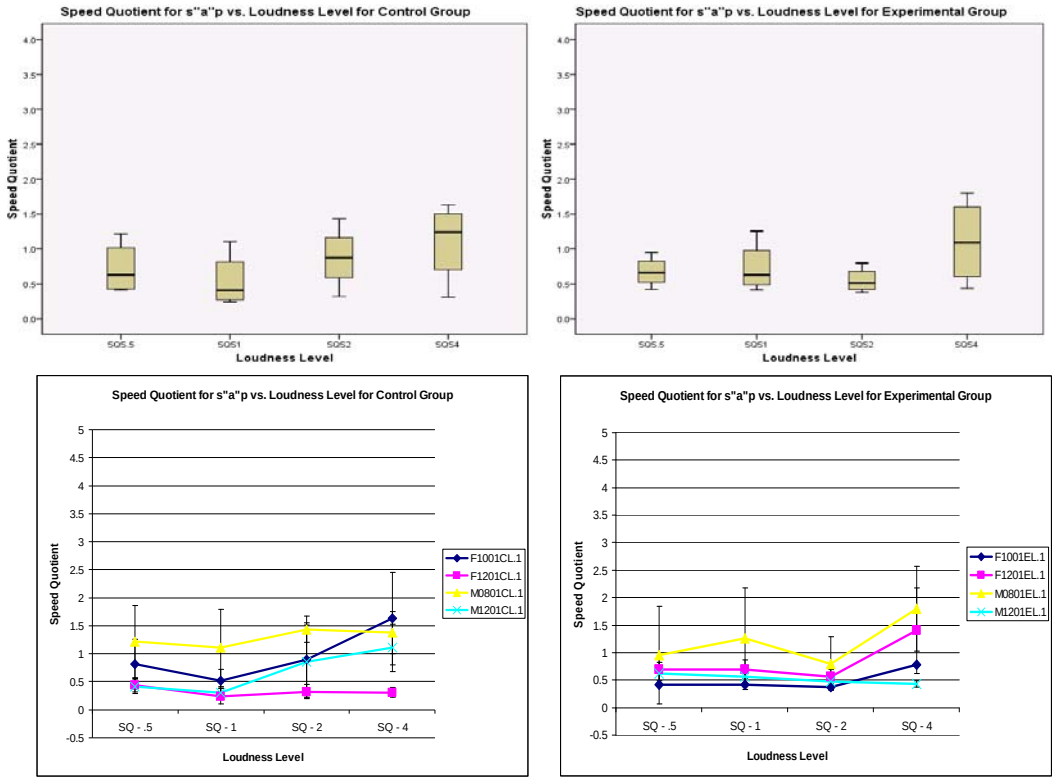

Figure 21. Group and individual data for SQ (the time taken for impedance to rise from 20% to 80% divided by the time taken for it to decrease from 80% to 20%) for the vowel in s "a"p produced during the sentence tasks. Four panels represent group and individual data. Data from the control group (typically developing children) are shown in the two panels on the left and data for the experimental group (children with cerebral palsy) are shown in the two panels on the right. In all four panels, speed quotient is depicted on the y-axis. In all four panels the loudness condition (SQ.5 = .5X Loud, SQ1 = Normal Loudness, SQ2 = 2X Normal Loudness, and SQ4 = 4X Normal Loudness) is depicted on the x-axis. The top two panels represent box and whisker plots,

indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation SQ values for individual participants. These matched pairs are displayed in the same color.

As can be seen in the top right panel of Figure 21, the box plot indicates the highest speed quotient median and the largest speed quotient range at four times normal loudness for the experimental group. Median values for speed quotient at the other levels of loudness were similar, though the lowest median value occurred at two times normal loudness. Experimental group averages for speed quotient (see Table 9) reveal a visual trend for speed quotient to increase with loudness, except at the two times normal loudness level (M = 0.67, .5X Loud; M = 0.73, Normal Loud; M = 0.55, 2X Loud; M = 1.10, 4X Loud).

Overall, the control group showed more variability in speed quotient than the experimental group. However, both groups had the largest range of speed quotients and performed very similarly at four times normal loudness. Moreover, the control group used higher speed quotients at half normal loudness and two times normal loudness, whereas the experimental group had a higher speed quotient at normal loudness.

Comparisons of matched participants can be made from examining the lower two panels in Figure 21. All participants increased or maintained a similar speed quotient from normal loudness to half normal loudness, with the exception of M0801EL.1 (M = 0.95, .5X Loud; M = 1.26, Normal Loud; M = 0.80, 2X Loud; M = 1.80, 4X Loud), who decreased speed quotient from normal loudness to half normal loudness. Furthermore, both groups of participants indicated the greatest increase in speed quotient at four times normal loudness, with the exception of M0801CL.1 (M = 1.22, .5X Loud; M = 1.10, Normal Loud; M =

1.43, 2X Loud; *M* = 1.38, 4X Loud), F1201CL.1 (*M* = 0.44, .5X Loud; *M* = 0.24, Normal Loud; *M* = 0.32, 2X Loud; *M* = 0.31, 4X Loud), and M1201EL.1 (*M* = 0.63, .5X Loud; *M* = 0.56, Normal Loud; *M* = 0.47, 2X Loud; *M* = 0.44, 4X Loud) (See Appendix L-1 for individual means and standard deviations).

Fundamental Frequency (Hz) – Maximum Sustained Phonation

Hypotheses:

14. a) In typical children, F_0 will increase from Normal to 2X loud maximum phonation productions.

    b) In children with cerebral palsy, F_0 will increase from Normal to 2X loud maximum phonation productions.

As can be seen in the top left panel of Figure 22, the box plot indicates a higher median value and a larger range for F_0 when children in the control group

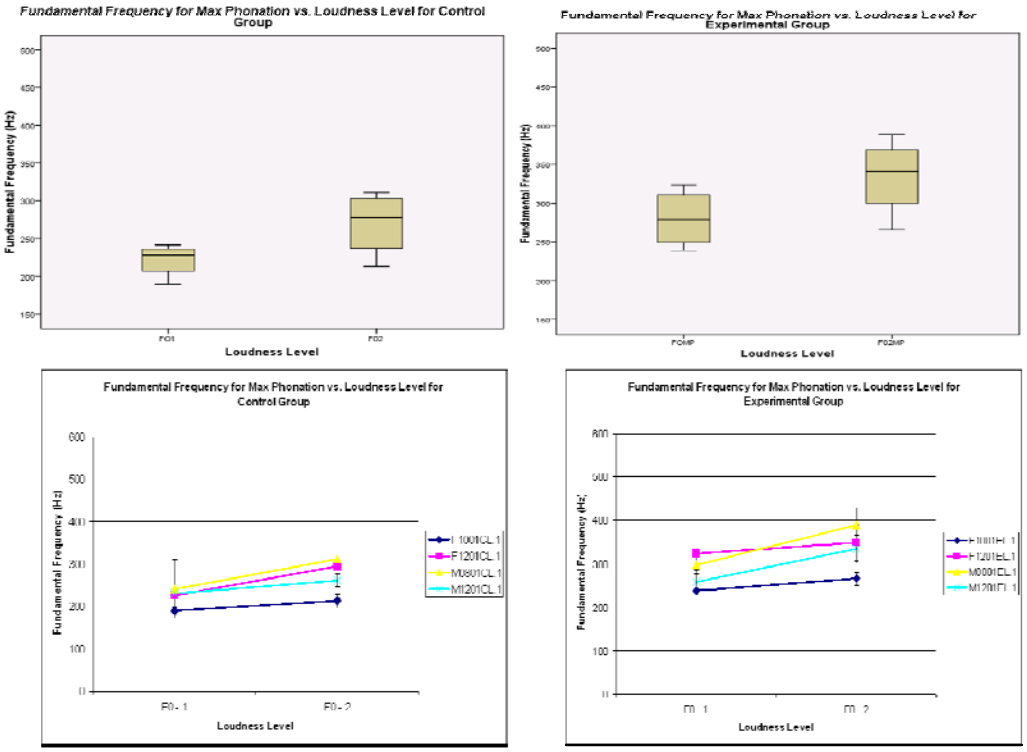

Figure 22. Group and individual data for F0 (in Hz) for maximum phonation tasks. Four panels represent group and individual data. Data from the control group (typically developing children) are shown in the two panels on the left and data for the experimental group (children with cerebral palsy) are shown in the two panels on the right. In all four panels, F0 is depicted on the y-axis in Hz. In all four panels the loudness condition (F01 = Normal Loudness and F02 = 2X Normal Loudness) is depicted on the x-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation F0 values for individual participants. These matched pairs are displayed in the same color.

were instructed to produce maximum phonations at twice normal loudness. Control group averages for F_0 (see Table 8) show an average increase in F_0 (M = 221.61 Hz, Normal Loud; M = 269.89 Hz, 2X Loud). A visual trend towards increasing F_0 with loudness was noted.

As can be seen in the top right panel of Figure 22, the box plot indicates a higher median value and a larger range of F_0 when children with CP were instructed to produce maximum phonations at twice normal loudness. Experimental group averages for F_0 (see Table 8) show an increase in F_0 for the louder condition (M = 280.00 Hz, Normal Loud; M = 334.46 Hz, 2X Loud).

Overall, the experimental group used a wider range of F_0 and on average, produced maximum phonation tasks at higher F_0 than typically developing children for both loudness conditions. However, both groups increased F_0 when producing phonations at twice normal loudness.

Comparisons of matched participants can be made from examining the lower two panels in Figure 22. All participants showed an increase in F_0 from normal loudness to twice normal loudness. Moreover, the experimental group used higher F_0 than the control group in both loudness conditions (See Appendix K for individual means and standard deviations). No exceptions were noted.

Fundamental Frequency (Hz) for "I" – Sentences

Hypotheses:

15. a) In typical children, F_0 for "I" will increase from .5X Loud to 4X loud sentence productions.

    b) In children with cerebral palsy, F_0 for "I" will increase from .5X Loud to 4X loud sentence productions.

As can be seen in the top left panel of Figure 23, the box plot indicates a larger range for F_0 for "I" in the sentence, "**I** sell a sapapple again," when children were instructed to produce the sentence at twice normal loudness and four times

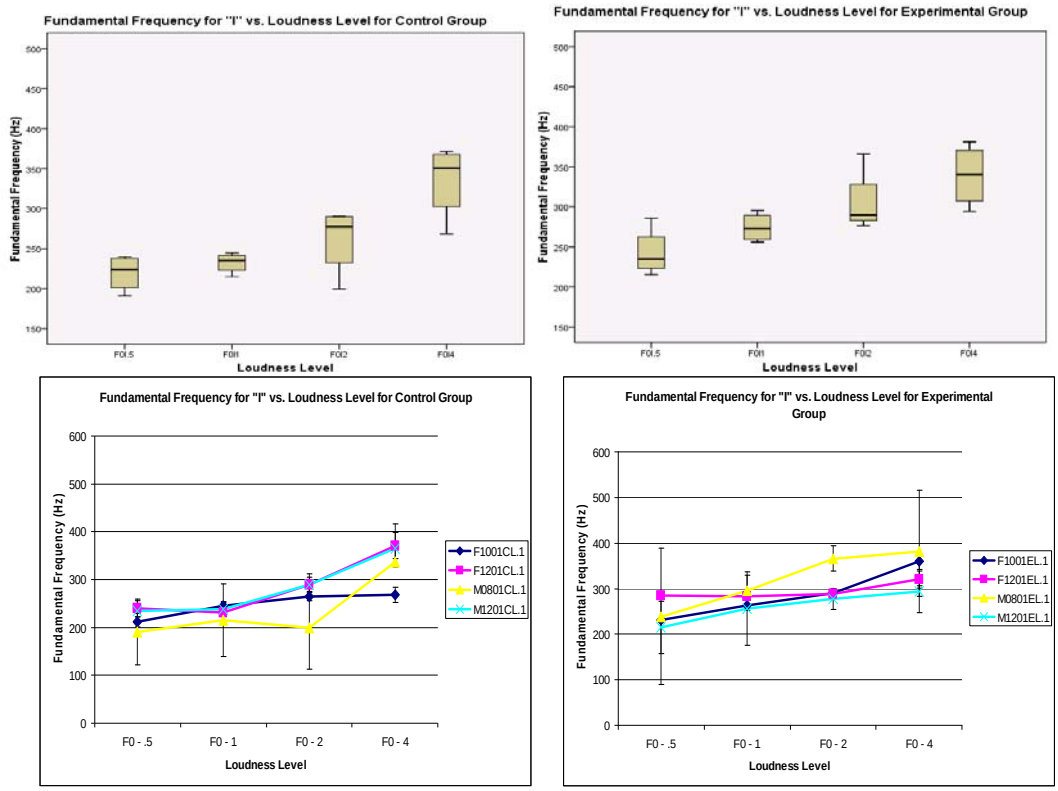

Figure 23. Group and individual data for F0 (in Hz) for "I" in sentence tasks. Four panels represent group and individual data. Data from the control group (typically developing children) are shown in the two panels on the left and data for the experimental group (children with cerebral palsy) are shown in the two panels on the right. In all four panels, F0 is depicted on the y-axis in Hz. In all four panels the loudness condition (F0.5 = .5X Loud, F01 = Normal Loudness, F02= 2X Normal Loudness, and F04 = 4X Normal Loudness) is depicted on the x-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation F0 fvalues for individual participants. These matched pairs are displayed in the same color.

normal loudness. Control group averages and medians for F_0 for "I" (see Table 9) show an average increase in F_0 with an increase in loudness (*M* = 219.53 Hz, .5X Loud; *M* = 232.35 Hz, Normal Loud; *M* = 261.11 Hz, 2X Loud; *M* = 335.31 Hz, 4X Loud). A visual trend towards increasing F_0 for "I" with loudness was noted.

As can be seen in the top right panel of Figure 23, the box plot indicates a larger range of F_0 for "I" when children with CP were instructed to produce the sentence, "I sell a sapapple again," at twice normal loudness and four times normal loudness. Experimental group averages for F_0 for "I" (see Table 9) show an increase in F_0 with an increase in loudness (*M* = 242.75 Hz, .5X Loud; *M* = 274.34 Hz, Normal Loud; *M* = 305.53 Hz, 2X Loud; *M* = 338.98 Hz).

Overall, the experimental group, on average, produced sentence tasks at a higher F_0 than typically developing children, for all loudness conditions. However, the pattern of change for F_0 across conditions was similar between the two groups.

Comparisons of matched participants can be made from examining the lower two panels in Figure 23. Study participants demonstrated a linear increase in F_0 for "I" as the loudness level increased, with the exception of F1201CL.1 (*M* = 239.69 Hz, .5X Loud; *M* = 231.18 Hz, Normal Loud; *M* = 290.17 Hz, 2X Loud; *M* = 371.20 Hz, 4X Loud) and her match F1201EL.1 (*M* = 285.60 Hz, .5X Loud; *M* = 282.94 Hz, Normal Loud; *M* = 288.62 Hz, 2X Loud; *M* = 320.24 Hz, 4X Loud). These two participants produced a higher F_0 at half normal loudness compared to normal loudness. Control participants produced a higher F_0 for "I" than experimental participants, excluding M1201CL.1 (*M* = 235.46 Hz, .5X

Loud; *M* = 238.85 Hz, Normal Loud; *M* = 289.64 Hz, 2X Loud; *M* = 364.68 Hz, 4X Loud) who produced a higher F_0 than his partner with CP, M1201EL.1(*M* = 215.417 Hz, .5X Loud; *M* = 256.01 Hz, Normal Loud; *M* = 276.73 Hz, 2X Loud; *M* = 294.61 Hz, 4X Loud), except at normal loudness (See Appendix L-2 for individual means and standard deviations).

Fundamental Frequency (Hz) for s"a"p – Sentences

Hypotheses:

16. a) In typical children, F_0 for s "a" p will increase from .5X Loud to 4X loud sentence productions.

    b) In children with cerebral palsy, F_0 for s "a" p will increase from .5X Loud to 4X loud sentence productions.

As can be seen in the top left panel of Figure 24, the box plot indicates a larger range for F_0 for s "a" p in the sentence, "I sell a s**a**papple again," when children were instructed to produce the sentence at increasing levels of loudness.

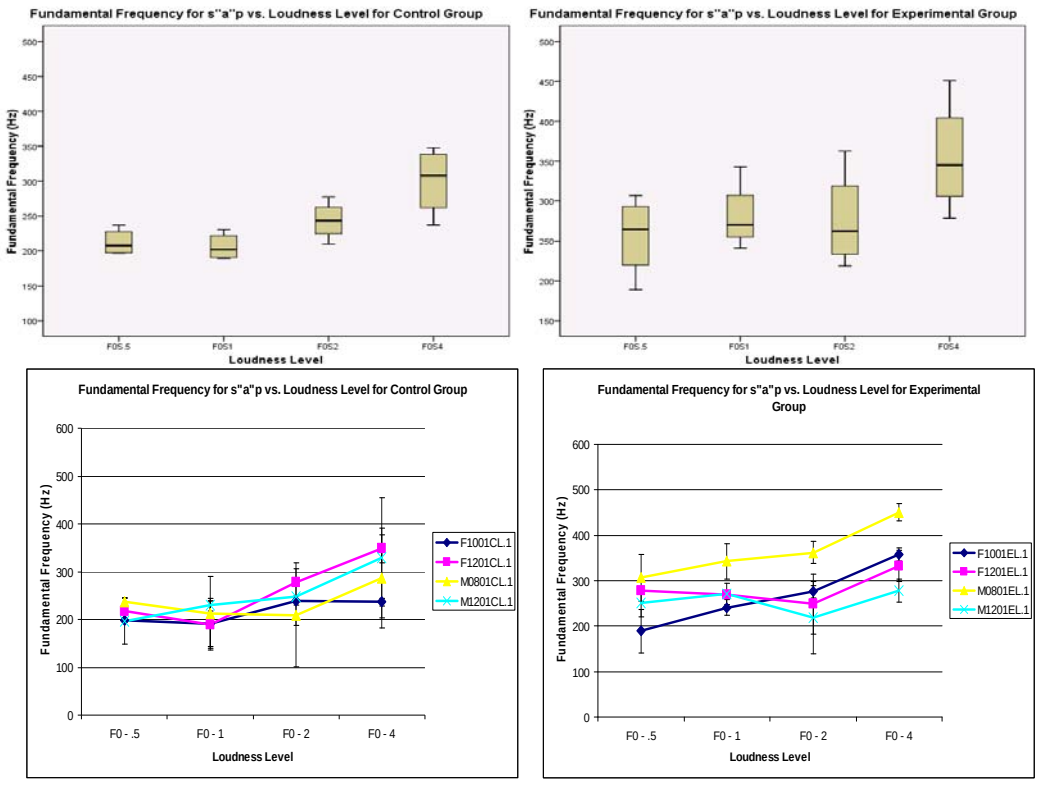

Figure 24. Group and individual data for F0 (in Hz) for s "a" p in sentence tasks. Four panels represent group and individual data. Data from the control group (typically developing children) are shown in the two panels on the left and data for the experimental group (children with CP) are shown in the two panels on the right. In all four panels, F0 is depicted on the y-axis in Hz. In all four panels the loudness condition (F0.5 = .5X Loud, F01 = Normal Loudness, F02= 2X Normal Loudness, and F04 = 4X Normal Loudness) is depicted on the x-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation F0 values for individual participants. These matched pairs are displayed in the same color.

Further, the box plot illustrates the highest median value at four times normal loudness, followed by two times normal loudness, and then half normal loudness. Control group averages for F_0 for s "a" p (see Table 9) show an average increase in F_0 from normal loudness to half normal loudness and from normal loudness to four times normal loudness (*M* = 212.34, .5X Loud; *M* = 206.09 Hz, Normal Loud; *M* = 243.46 Hz, 2X Loud; *M* = 307.86 Hz, 4X Loud). A visual trend towards increasing F_0 for s "a" p with loudness was noted.

As can be seen in the top right panel of Figure 24, the box plot demonstrates an increasing range of fundamental frequencies for s “a” p when children with CP were instructed to produce the sentence, “I sell a sapapple again,” at twice normal loudness and four times normal loudness. Further, the box plot illustrates the highest median value occurred at four times normal loudness, followed by normal loudness, half normal loudness, and finally two times normal loudness. Experimental group averages for F_0 for s “a” p (see Table 9) show an increase in F_0 from half normal loudness to normal loudness. A decrease in F_0 occurs at two times normal loudness. The largest average F_0 occured at four times normal loudness (*M* = 256.36 Hz, .5X Loud; *M* = 281.04 Hz, Normal Loud; *M* = 276.29 Hz, 2X Loud; *M* = 354.99 Hz).

Overall, the experimental group, on average, produced sentence tasks at a higher F_0 and with a larger range than typically developing children, across loudness conditions. However, both groups exhibited a similar pattern of F_0 change especially when producing sentences at four times normal loudness.

Comparisons of matched participants can be made from examining the lower two panels in Figure 24. Control participants showed an increase in F_0 for s “a”p from normal loudness to half normal loudness, with the exception of M1201CL.1 (*M* = 197.06 Hz, .5X Loud; *M* = 230.87 Hz, Normal Loud; *M* = 246.92 Hz, 2X Loud; *M* = 329.29 Hz, 4X Loud). F1201EL.1 also demonstrated an increase in F_0 for s “a”p from normal loudness to half normal loudness. M1201CL.1, M0801EL.1, and F1001EL.1 showed a linear increase in F_0 for s “a” p as loudness increased. M0801CL.1, F1201EL.1, and M1201EL.1 tended to

decrease F_0 from normal loudness to twice normal loudness. All participants indicated their highest F_0 for s “a”p at four times normal loudness, with the exception of F1001CL.1 (*M* = 197.48 Hz, .5X Loud; *M* = 190.90 Hz, Normal Loud; *M* = 239.81 Hz, 2X Loud; *M* = 236.82 Hz, 4X Loud) (See Appendix L-2 for individual means and standard deviations).

Fundamental Frequency (semitones) – Max Phonation

Hypotheses:

17. a) In typical children, semitone values will increase from Normal to 2X loud maximum phonation productions.

    b) In children with cerebral palsy, semitone values will increase from Normal to 2X loud maximum phonation productions.

As can be seen in the top left panel of Figure 25, the box plot indicates a higher median value and an increase in semitones when children were instructed to produce maximum phonations at twice normal loudness. Control group

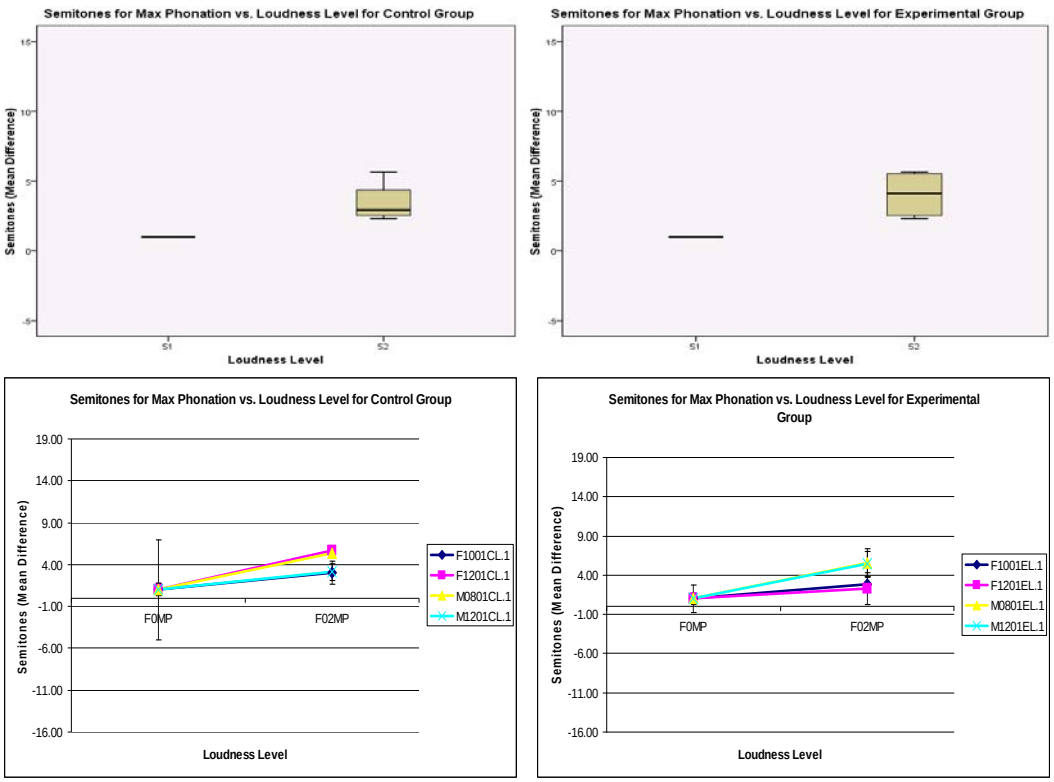

Figure 25. Group and individual data for semitone values for maximum phonation tasks, relative to a semitone value of 1 at normal loudness. Four panels represent group and individual data. Data from the control group (typically developing children) are shown in the two panels on the left and data for the experimental group (children with CP) are shown in the two panels on the right. In all four panels, semitone values are depicted on the y-axis. In all four panels loudness condition (S1 (F0MP) = Normal Loudness and S2 (F02MP) = 2X Normal Loudness) is depicted on the x-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation semitone values for individual participants. These matched pairs are displayed in the same color.

averages for semitones (see Table 8) show an average increase in semitone values (M = 1.00 semitones, Normal Loud; M = 3.46 semitones, 2X Loud). A visual trend towards increasing semitone values with loudness was noted.

As can be seen in the top right panel of Figure 25, the box plot indicates a higher median value and an increase in semitone values when children with CP were instructed to produce maximum phonations at twice normal loudness. Experimental group averages for semitones (see Table 8) show an increase in semitone values for the louder condition (M = 1.00 semitones, Normal Loud; M =

4.05 semitones, 2X Loud). Overall, the experimental group used a wider range of semitone values and on average, produced maximum phonation tasks at higher semitone values (larger adjustment from normal loudness to two times normal loudness) than typically developing children for both loudness conditions.

Comparisons of matched participants can be made from examining the lower two panels in Figure 25. F1001CL.1 and F1201CL.1 produced phonations at higher semitone values than their partners with CP. M0801CL.1 produced phonations at similar semitone values as his match M0801EL.1. M1201CL.1 produced phonations at lower semitone values than his match M1201EL.1 (See Appendix K for individual means and standard deviations).

Fundamental Frequency (semitones) for "I" – Sentences

Hypotheses:

18. a) In typical children, semitone values for "I" will increase from .5X Loud to 4X loud sentence productions.

    b) In children with cerebral palsy, semitone values for "I" will increase from .5X Loud to 4X loud sentence productions.

As can be seen in the top left panel of Figure 26, the box plot indicates a larger range for semitone values for "I" in the sentence, "**I** sell a sapapple again,"

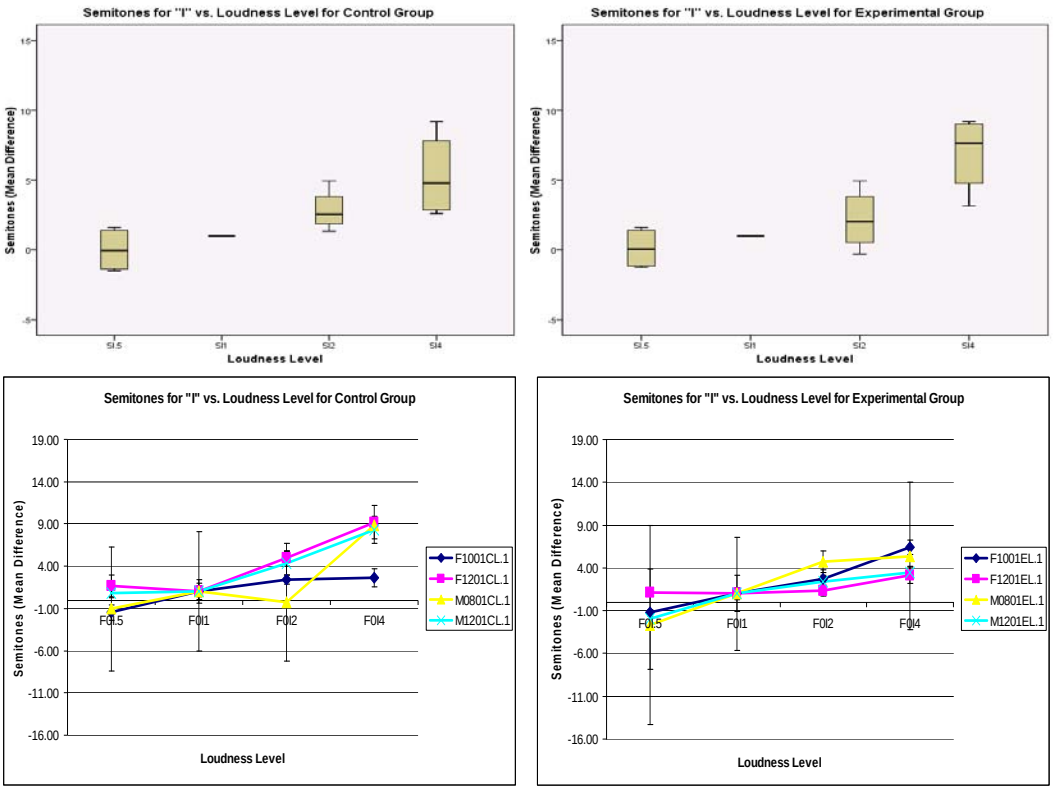

Figure 26. Group and individual data for semitone values, relative to a semitone value of 1 at normal loudness, for "I" in sentence tasks. Four panels represent group and individual data. Data from the control group (typically developing children) are shown in the two panels on the left and data for the experimental group (children with CP) are shown in the two panels on the right. In all four panels, semitone values are depicted on the y-axis. In all four panels the loudness condition (SI.5 (F0I.5) = .5X Loud, SI1 (F0I1) = Normal Loudness, SI2 (F0I2)= 2X Normal Loudness, and SI4 (F0I4) = 4X Normal Loudness) is depicted on the x-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation semitone values for "I" for individual participants. These matched pairs are displayed in the same color.

when children were instructed to produce the sentence at four times normal loudness, compared to twice normal loudness and half normal loudness. Control group averages and medians for semitone values for "I" (see Table 9) show an average increase in semitone values with an increase in loudness (*M* = 0.02 semitones, .5X Loud; *M* = 1.00 semitones, Normal Loud; *M* = 2.85 semitones, 2X Loud; *M* = 5.35 semitones, 4X Loud). A visual trend towards increasing the semitone values for "I" with loudness was noted.

As can be seen in the top right panel of Figure 26, the box plot indicates a higher median for "I" when children with CP were instructed to produce the sentence, "I sell a sapapple again," at increasing levels of loudness. Experimental group averages for semitone values for "I" (see Table 9) show an increase in semitone values with an increase in loudness (*M* = 0.13 semitones, .5X Loud; *M* = 1.00 semitones, Normal Loud; *M* = 2.18 semitones, 2X Loud; *M* = 6.90 semitones). Overall, the experimental group, on average, produced sentence tasks for four times normal loud at higher semitone values than typically developing children.

Comparisons of matched participants can be made from examining the lower two panels in Figure 26. Study participants tended to show a linear increase in semitone values as loudness increased, with the exception of F1201CL.1 (*M* = 1.63 semitones, .5X Loud; *M* = 1.00 semitones, Normal Loud; *M* = 4.93 semitones, 2X Loud; *M* = 9.20 semitones, 4X Loud), F1201EL.1 (*M* = 1.16 semitones, .5X Loud; *M* = 1.00 semitones, Normal Loud; *M* = 1.34 semitones, 2X Loud; *M* = 3.14 semitones, 4X Loud), and M0801CL.1 (*M* = -1.04 semitones, .5X Loud; *M* = 1.00 semitones, Normal Loud; *M* = -0.29 semitones, 2X Loud; *M* = 8.80 semitones, 4X Loud). Overall, the typically developing children produced higher semitone values than the children with CP, with the exception of F1001CL.1 who produced sentences at lower semitone values (*M* = -1.47 semitones, .5X Loud; *M* = 1.00 semitones, Normal Loud; *M* = 2.40 semitones, 2X Loud; *M* = 2.61 semitones, 4X Loud) than her match F1001EL.1 (*M* = -1.24 semitones, .5X Loud; *M* = 1.00 semitones, Normal Loud; *M* = 2.74 semitones, 2X

Loud; *M* = 6.44 semitones, 4X Loud). Moreover, M0801CL.1 (*M* = -1.04 semitones, .5X Loud; *M* = 1.00 semitones, Normal Loud; *M* = -0.29 semitones, 2X Loud; *M* = 8.80 semitones, 4X Loud) produced lower F_0 (in semitones) at twice normal loudness compared to his match M0801EL.1 (*M* = -2.67 semitones, .5X Loud; *M* = 1.00 semitones, Normal Loud; *M* = 4.71 semitones, 2X Loud; *M* = 5.41 semitones, 4X Loud) (See Appendix L-2 for individual means and standard deviations).

Fundamental Frequency (semitones) for s "a" p – Sentences

Hypotheses:

19. a) In typical children, semitone values for s "a" p will increase from .5X Loud to 4X loud sentence productions.

    b) In children with cerebral palsy, semitone values for s "a" p will increase from .5X Loud to 4X loud sentence productions.

As can be seen in the top left panel of Figure 27, the box plot indicates a higher median value for semitone values for s "a" p in the sentence "I sell a

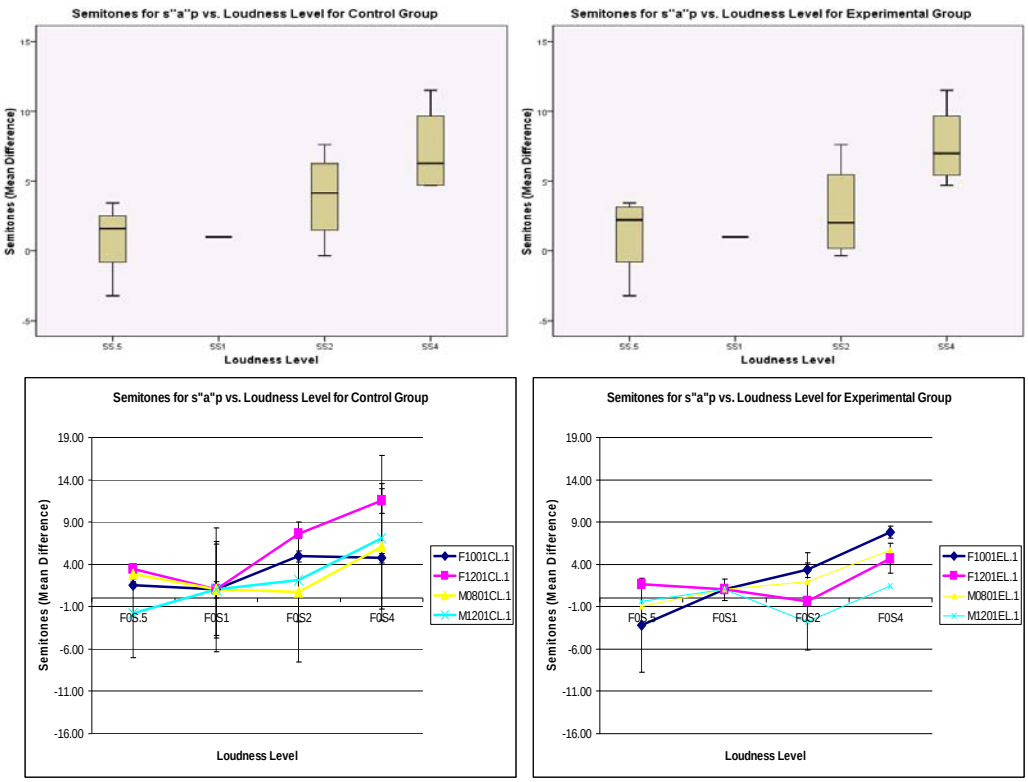

Figure 27. Group and individual data for semitone values, relative to a semitone value of 1 at normal loudness, for s "a" p in sentence tasks. Four panels represent group and individual data. Data from the control group (typically developing children) are shown in the two panels on the left and data for the experimental group (children with cerebral palsy) are shown in the two panels on the right. In all four panels, semitone values are depicted on the y-axis. In all four panels the loudness condition (SS.5 (F0I.5) = .5X Loud, SS1 (F0I1) = Normal Loudness, SS2 (F0I2)= 2X Normal Loudness, and SS4 (F0I4) = 4X Normal Loudness) is depicted on the x-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation semitone values for s "a" p for individual participants. These matched pairs are displayed in the same color.

s**a**papple again," when children were instructed to produce the sentence at two times normal loudness and four times normal loudness. Control group averages for semitone values produced for s "a" p (see Table 9) show an average increase in semitone values with an increase in loudness. A visual trend towards increasing semitone values for s "a" p with loudness was noted (*M* = 0.87 semitones, .5X Loud; *M* = 1.00 semitones, Normal Loud; *M* = 3.88 semitones, 2X Loud; *M* = 7.19 semitones, 4X Loud).

As can be seen in the top right panel of Figure 22, the box plot indicates the highest median value for semitone values for s “a” p occurred when children with CP were instructed to produce sentence, “I sell a s**a**papple again,” at four times normal loudness and two times normal loudness respectively. Experimental group averages for semitone values for s “a” p (see Table 9) show an increase in semitone values from normal loudness to half normal loudness and from normal loudness to four times normal loudness (*M* = 1.18 semitones, .5X Loud; *M* = 1.00 semitones, Normal Loud; *M* = 2.83 semitones, 2X Loud; *M* = 7.54 semitones).

Overall, the experimental group and the control group performed similarly. That being said, the typically developing children demonstrated a more linear increase in semitone values with loudness. Further the control group showed a larger adjustment in semitone values from normal loudness to two times normal loudness than the experimental group. However, the experimental group showed a larger adjustment in semitone values from normal loudness to four times normal loudness.

Comparisons of matched participants can be made from examining the lower two panels in Figure 27. Control participants showed an increase in semitone values for s “a”p from normal loudness to half normal loudness, with the exception of M1201CL.1 (*M* = -1.74 semitones, .5X Loud; *M* = 1.00 semitones, Normal Loud; *M* = 2.16 semitones, 2X Loud; *M* = 7.15 semitones, 4X Loud). F1201EL.1 also demonstrated an increase in semitone values for s “a”p from normal loudness to half normal loudness. M1201CL.1, M0801EL.1, and F1001EL.1 showed a linear increase in semitone values for s “a” p as loudness

increased. M0801CL.1, F1201EL.1, and M1201EL.1 tended to decrease semitone values from normal loudness to twice normal loudness. All participants indicated their highest semitone values for s “a”p at four times normal loudness, with the exception of F1001CL.1 (*M* = 1.59 semitones, .5X Loud; *M* = 1.00 semitones, Normal Loud; *M* = 4.95 semitones, 2X Loud; *M* = 4.73 semitones, 4X Loud). Control participants used higher semitone values compared to experimental participants, excluding F1001CL.1 at four times normal loudness and M0801CL.1 at twice normal loudness (See Appendix L-2 for individual means and standard deviations).

Area of Mouth Opening – Max Phonation

Hypotheses:

20. a) In typical children, MA will increase from Normal to 2X loud maximum phonation productions.

    b) In children with cerebral palsy, MA will not change from Normal to 2X loud maximum phonation productions.

As can be seen in the top left panel of Figure 28, the box plot indicates a lower median value when children in the control group were instructed to

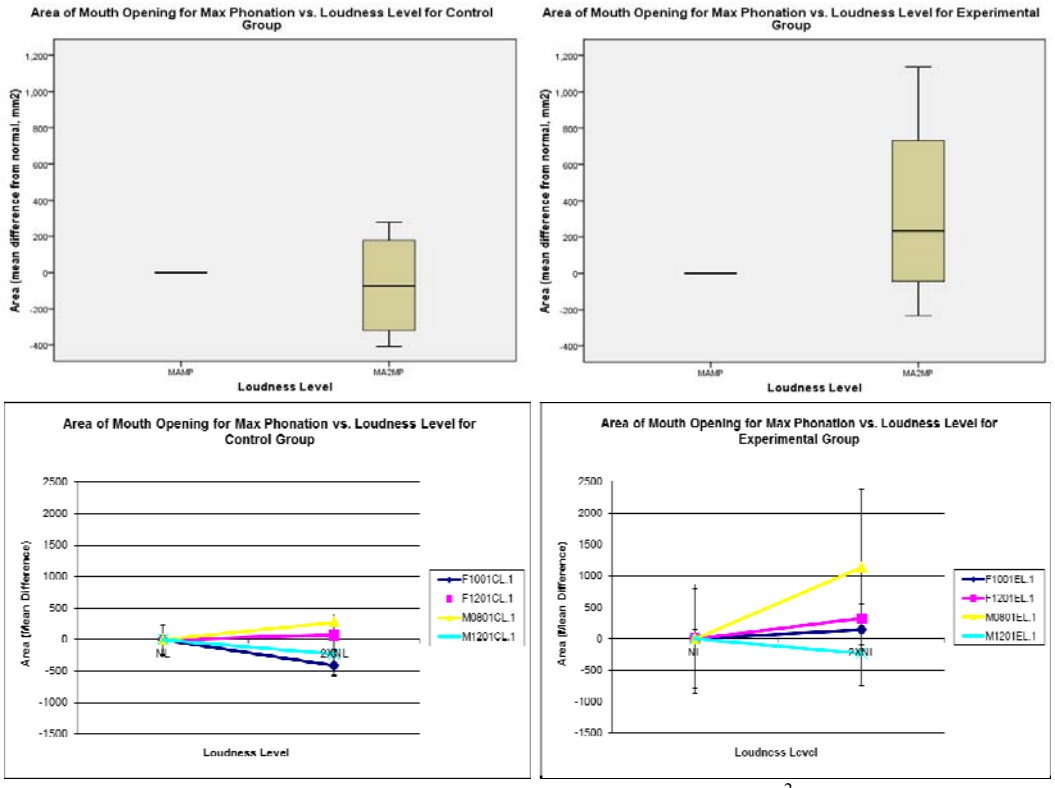

Figure 28. Group and individual data for area of mouth opening (in mm^2), calculated as a mean difference score (e.g., area derived from normal loudness condition – area derived from experimental conditions), for maximum phonation tasks. Mean difference scores were inverted to allow intuitive interpretation of the figure. Four panels represent group and individual data. Data from the control group (typically developing children) are shown in the two panels on the left and data for the experimental group (children with CP) are shown in the two panels on the right. In all four panels, area of mouth opening is depicted on the y-axis in mm2 (mean difference score). Positive values represent an increase in area of mouth opening and negative values represent a decrease in area of mouth opening. In all four panels, loudness condition (MA1 (NL) = Normal Loudness and MA2 (2XNL) = 2X Normal Loudness) is depicted on the x-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation area of mouth opening values for individual participants. These matched pairs are displayed in the same color.

produce maximum phonations at twice normal loudness. Control group averages for area of mouth opening (see Table 8) show an average decrease in area of mouth opening (M = 0.00 mm^2, Normal Loud; M = 69.69 mm^2, 2X Loud). A visual trend towards a slight decrease in area of mouth opening with loudness was noted. Be reminded that a positive number indicates smaller area and a negative number represents a larger area based on the calculations away from 0mm^2 derived from the normal loudness conditions.

As can be seen in the top right panel of Figure 28, children with CP produced maximum phonation tasks at normal and two times normal loudness, across a large range of mouth opening area at twice normal loudness. The box plot indicates a higher median value and a larger range of mouth opening area when children with CP were instructed to produce maximum phonations at twice normal loudness. Experimental group averages for area of mouth opening (see Table 8) show an increase in area of mouth opening for the louder condition (M = 0.00 mm^2, Normal Loud; M = -342.62 mm^2, 2X Loud).

Overall, the experimental group used a wider range of mouth opening area and on average, produced maximum phonation tasks using a larger area of mouth opening at twice normal loudness, than typically developing children. Absolute measures of mouth opening indicated that on average children with CP had a greater area of mouth opening at Normal Loud than the typically developing children (M = 1918 mm^2 and 1397 mm^2, respectively).

Comparisons of matched participants can be made from examining the lower two panels in Figure 28. Study participants increased area of mouth opening from normal loudness to twice normal loudness, with the exception of F1001CL.1, M1201CL.1, and M1201EL.1 who all decreased area of mouth opening in the louder condition. F1201CL.1 and M0801CL.1 increased area of mouth opening to a much lesser extent than their counterparts with CP (See Appendix M for individual means and standard deviations).

Area of Mouth Opening – Sentences

Hypotheses:

21. a) In typical children, area of mouth opening will increase from .5X Loud to 4X loud sentence productions.

    b) In children with cerebral palsy, area of mouth opening will not change from .5X Loud to 4X loud sentence productions.

As can be seen in the top left panel of Figure 29, the box plot indicates a lower median value when children in the control group were instructed to

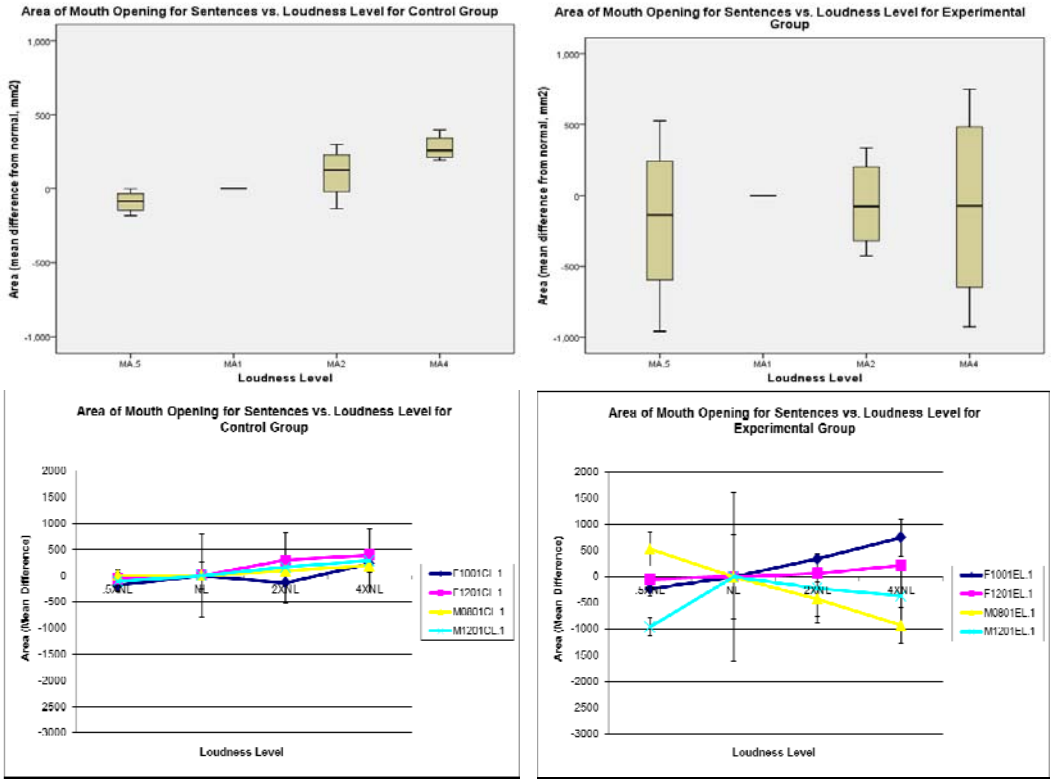

Figure 29. Group and individual data for area of mouth opening (in mm^2), calculated as a mean difference score (e.g., area derived from normal loudness condition – area derived from experimental conditions), for sentence tasks. Mean difference scores were inverted to allow intuitive interpretation of the figure. Four panels represent group and individual data. Data from the control group (typically developing children) are shown in the two panels on the left and data for the experimental group (children with CP) are shown in the two panels on the right. In all four panels, area of mouth opening is depicted on the y-axis in mm2. Positive values represent an increase in area of mouth opening and negative values represent a decrease in area of mouth opening. In all four panels, loudness condition (MA.5 (.5XNL) = .5X Loud, MA1 (NL) = Normal Loudness, MA2 (2XNL) = 2X Normal Loudness, and MA4 (4XNL) = 4X Normal Loudness) is depicted on the x-axis. The top two panels represent box and whisker plots, indicating the median, upper and lower quartiles and inner fences for each group. The bottom two panels show the average and standard deviation area of mouth opening values for individual participants. These matched pairs are displayed in the same color.

produce the sentence at increasing levels of loudness. The largest range of mouth area opening occurred at twice normal loudness. Control group averages for area of mouth opening (see Table 9) show an average increase in area of mouth opening (M = 88.96 mm^2, .5X Loud; M = 0.00 mm^2, Normal Loud; M = -104.80 mm^2, 2X Loud; M = -276.74 mm^2, 4X Loud). A visual trend towards increasing area of mouth opening with loudness was noted.

As can be seen in the top right panel of Figure 29, children with CP produced sentences at half normal loudness, normal, two times normal loudness and four times loudness, across a large range of mouth opening area. The box plot indicates the lowest median value occurred when children with CP were instructed to produce sentences at normal loudness. The median values for two times normal loudness and four times normal loudness were very similar. The largest median value occurred at half normal loudness. The largest range of area of mouth opening values occurred at half normal loudness and four times normal loudness. Experimental group averages for area of mouth opening (see Table 9) show an increase in area of mouth opening from half normal loudness to normal loudness and from twice normal loudness to four times normal loudness, with normal loudness having the greatest area of mouth opening (M = 176.31 mm^2, .5X Loud; M = 0.00 mm^2, Normal Loud; M = 59.82 mm^2, 2X Loud; M = 80.76 mm^2, 4X Loud).

Overall, the control group, on average, produced sentences with less variability in area of mouth opening. Absolute measures of area indicated that typically developing children, on average, had a smaller area of mouth opening than the children with CP (M =1253 mm^2 and 2139 mm^2, respectively). Further typically developing children demonstrated a linear increase in area of mouth opening as loudness increased.

Comparisons of matched participants can be made from examining the lower two panels in Figure 29. Overall, a linear increase in area of mouth opening was observed in study participants, with the exception of M0801EL.1 and

M1201EL.1. M0801EL.1 demonstrated a linear decrease in area of mouth opening as loudness increased (M = -526.55 mm^2, .5X Loud; M = 0.00 mm^2, Normal Loud; M = 424.93 mm^2, 2X Loud; M = 925.40 mm^2, 4X Loud) and M1201EL.1 indicated an increase in area of mouth opening from half normal loudness to normal loudness and then a decrease in area of mouth opening from normal loudness to four times normal loudness (M = 957.97 mm^2, .5X Loud; M = 0.00 mm^2, Normal Loud; M = 214.5 mm^2, 2X Loud; M = 365.74 mm^2, 4X Loud). Another exception was noted in F1001CL.1 because she indicated a small decrease in area of mouth opening from normal loudness to twice normal loudness. F1001CL.1 produced sentences with less adjustment in area of mouth opening for increased loudness (M = 181.78 mm^2, .5X Loud; M = 0.00 mm^2, Normal Loud; M = 134.53 mm^2, 2X Loud; M = -230.82 mm^2, 4X Loud) compared to her match, F1001EL.1 (M = 232.05 mm^2, .5X Loud; M = 0.00 mm^2, Normal Loud; M = -337.73 mm^2, 2X Loud; M = -748.05 mm^2, 4X Loud), whereas F1201CL.1 produced sentences with a greater adjustment in area of mouth opening for increased loudness (M = 63.93 mm^2, .5X Loud; M = 0.00 mm^2, Normal Loud; M = -299.31 mm^2, 2X Loud; M = -396.82 mm^2, 4X Loud) compared to her match, F1201EL.1 (M = 41.77 mm^2, .5X Loud; M = 0.00 mm^2, Normal Loud; M = -62.4 mm^2, 2X Loud; M = -220.07 mm^2, 4X Loud) (See Appendix N for individual means and standard deviations).

Statistical Analyses

Exploratory statistics were performed for the purpose of seeking corroboration with the observations derived from visual trend analyses. Due to the

small number of participants in this study, it is important to exercise extreme caution when interpreting the results of these statistical analyses. Due to the exploratory nature of this study, a *familywise* error rate was not applied to the post-hoc statistical analyses. Any changes in the values of interest are believed to be of importance to the investigative nature of this study. A more conservative *p*-value may miss crucial changes in these values. The Multiple Analysis of Variance (MANOVA) will be discussed, followed by the one-way within subjects, Analysis of Variance (ANOVA) statistics.

MANOVA

A MANOVA was conducted to determine whether any statistically significant or statistically significant trends (e.g.,. $p = 0.05$ to $p = 0.085$) in group differences existed between the typically developing children and the children with cerebral palsy. All dependent variables were included in the full model along with the two grouping variables (control and experimental).

Table 10. Statistically significant between group effects. Left – Right: The first column indicates the type of task performed; the second column indicates the loudness condition of the task; the third column shows the dependent variable; and, the fourth column reveals the statistic.

Task	Variable	Condition	Statistic
Sentence	Intercostal Muscle Activity	.5X Loud	*$F(1,6) = 6.712$, $p < 0.05$
Sentence	Intercostal Muscle Activity	Normal	$F(1,6) = 5.543$, $p = 0.057$
Sentence	Intercostal Muscle Activity	2X Loud	*$F(1,6) = 18.807$, $p \leq 0.005$
Sentence	Intercostal Muscle Activity	4X Loud	*$F(1,6) = 10.803$, $p < 0.05$
Max Phonation	Fundamental Frequency	Normal	*$F(1,6) = 7.011$, $p< 0.05$
Sentence	Fundamental Frequency for "I"	Normal	*$F(1,6) = 14.214$, $p < 0.01$
Sentence	Fundamental Frequency for s "a" p	Normal	*$F(1,6) = 9.815$, $p < 0.05$

*** represents a statistically significant group effect**

The significant group effects for the dependent variables: (1) intercostal muscle activity and (2) fundamental frequency, support the visual differences that

were noted between typically developing children and children with cerebral palsy.

ANOVA

One-way within-subjects ANOVAs were conducted for each dependent variable for each group, across four loudness levels, in order to determine if each group performed differently from one loudness condition to the next. Table 11 reveals the statistically significant or a statistically significant trend (e.g., $p = 0.05$ to $p = 0.085$) within subjects effects for the control group.

Table 11. Statistically significant within-subjects effects for the control group. Left – Right: The first column indicates the dependent variable and the second column reveals the statistic.

Control Group

Variable	Statistic
Lung Volume Initiation (cm^3)	F (5,15) = 2.430, p = 0.084
Lung Volume Termination (cm^3)	*F(5,15) = 5.467, $p \leq 0.005$
Lung Volume Excursion (% VC)	*F(5,15) = 3.292, $p < 0.05$
Intercostal Muscle Activity (% MVE)	*F(5,15) = 40.851, $p < 0.001$
Oblique Muscle Activity (% MVE)	*F(5,15) = 6.914, $p < 0.005$
Fundamental Frequency (Hz)	*F(9,27) = 5.366, $p < 0.001$
Fundamental Frequency (semitones)	*F(9,27) = 7.984, $p < 0.001$
Area of Mouth Opening (mm^2)	*F(3,9) = 4.022, $p < 0.05$

*** represents a statistically significant within-subjects effect**

The statistically significant within-subjects effects for the typically developing children supported the differences observed in the visual trend analyses for lung volume initiation, lung volume termination, lung volume excursion, intercostal muscle activity, oblique muscle activity, fundamental frequency (in Hz and semitones), and area of mouth opening. A visual trend for speed quotient to increase was noted during the following tasks: (1) sustained maximum phonation tasks from normal loudness to twice normal loudness, (2) sentence tasks for "I" from normal loudness to half, twice and four times normal loudness, and (3) for s "a"p from half normal loudness to normal, twice, and four

times normal loudness; however, these visual trends were not supported statistically. Table 12 shows the statistically significant (or statistical trends) within-subjects effects for the experimental group.

Table 12. Statistically significant within-subjects effects for the experimental group. Left – Right: The first column indicates the dependent variable and the second column reveals the statistic.

Experimental Group

Variable	**Statistic**
Lung Volume Initiation (cm^3)	$F(5,15) = 2.619$, $p = 0.068$
Lung Volume Excursion (% VC)	$*F(5,15) = 4.403$, $p < 0.05$
Oblique Muscle Activity (% MVE)	$*F(5,15) = 3.342$, $p<0.05$
Fundamental Frequency (Hz)	$*F(9,27) = 8.580$, $p < 0.001$
Fundamental Frequency (semitones)	$*F(9,27) = 7.092$, $p < 0.001$

*** represents a statistically significant within-subjects effect**

The statistically significant (or statistical trend) within-subjects effects for the children with cerebral palsy supported the differences noted in the visual trend analyses for lung volume initiation, lung volume excursion, oblique muscle activity, and fundamental frequency (in Hz and semitones). Although, visual differences within-subjects were noted for lung volume termination, intercostal muscle activity, speed quotient, and area of mouth opening, these differences were not supported statistically.

Table 13 indicates the statistically significant (or statistical trend) pair-wise comparisons for the control group.

Table 13. Statistically significant pair-wise comparisons for the control group. Left – Right: the first column indicates the task performed; the second column shows the dependent variable; the third and fourth columns reveal the significantly different conditions; the fourth column indicates the exploratory statistic; and, the final column indicates the presence of a visual trend .

Control Group

Task	Variable	Condition 1	Condition 2	Statistic	Visual Trend
Max Phonation	Lung Volume Initiation (cm^3)	Normal	2X Loud	NS	Y
Max Phonation	Lung Volume Termination (cm3)	Normal	2X Loud	NS	N
Max Phonation	Lung Volume Excursion (% VC)	Normal	2X Loud	NS	Y
Max Phonation	Intercostal Muscle Activity (%MVE)	Normal	2X Loud	NS	Y
Max Phonation	Oblique Muscle Activity (%MVE)	Normal	2X Loud	NS	Y
Max Phonation	Speed Quotient	Normal	2X Loud	NS	Y
Max Phonation	Fundamental Frequency (Hz)	Normal	2X Loud	*$p < 0.05$	Y
Max Phonation	Semitones	Normal	2X Loud	*$p < 0.05$	Y
Max Phonation	Area of Mouth Opening (mm^2)	Normal	2X Loud	NS	Y
Sentence	Lung Volume Initiation (cm^3)	.5X Loud	Normal	NS	Y
Sentence		.5X Loud	2X Loud	NS	Y
Sentence		.5X Loud	4X Loud	$p = 0.061$	Y
Sentence		Normal	2X Loud	NS	Y
Sentence		Normal	4X Loud	NS	Y
Sentence		2X Loud	4X Loud	NS	Y
Sentence	Lung Volume Termination (cm^3)	.5X Loud	Normal	$p = 0.061$	N
Sentence		.5X Loud	2X Loud	NS	N
Sentence		.5X Loud	4X Loud	NS	N
Sentence		Normal	2X Loud	NS	N
Sentence		Normal	4X Loud	NS	N
Sentence		2X Loud	4X Loud	NS	N
Sentence	Lung Volume Excursion (% VC)	.5X Loud	Normal	NS	Y
Sentence		.5X Loud	2X Loud	*$p < 0.05$	Y
Sentence		.5X Loud	4X Loud	*$p < 0.05$	Y
Sentence		Normal	2X Loud	NS	Y
Sentence		Normal	4X Loud	NS	Y

Sentence		2X Loud	4X Loud	NS	Y
Sentence	Intercostal Muscle Activity (%MVE)	.5X Loud	Normal	NS	Y
Sentence		.5X Loud	2X Loud	NS	Y
Sentence		.5X Loud	4X Loud	$p = 0.055$	Y
Sentence		Normal	2X Loud	NS	N
Sentence		Normal	4X Loud	$p = 0.051$	Y
Sentence		2X Loud	4X Loud	NS	Y
Sentence	Oblique Muscle Activity (%MVE)	.5X Loud	Normal	$*p < 0.01$	Y
Sentence		.5X Loud	2X Loud	$p = 0.052$	Y
Sentence		.5X Loud	4X Loud	NS	Y
Sentence		Normal	2X Loud	NS	Y
Sentence		Normal	4X Loud	NS	Y
Sentence		2X Loud	4X Loud	NS	Y
Sentence	Speed Quotient "I"	.5X Loud	Normal	NS	Y
Sentence		.5X Loud	2X Loud	NS	Y
Sentence		.5X Loud	4X Loud	NS	Y
Sentence		Normal	2X Loud	NS	Y
Sentence		Normal	4X Loud	NS	Y
Sentence		2X Loud	4X Loud	NS	Y
Sentence	Speed Quotient s"a"p	.5X Loud	Normal	$*p < 0.05$	Y
Sentence		.5X Loud	2X Loud	NS	Y
Sentence		.5X Loud	4X Loud	NS	Y
Sentence		Normal	2X Loud	$*p < 0.05$	Y
Sentence		Normal	4X Loud	NS	Y
Sentence		2X Loud	4X Loud	NS	Y
Sentence	Fundamental Frequency (Hz) for "I"	.5X Loud	Normal	NS	Y
Sentence		.5X Loud	2X Loud	$*p < 0.05$	Y
Sentence		.5X Loud	4X Loud	$*p \leq 0.01$	Y
Sentence		Normal	2X Loud	NS	Y
Sentence		Normal	4X Loud	$*p < 0.05$	Y
Sentence		2X Loud	4X Loud	NS	Y

Sentence	Fundamental Frequency (Hz) for s"a"p	.5X Loud	Normal	NS	Y
Sentence		.5X Loud	2X Loud	NS	Y
Sentence		.5X Loud	4X Loud	*$p < 0.05$	Y
Sentence		Normal	2X Loud	NS	Y
Sentence		Normal	4X Loud	*$p < 0.05$	Y
Sentence		2X Loud	4X Loud	$p = 0.066$	Y
Sentence	Semitones for "I"	.5X Loud	Normal	NS	Y
Sentence		.5X Loud	2X Loud	$p = 0.051$	Y
Sentence		.5X Loud	4X Loud	*$p < 0.05$	Y
Sentence		Normal	2X Loud	NS	Y
Sentence		Normal	4X Loud	$p = 0.066$	Y
Sentence		2X Loud	4X Loud	NS	Y
Sentence	Semitones for s "a"p	.5X Loud	Normal	NS	Y
Sentence		.5X Loud	2X Loud	NS	Y
Sentence		.5X Loud	4X Loud	*$p < 0.05$	Y
Sentence		Normal	2X Loud	NS	Y
Sentence		Normal	4X Loud	*$p < 0.05$	Y
Sentence		2X Loud	4X Loud	$p = 0.070$	Y
Sentence	Area of Mouth Opening (mm^2)	.5X Loud	Normal	NS	Y
Sentence		.5X Loud	2X Loud	NS	Y
Sentence		.5X Loud	4X Loud	*$p < 0.01$	Y
Sentence		Normal	2X Loud	NS	Y
Sentence		Normal	4X Loud	*$p < 0.01$	Y
Sentence		2X Loud	4X Loud	NS	Y

*** represents a statistically significant pair-wise comparison**
NS represents a non significant pair-wise comparison
Y represents a noted visual trend
N represents no noted visual trend

Twenty-five comparisons were supported by both a visual trend and a significant (or near significant) statistic (see highlighted sections in Table 13). Table 14 indicates the statistically significant (or near statistically significant) pair-wise comparisons for the experimental group.

Table 14. Statistically significant pair-wise comparisons for the experimental group. Left – Right: the first column indicates the task performed; the second column shows the dependent variable; the third and fourth columns reveal the significantly different conditions; the fourth column indicates the exploratory statistic; and, the final column indicates the presence of a visual trend.

Experimental Group

Task	Variable	Condition 1	Condition 2	Statistic	Visual Trend
Max Phonation	Lung Volume Initiation (cm^3)	Normal	2X Loud	NS	Y
Max Phonation	Lung Volume Termination (cm3)	Normal	2X Loud	NS	Y
Max Phonation	Lung Volume Excursion (% VC)	Normal	2X Loud	NS	Y
Max Phonation	Intercostal Muscle Activity (%MVE)	Normal	2X Loud	NS	Y
Max Phonation	Oblique Muscle Activity (%MVE)	Normal	2X Loud	NS	Y
Max Phonation	Speed Quotient	Normal	2X Loud	NS	Y
Max Phonation	Fundamental Frequency (Hz)	Normal	2X Loud	$*p < 0.05$	Y
Max Phonation	Semitones	Normal	2X Loud	$*p < 0.05$	Y
Max Phonation	Area of Mouth Opening (mm^2)	Normal	2X Loud	NS	Y
Sentence	Lung Volume Initiation (cm^3)	.5X Loud	Normal	NS	Y
Sentence		.5X Loud	2X Loud	NS	Y
Sentence		.5X Loud	4X Loud	NS	Y
Sentence		Normal	2X Loud	NS	Y
Sentence		Normal	4X Loud	NS	Y
Sentence		2X Loud	4X Loud	$p = 0.067$	Y
Sentence	Lung Volume Termination (cm^3)	.5X Loud	Normal	NS	Y
Sentence		.5X Loud	2X Loud	NS	Y
Sentence		.5X Loud	4X Loud	NS	Y
Sentence		Normal	2X Loud	NS	Y
Sentence		Normal	4X Loud	NS	Y
Sentence		2X Loud	4X Loud	NS	Y
Sentence	Lung Volume Excursion (% VC)	.5X Loud	Normal	NS	N
Sentence		.5X Loud	2X Loud	NS	N
Sentence		.5X Loud	4X Loud	NS	N
Sentence		Normal	2X Loud	$p = 0.068$	N
Sentence		Normal	4X Loud	NS	N
Sentence		2X Loud	4X Loud	NS	N
Sentence	Intercostal Muscle Activity (%MVE)	.5X Loud	Normal	NS	Y
Sentence		.5X Loud	2X Loud	NS	Y
Sentence		.5X Loud	4X Loud	NS	Y

Sentence		Normal	2X Loud	NS	N
Sentence		Normal	4X Loud	NS	Y
Sentence		2X Loud	4X Loud	*$p < 0.05$	Y
Sentence	Oblique Muscle Activity (%MVE)	.5X Loud	Normal	NS	Y
Sentence		.5X Loud	2X Loud	NS	Y
Sentence		.5X Loud	4X Loud	NS	Y
Sentence		Normal	2X Loud	NS	Y
Sentence		Normal	4X Loud	NS	Y
Sentence		2X Loud	4X Loud	NS	Y
Sentence	Speed Quotient "I"	.5X Loud	Normal	NS	Y
Sentence		.5X Loud	2X Loud	NS	Y
Sentence		.5X Loud	4X Loud	NS	N
Sentence		Normal	2X Loud	*$p < 0.05$	Y
Sentence		Normal	4X Loud	NS	Y
Sentence		2X Loud	4X Loud	NS	Y
Sentence	Speed Quotient s"a"p	.5X Loud	Normal	NS	Y
Sentence		.5X Loud	2X Loud	*$p < 0.05$	Y
Sentence		.5X Loud	4X Loud	NS	Y
Sentence		Normal	2X Loud	NS	Y
Sentence		Normal	4X Loud	NS	Y
Sentence		2X Loud	4X Loud	NS	Y
Sentence	Fundamental Frequency (Hz) for "I"	.5X Loud	Normal	NS	Y
Sentence		.5X Loud	2X Loud	NS	Y
Sentence		.5X Loud	4X Loud	*$p < 0.05$	Y
Sentence		Normal	2X Loud	NS	Y
Sentence		Normal	4X Loud	*$p < 0.05$	Y
Sentence		2X Loud	4X Loud	NS	Y
Sentence	Fundamental Frequency (Hz) for s"a"p	.5X Loud	Normal	NS	Y
Sentence		.5X Loud	2X Loud	NS	Y
Sentence		.5X Loud	4X Loud	$p = 0.063$	Y
Sentence		Normal	2X Loud	NS	N
Sentence		Normal	4X Loud	$p = 0.060$	Y
Sentence		2X Loud	4X Loud	*$p \leq 0.001$	Y
Sentence	Semitones for "I"	.5X Loud	Normal	NS	Y
Sentence		.5X Loud	2X Loud	NS	Y
Sentence		.5X Loud	4X Loud	*$p < 0.05$	Y

Sentence		Normal	2X Loud	NS	Y
Sentence		Normal	4X Loud	*$p < 0.05$	Y
Sentence		2X Loud	4X Loud	$p = 0.056$	Y
Sentence	Semitones for s "a"p	.5X Loud	Normal	NS	N
Sentence		.5X Loud	2X Loud	NS	Y
Sentence		.5X Loud	4X Loud	*$p < 0.05$	Y
Sentence		Normal	2X Loud	NS	Y
Sentence		Normal	4X Loud	*$p < 0.05$	Y
Sentence		2X Loud	4X Loud	*$p \leq 0.001$	Y
Sentence	Area of Mouth Opening (mm^2)	.5X Loud	Normal	NS	N
Sentence		.5X Loud	2X Loud	NS	N
Sentence		.5X Loud	4X Loud	NS	N
Sentence		Normal	2X Loud	NS	N
Sentence		Normal	4X Loud	NS	N
Sentence		2X Loud	4X Loud	NS	N

*** represents a statistically significant pair-wise comparison**
NS represents a non significant pair-wise comparison
Y represents a noted visual trend
N represents no noted visual trend

Seventeen comparisons were supported by both a visual trend and a significant (or near significant) statistic (see highlighted sections in Table 14). The control group had more significant pairwise comparisons than the experimental group. The following pair-wise comparisons were statistically significant (or near statistically significant) in both groups: F_0 (in Hz and semitones) for maximum sustained phonation tasks (Normal Loud vs. 2X Normal Loud), for "I" (.5X Normal Loud vs. 4X Normal Loud; Normal Loud vs. 4X Normal Loud) in sentence tasks, and for s "a"p (.5X Normal Loud vs. 4X Normal Loud; Normal Loud vs. 4X Normal Loud; 2X Normal Loud vs. 4X Normal Loud) in sentence tasks.

DISCUSSION

The purpose of the present study was to provide a comprehensive understanding of the physiological adjustments made by the speech mechanism when typically developing children and children with cerebral palsy (CP) produced maximum sustained phonation tasks and sentences differing in vocal loudness. Descriptive and exploratory parametric statistical analyses were conducted on nine variables for maximum phonation tasks and twelve variables for sentence tasks. These selected variables represented respiratory behaviours, chest wall muscle activation patterns, laryngeal adjustments and mouth opening changes responsive to increases or decreases in vocal loudness. The current findings provided some initial observations about how children adjusted the speech mechanism to achieve changes in vocal loudness. Whereas differences pertaining to the recruitment of the speech subsystems were observed between typically developing children and those with CP, several similarities also were found.

All of the participants in the study were able to produce maximum sustained phonations at two loudness levels. In addition, all participants with the exception of one child with CP, were able to produce sentences consistently at the four targeted loudness levels. Moreover, all children in the present study exhibited predicted changes in estimated subglottal pressure associated with changes in vocal loudness.

Table 15. Hypotheses for the maximum sustained phonation task. Hypotheses for the control group are listed on the left and the hypotheses for the experimental group are listed on the right. Whether or not the hypothesis was supported is indicated.

Control Group		Experimental Group	
Hypothesis *From Normal to 2X Loud,*	**Supported?**	**Hypothesis** *From Normal to 2X Loud,*	**Supported?**
LVI will increase.	YES	LVI will not change.	---
LVT will decrease.	YES	LVT will not change.	---
LVE will increase.	YES	LVE will not change.	---
IC %MVE will increase.	YES	IC %MVE will increase.	YES
OB %MVE will increase.	YES	OB %MVE will increase.	YES
There will be no change in SQ.	NO	There will be an increase in SQ.	NO
F_0 will increase.	YES	F_0 will increase.	YES
MA will increase.	NO	MA will not change.	NO

--- = Inconclusive

Table 16. Hypotheses for the sentence task. Hypotheses for the control group are listed on the left and the hypotheses for the experimental group are listed on the right. Whether or not the hypothesis was supported is indicated.

Control Group		Experimental Group	
Hypothesis *From .5X Loud to 4X Loud,*	**Supported?**	**Hypothesis** *From .5X Loud to 4X Loud,*	**Supported?**
LVI will increase.	YES	LVI will not change.	NO
LVT will decrease.	NO	LVT will not change.	NO
LVE will increase.	YES	LVE will not change.	---
IC %MVE will increase.	YES	IC %MVE will increase.	YES
OB %MVE will increase.	---	OB %MVE will increase.	YES
There will be no change in SQ.	NO	There will be an increase in SQ.	---
F_0 will increase.	YES	F_0 will increase.	YES
MA will increase.	YES	MA will not change.	---

--- = Inconclusive

When asked to produce maximum sustained phonation tasks at normal and twice normal loudness levels, typically developing children (1) increased lung volume initiations (cm^3), (2) slightly lowered lung volume terminations (cm^3), (3) increased lung volume excursions (% VC), (4) increased intercostal and oblique muscle activity (% MVE), (5) increased speed quotient, (6) increased fundamental frequency (in Hz and semitones), and (7) decreased area of mouth opening (mean difference from normal in mm^2). In contrast, children with CP (1) slightly increased lung volume initiation (cm^3), (2) lowered lung volume terminations (cm^3), (3) marginally increased lung volume excursions (% VC), (4) increased oblique muscle activity (% MVE), (5) decreased speed quotient, (6) increased

fundamental frequency (in Hz and semitones), and (7) increased area of mouth opening (mean difference from normal in mm^2), for the louder condition during maximum sustained phonation tasks.

When asked to produce sentences at twice and four times louder than normal levels, typically developing children (1) increased lung volume initiations (cm^3), (2) increased lung volume excursions (% VC), (3) increased intercostal and oblique muscle activity (% MVE), (4) increased speed quotient, (5) increased fundamental frequency (in Hz and semitones), and (6) increased area of mouth opening (mean difference from normal in mm^2). When asked to produce sentences at half normal loudness levels, typically developing children (1) decreased lung volume initiations (cm^3), (2) decreased lung volume excursions (%VC), (3) decreased intercostal muscle activity (%MVE), (4) increased oblique muscle activity (% MVE), (5) increased speed quotient, (6) decreased fundamental frequency (in Hz and semitones), and (7) decreased area of mouth opening (mean difference from normal in mm^2).

When asked to produce sentences at twice and four times louder than normal levels, children with CP (1) increased lung volume initiations (cm^3) (excluding four times normal loudness), (2) lowered lung volume terminations (cm^3), (3) marginally increased lung volume excursions (% VC) (excluding four times normal loudness), (4) increased intercostal and oblique muscle activity (% MVE), (5) increased fundamental frequency (in Hz and semitones), and (6) decreased area of mouth opening (mean difference from normal in mm^2). When asked to produce sentences at half normal loudness levels, children with CP (1)

decreased lung volume initiations (cm^3), (2) lowered lung volume terminations (cm^3), (3) decreased intercostal and oblique muscle activity (%MVE), (4) decreased fundamental frequency (in Hz and semitones), and (5) decreased area of mouth opening (mean difference from normal in mm^2).

Typically developing children made similar adjustments to the speech mechanism when asked to produce maximum sustained phonations and sentences at louder than normal levels. In contrast, children with CP recruited speech subsystems in a different manner for maximum sustained phonation tasks than they did for speech tasks, particularly at the levels of respiration, muscular activation of the chest wall and laryngeal movements. The pattern of adjustments by the speech mechanism was similar between control children and children with CP when producing sustained maximum phonations. However, children with CP had a noticeably smaller performance envelope than their matched counterparts. Adjustments made at the level of the larynx (relative to pitch) and mouth opening were similar across loudness levels between the two groups for sentence productions. However, respiratory, muscular and laryngeal (relative to speed of vocal fold movement) adjustments were different across loudness levels between the two groups. More specific interpretations of results are presented in the context of each speech subsystem as well as interactions among subsystems across tasks and participant groups.

Respiratory Subsystem

Lung volume adjustments and activity of chest wall muscles contribute to changes in loudness during conversational speech. The key role of the respiratory

system in loudness control is to modulate tracheal (subglottal) pressure. Subglottal pressure must be maintained between 5 and 7 cmH_2O for speech production at conversational loudness (Hixon et al., 1973). Finnegan et al. (2000) suggested that a change in tracheal pressure was largely due to a change in alveolar pressure. During maximum sustained phonation and speech tasks, increases in muscular pressure are related to the difference between the alveolar pressure desired and the prevailing relaxation pressure of the respiratory system. According to Hixon and colleagues (Hixon et al., 1976), greater inspiratory muscular pressures are used by the chest wall during inspiration and increased expiratory muscular pressures are used during expiration for loud conversational speech, compared to typical conversational speech. A larger inspiration and thus, higher lung volume initiation, results in greater potential respiratory energy (e.g., prevailing recoil pressures of the lung and chest wall). The release of this energy and the increased expiratory muscle activity raises alveolar pressure above its typical level necessary for speech produced at normal loudness (Hixon et al., 1976). Stathopoulos and Sapienza (1997) indicated that children are similar to adults and maintained tracheal pressures between 9 and 15 cmH_20 for loud speech productions.

Lung Volume Events

Maximum Sustained Phonation

Based on what is known about speech breathing, it was predicted that typical children would increase lung volume initiations (cm^3) relative to end expiratory level (EEL), lower lung volume terminations (cm^3) relative to EEL,

which would subsequently result in larger lung volume excursions (in % VC) when asked to produce maximum sustained phonations at twice normal loudness levels. The control group in the current study generally supported these predictions. However, these children exhibited a great deal of variability and a wide range of lung volume excursions (40 to 95% VC) when performing these tasks.

Even with practice, the typically developing children in the current study were not always able to phonate throughout the range of their vital capacity consistently. The range in %VC observed in these children is similar to the range reported for typical adult speakers (e.g., 33 to 95% VC) by Solomon, Garlitz, and Milbrath (2000). According to Solomon and colleagues (Solomon et al., 2000) a small portion of the vital capacity may be wasted before phonation begins, due to high passive alveolar pressure and/or incomplete vocal fold closure. Furthermore, some amount of lung volume must remain after a sustained maximum phonation is performed in order for the chest wall to generate adequate pressure to sustain vocal fold oscillation. The ability to perform a sustained phonation depends on vital capacity, control of the expiratory air stream and laryngeal valving of the airstream (Wit, Massen, Gabreel, & Thoonen, 1993).

In the present study, it was predicted that children with CP would not change lung volume initiation (cm^3), lung volume termination (cm^3), and lung volume excursion (% VC) when producing maximum sustained phonations at twice normal loudness levels. The children with CP in the current study, showed a slight increase in lung volume initiations (cm^3) relative to EEL. They also

terminated at slightly lower lung volume terminations (cm^3) relative to EEL, resulting in a slight increase lung volume excursion (% VC). Thus, the children with cerebral palsy used a much smaller range of lung volume excursions then did their typical counterparts during maximum phonation tasks. This reduced range of lung volume excursions may have reflected a smaller performance envelope in the children with cerebral palsy, and as such they demonstrated a reduced range of performance in comparison to the typically developing children. Netsell, Lotz, Peters, & Schulte (1994) noted that children with cerebral palsy have smaller vital capacities than typical children. Whereas the current study did not perform standardized lung function testing, it was noted that vital capacity maneuvers as measured via chest wall kinematics, resulted in significantly smaller absolute lung volume excursions from the CP group. Moreover, maximum sustained phonations were produced within a very small range of the vital capacity (e.g., 20 - 40 %VC), supporting previous observations of reduced inspiratory capacity, reduced vital capacity, reduced expiratory reserve volume and respiratory muscular weakness, relative to typical children (Workinger, 2005; Netsell et al., 1994). Because these children with CP were not able to take advantage of increased respiratory recoil occurring at higher lung volumes, they appeared to compensate by increasing the activity from both intercostal and oblique muscle groups which can be an effective strategy as suggested by Hixon and colleagues, 1976.

Maximum sustained phonation tasks provided an opportunity to observe how typically developing children and children with CP would adjust the respiratory system in the context of their performance envelope. Both groups of

children were not able to consistently produce phonations throughout their vital capacities. Most of the time, typical children took advantage of respiratory energy (e.g., recoil pressure of the lung and chest wall) to produce sustained phonations at two loudness levels. In contrast, children with CP phonated across a significantly smaller vital capacity range and operated at lower lung volumes relative to EEL than their counterparts. Therefore, to maintain the necessary subglottal pressures for meeting loudness targets, these children had to employ much more muscular effort. McFarland and Smith (1992) suggested that at low lung volumes, active expiratory forces are necessary to supplement reduced recoil pressures that are likely insufficient for initiating and maintaining phonation. They concluded that there is considerable flexibility in the configuration of the respiratory system for performing phonation tasks.

Sentences

Lung volume events associated with running speech typically occur in the midrange of the vital capacity (VC) (Hixon, 1973). Generally, conversational speaking begins at twice the resting tidal breathing depth and continues to near resting expiratory level or end expiratory level (EEL) of the breathing apparatus (Hixon et al., 1973). Compared to lung volumes used for speech produced at a typical loudness level (40 to 60% VC), loud speech demands higher alveolar pressures and is typically initiated from higher lung volumes (approximately 60 to 80% VC) to take advantage of the higher respiratory recoil forces available. Soft speech requires low alveolar pressures, often initiated at lower lung volumes (generally above end expiratory level) compared to speech of normal loudness. In

adults, expiratory phrases produced using soft speech, end near the same lung volumes as speech of normal loudness (slightly above or at resting expiratory level; 35 to 40% VC), whereas loud utterances are often terminated at lung volumes above the resting expiratory level (Hixon, 1973). Previous studies with children indicated that lung volume terminations are often well below EEL for both normal and louder productions (Boliek et al, 2009; Stathopoulos & Sapienza, 1997). To increase loudness of speech, adjustments are made on both the inspiratory and expiratory sides of the respiratory cycle (Hixon, 1973).

Based on previous literature regarding respiration and vocal loudness, it was predicted that from half normal loudness to four times normal loudness, lung volume initiation (cm^3) would increase relative to EEL, lung volume termination (cm^3) would be lower relative to EEL, and lung volume excursion (% VC) would increase. The control group in the current study generally supported these predictions. For the most part, typically developing children took advantage of the higher respiratory recoil forces available at higher lung volumes, supporting previous observations that overall, typically developing children use higher lung volume initiations for louder speech (Stathopoulos & Sapienza, 1997). The control group averages derived from the current study did not indicate a visual trend for lung volume terminations at lower lung volumes relative to EEL with increased loudness, as observed by Stathopoulos & Sapienza (1997). Previous research has shown that by approximately twelve years of age, children may begin to demonstrate more "adult-like" lung volume terminations which approach EEL (Stathopoulos & Sapienza, 1997). Hoit and colleagues (Hoit et al., 1990) reported

that substantial differences exist between seven year old children and ten year old children. For example, younger children have larger lung volume initiations and terminations for breath groups and thus, larger lung volume excursions per breath group, compared to children over ten years of age. In addition, younger children have increased performance variability compared to older children (Hoit et al., 1990). It is possible that the typically developing children in this study were at different points in their developmental trajectory for respiration and thus, lung volume terminations were highly variable and did not reveal a clear trend in any direction.

Verschuren and Takken (2010) noted that children with cerebral palsy have significantly lower respiratory capacities compared to typically developing children. Further, Netsell, Lotz, Peters, & Schulte (1994) reported that children with cerebral palsy expire more air per syllable than typical children, demonstrating poor muscle control. Inadequate valving at the level of the larynx, velopharynx, and/or orofacial structures, may exaggerate respiratory inefficiencies (Workinger, 2005). From half normal loudness to four times normal loudness, it was predicted that lung volume initiation (cm^3), lung volume termination (cm^3), and lung volume excursion (% VC) would not change for the experimental group. Again, children with cerebral palsy used a smaller range of lung volume excursions than did their typical counterparts during sentence tasks. Experimental group averages for lung volume initiation showed an increase in lung volume initiation relative to EEL with increases in vocal loudness, except at four times normal loudness, where lung volume initiation relative to EEL was the lowest.

Experimental group averages for lung volume termination indicated a decrease in lung volume termination relative to EEL for half normal loudness and four times normal loudness. Lung volume excursions increased only marginally for sentences produced at two times normal loudness, followed by a slight decrease in lung volume excursion for four times normal loudness. The four times normal loudness condition appeared to lead the experimental group to "abandon" a lung volume adjustment for another more accessible strategy, in order to increase loudness (e.g. recruitment of intercostal muscles). The children with CP were not able to take advantage of increased respiratory recoil available at higher lung volumes and thus it appeared it was necessary that they increase muscle activity in order to achieve higher subglottal pressure associated with louder speech.

Sentence tasks provided an opportunity to observe how typically developing children and children with CP adjusted the respiratory system in the context of their operating range. Typically developing children used the biomechanical efficiency associated with higher lung volume initiations as suggested by Hixon and colleagues (1976). However, as a group, these children exhibited a range of lung volume initiations, terminations and excursions for these sentence tasks. Variability is the hallmark of speech breathing in children for maximum performance tasks as well as for conversational speaking (Boliek, et al., 2009; Hoit et al, 1990; Stathopoulos & Sapienza, 1997). Stathopoulos and Sapienza (1997) found significant variability for lung volume events among children in the same age range of the current sample. They attributed variability to a lack of speech breathing efficiency in the developing system. Respiratory

inefficiency can be observed in typically children and to a greater extent in children with cerebral palsy. Consequently, lung volume terminations often occur well below EEL requiring more muscular effort to maintain subglottal pressure for speech as demonstrated by the experimental group in the current study (Boliek, et al., 2009; Stathopoulos & Sapienza, 1997).

Muscular Activity

Maximum Sustained Phonation

Intercostal muscle activity. For both groups, it was expected that intercostal muscle activity would increase from normal loudness to twice normal loudness during maximum phonation tasks. Based on their location and action on the chest wall, internal intercostal muscles are used by healthy adults to make small rib cage adjustments for the purpose of maintaining steady alveolar pressure needed for sustained phonations at both normal and loud levels (Hixon, 1973).

As expected, control group averages for percent maximum voluntary effort for intercostal muscles indicated a small average increase of approximately 10%MVE. Children with CP were more variable but also showed slight increases in intercostal %MVE for the louder condition. Given their smaller performance envelope for lung volume events, a larger average increase in muscular effort was expected for the children with CP. A larger increase in intercostal muscle activity from normal loudness to twice normal loudness was observed in three of four participants with cerebral palsy. One participant actually decreased intercostal %MVE for phonations produced at twice normal loudness levels but significantly increased oblique %MVE in the same condition. This participant started

maximum phonation tasks much lower in her lung volume range and terminated her phonations well below EEL. Another participant increased intercostal %MVE at twice normal loudness levels, but increased his oblique %MVE to a greater extent. This participant also terminated his phonations well below EEL. At these low levels, the rib cage, but not the abdomen, may have reached its maximum inward displacement. In this case, muscular recruitment from the obliques would assist in the inward displacement of the abdomen and subsequent maintenance of the proper subglottal pressures at these lower lung volumes (Boliek et al., 1997). Inspiratory capacity appears to be limited in the experimental group.

Some intercostal muscle activation was necessary for maintaining alveolar pressure during sustained maximum phonation tasks at both loudness levels as previously shown by Hixon et al. (1976). However, on average intercostal recruitment was around 50% MVE for normal loudness phonations and only increased by 10% MVE for the louder productions. These observations indicated that small changes in muscular effort from these muscle groups along with lung and chest wall recoil pressures are sufficient for achieving targeted loudness levels and associated subglottal pressures, as both relaxation pressure and muscular pressure contribute to sentence production (Hixon, 1973; Hixon et al., 1976; Weismer, 1985).

Oblique muscle activity. For both groups, it was expected that oblique muscle activity would increase from normal loudness to twice normal loudness during maximum phonation tasks. Hoit et al. (1988) suggested that the oblique muscles play an important role in configuring the chest wall during upright resting

tidal breathing and speech production. Large changes in pressure involve increased abdominal (oblique & transverse) muscle activity (Hixon et al., 1976).

Relative to intercostal %MVEs, oblique %MVEs were much lower on average for both groups. Control group averages for %MVE for oblique muscles indicated a small average increase (6%) for phonations produced at twice normal loudness levels. Based on current literature, this was an expected finding. The muscles most significant for maintaining steady alveolar pressure are the internal intercostals (Hixon, 1973); large changes in pressure, such as those required for loud utterances, involve additional abdominal muscle activity (e.g. oblique muscles) (Hixon et al., 1976). The experimental group exhibited a much greater increase (13%) from the normal loudness condition to the two times normal loudness condition. The current findings indicate that a small increase in muscular effort from the oblique muscles in combination with intercostal muscle groups assist in the maintenance of subglottal pressures needed for sustained phonations. Patterns of recruitment were similar for both groups and support observations made previously (Hoit et al., 1988).

Sentences

Intercostal muscle activity. For both groups, from half normal loudness to four times normal loudness, intercostal muscle activity was predicted to increase with loudness. This prediction was based on the knowledge that internal intercostal muscles influence loudness because they can create rapid, small variations in driving pressure supplied to the larynx and upper airway (Hixon, 1973).

As expected, a visual trend towards increasing intercostal muscle activity with loudness, predominately at four times normal loudness, was observed in both groups. Overall, the control group exhibited intercostal %MVE values that were much lower for speaking (13 to 26%MVE) than for producing maximum sustained phonations (40 to 50%MVE). Given the differences in tasks (e.g., performance envelope vs. operating range) it makes sense that intercostal recruitment may not need to be as much even when producing sentences at louder levels.

Interestingly, the children with CP showed average intercostal %MVEs for sentence productions (32 to 58%) that were similar to their average %MVEs for maximum sustained phonations (48 to 49%). All children with CP exhibited larger intercostal %MVE values than their matched counterparts. In general, children with CP initiated and terminated sentences at lower lung volumes regardless of loudness condition. Lung volume events associated with sentence productions were not largely dissimilar to events observed when children with CP were asked to produce maximum sustained phonations. Similar levels of intercostal recruitment for sentence productions may indicate that these children with CP may have been functioning at or near their performance envelope. Internal intercostal recruitment may indicate a muscular-based compensatory strategy to achieve subglottal pressures for normal and loud speech in this population. During speech, the respiratory apparatus adds muscular pressure at each instant that is precisely equal to the difference between the alveolar pressure desired and the relaxation pressure available. Each alveolar pressure produced in

speech demands a different muscular pressure at each lung volume (Finnegan et al., 2000). Thus, a reduced lung volume requires increased muscular pressure, in order to meet the target loudness level.

Children with CP show an inability to maintain a constant subglottal pressure across an utterance (Netsell et al., 1994) and thus, may require greater intercostal muscular effort to do so. Because intercostal muscles create rapid, small variations in driving pressure, they appear to play a significant role in loudness control for sentence tasks in children with CP (Hixon, 1973).

Oblique muscle activity. For both groups, from half normal loudness to four times normal loudness, oblique muscle activity was predicted to increase with loudness. Hoit et al. (1988) noted that abdominal (obliques & transverse abdominis) EMG activity was greatest during loud speech production and during softer speech produced at low lung volumes. To reiterate, large changes in pressure involve intercostal muscle activity and the addition of abdominal muscle activity (Hixon et al., 1976).

As supported by current literature, a visual trend towards increasing oblique muscle activity from normal loudness to half normal loudness and from normal loudness to four times normal loudness was noted in the typically developing children. Overall, control children exhibited oblique %MVE values that were much lower for speaking (11 to 24% MVE) than for producing maximum sustained phonations (37 to 44%MVE). Again, given the differences in tasks (e.g., performance envelope vs. operating range) it makes sense that oblique muscle recruitment may not need to be as much as needed for maximum

phonation, even when producing sentences at louder levels. In addition, because typical children adjusted lung volume events to meet the demands of speaking softer or more loudly, relatively small amounts of muscle activity were needed to achieve targeted subglottal pressures at these prevailing lung volumes (Finnegan et al., 2000).

Overall, children with CP exhibited oblique %MVE values that were similar or slightly lower for speaking (13 to 34% MVE) than for producing maximum sustained phonations (27 to 40%MVE). A linear visual trend for increasing %MVE for oblique muscles with increased loudness was noted in the experimental group. On average, children with CP appeared to recruit more effort from the oblique muscles than children in the control group, with the exception of sentences produced at half normal loudness where the %MVE for obliques was slightly greater for control subjects. Greater muscle activity in the experimental group, compared to the control group, is again likely a compensation technique for reduced lung volume initiations in the experimental group. In addition, these muscular compensations observed in the children with CP may be partly related to downstream inadequacies of the speech mechanism, such as difficulty abducting and adducting vocal folds, insufficient maintenance of vocal fold tension, and abnormal oral movement patterns (Workinger, 2005).

Laryngeal Subsystem

Changes in vocal loudness depend on an interaction between increases in subglottal pressure and vocal fold tension (Ludlow, 2005). Increases in vocal loudness are associated with a longer closed phase of the vocal folds (i.e. a higher

speed quotient) and greater adductory forces, resulting in an increase in laryngeal opposing pressure (Stathopoulos & Sapienza, 1993). These forces facilitate the build-up of tracheal air pressure which increases as speech becomes louder (Hirano et al., 1969). In order to contain the increased tracheal air pressure and prevent it from escaping, the opposing pressure must increase by contractions of the lateral cricoarytenoid and interartyenoid muscles (Ludlow, 2005) resulting in increased laryngeal airway resistance. Activation of the laryngeal muscles affects the amplitude and frequency of vocal fold vibration (Finnegan et al., 2000). Increases in tracheal pressure may be related to increased speed of vocal fold closure, increased air particle velocity through the glottis, increased fundamental frequency (perceived as pitch change), and increased amplitude of vocal fold vibration (Stathopoulos & Sapienza, 1997).

Speed Quotient

Maximum Sustained Phonation

Based on the results from children studied by Stathopoulos and Sapienza (1997) it was predicted that the children in the current study would not systematically change laryngeal speed quotient with increases in vocal loudness during maximum phonation tasks. Contrary to what was expected, typically developing children increased speed quotient from normal loudness to twice normal loudness. This finding can be partially explained by previous research reported by Woo (1996). He studied vocal fold behavior in thirty-two male and thirty-three female adult speakers, with typical speech productions, using videostrobolaryngoscopic images. He observed that as vocal loudness increased,

the closing phase of the vocal fold vibratory cycle became shorter and the closed period became longer. This resulted in an increase in speed quotient. If the current group of typical children also increased the rate of the closing phase and subsequently increased the vocal fold closed period as suggested by Woo (1996), then the speech quotient measurement would increase as children produced louder tokens. These results also make sense in light of the fact that these children sometimes, produced phonations starting from very high lung volumes and probably increased subglottal pressure as inferred by the oral pressure measures. So as suggested by Ludlow (2005), these children probably employed increases in lateral cricoarytenoid and interartyenoid muscle contractions which, in turn would increase the speed quotient.

Stathopoulos & Sapienza (1997) measured airflow open quotient, maximum flow declination rate, tracheal pressure, translaryngeal airflow, estimated laryngeal airway resistance, and fundamental frequency in both adults and children during repetitions of /pa/ at low, medium, and high sound pressure levels. They found that children did not decrease open quotient as loudness increased, but they did note an increase in maximum flow declination rate, greater alternating flow, higher tracheal pressure and laryngeal airway resistance, and higher fundamental frequency as loudness increased. Stathopoulos & Sapienza (1997) may not have noted a change in open quotient as loudness increased because open quotient is less sensitive to changes in dB SPL compared to speed quotient (Sapienza et al., 1998). Further, Stathoplous & Sapienza (1997) did not use an EGG waveform to make their open quotient measure. Rather, they used a

glottal airflow waveform. A glottal airflow waveform indicates greater change in the open phase, whereas the EGG waveform manifests greater change in the closed phase; the closed phase is more sensitive to changes in dB SPL than the open phase (Woo, 1996; Sapienza et al., 1998). Stathopoulos & Sapienza (1997) did, however, find that maximum flow declination rate increased as loudness increased. Maximum flow declination rate reflects how the closing phase responds to changes in dB SPL. The maximum flow declination rate has a strong positive correlation with SPL (Sapienza et al., 1998). Thus, it appears that the children in the Stathopoulos & Sapienza (1997) study made a laryngeal adjustment as loudness increased, though this adjustment was not indicated by their open quotient measure.

During maximum phonation tasks, it was predicted that speed quotient would increase from normal loudness to twice normal loudness for the experimental group. It was expected that children with cerebral palsy would make a laryngeal adjustment in order to compensate for a compromised respiratory system. Further, it was anticipated that hyperfunctioning vocal folds, typical of children with CP (Workinger, 2005), may cause significant laryngeal "squeezing" during loudness adjustments, especially when asking these children to phonate maximally.

As a group, children with cerebral palsy did not demonstrate an average increase in speed quotient from normal loudness to twice normal loudness. Two of four participants with CP demonstrated an increase in speed quotient. One of these children also increased lung volume initiation in the louder condition and

likely also increased subglottal pressure. The other child made a minimal lung volume adjustment and increased intercostal muscle activity in the louder condition; she likely relied on a laryngeal adjustment and a muscle adjustment in order to increase loudness. One participant did not change speed quotient. However, he showed an increase in lung volume initiation and a lower lung volume termination, along with increased intercostal and oblique muscle amplitude, and increased mouth opening in the louder condition; thus a speed quotient adjustment was likely not necessary or maybe possible. Laryngeal airway resistance can increase with an increase in tracheal pressure, without a change in adduction of the vocal folds (Finnegan et al., 2000). One other participant showed a large decrease in speed quotient. This participant appeared to increase loudness primarily by increasing oblique muscle activity, fundamental frequency and area of mouth opening. For the two CP participants who did not adjust speed quotient, a combination of motor coordination between the respiratory and laryngeal subsystems and refined vocal fold control may have contributed.

It is important to note that fundamental frequency is not related to speed quotient, as the opening and closing glottal slopes do not change with fundamental frequency (Woo, 1996); that is an increase in vocal fold tension can occur without effecting speed quotient.

Stathopoulos and Sapienza (1993) noted three respiratory-laryngeal patterns in adults as they increased vocal intensity during syllable trains. The most common pattern exemplified a decrease in open quotient, an increase in maximum flow declination rate, an increase in lung volume initiation and excursion, and an

increase in tracheal pressure. The second most common pattern was described by an increase in open quotient, an increase in lung volume initiation and excursion, and an increase in tracheal pressure. The least common pattern indicated a decrease in open quotient, an increase in maximum flow declination rate, an increase in laryngeal airway resistance, and an increase in tracheal pressure. These different laryngeal-respiratory patterns observed by Stathopoulos and Sapienza (1993) support the idea that a target dB SPL can be achieved by a variety of adjustments in the speech mechanism. Children with CP often have difficulties making appropriate adductions and abductions of the vocal folds (Workinger, 2005), and thus they must rely on other speech mechanism adjustments to reach a target SPL. Further inefficient thryoarytenoid and lateral cricoartyenoid muscles could interfere with proper valving of the expiratory airstream and thus the regulation of vocal loudness (Baker et al., 2001).

Sentences

From half normal loudness to four times normal loudness, it was predicted that speed quotient would not change in typically developing children, based on previous findings which showed that children did not increase glottal closure time, as measured by open quotient, with increased vocal loudness (Stathopoulos & Sapienza, 1997).

Contrary to what was predicted, typically developing children increased speed quotient in sentences from normal to half normal loudness and from normal loudness to four times normal loudness. The increase in speed quotient from normal loudness to half normal loudness may have resulted from an increase in

thyroarytenoid (TA) muscle activity. Baker and colleagues (Baker et al., 2001) noted that both soft and loud vocal loudness conditions indicated similar EMG activity in the TA muscle in older adults. Both older adults and children have reduced vocal efficiency in that both groups do not achieve the same degree of medial adductory comprehension as typical adults do (Baker et al., 2000; Tang & Stathopoulos, 1995). TA EMG muscle activity is not well correlated with laryngeal airway resistance, which suggests that when the TA muscle contracts it may allow greater amplitude of vocal fold vibration without increasing laryngeal airway resistance and fundamental frequency (Finnegan et al., 2000). An increase in speed quotient also can be expected at increasing levels of loudness because as loudness increases, the closing phase becomes shorter, the slope expressing area change per frame becomes greater and steeper, and the closed period becomes longer. This can be expressed as an increase in speed quotient (Woo, 1996).

From half normal loudness to four times normal loudness, it was predicted that speed quotient would increase in children with cerebral palsy. Children with cerebral palsy showed an average increase in speed quotient with loudness, except at twice normal loudness. Three out of four participants with CP indicated their lowest speed quotient at twice normal loudness for both “I” and s “a”p. The participant with CP who did not demonstrate this decreased speed quotient at twice normal loudness did not reveal a clear pattern in his use of laryngeal adjustments with loudness. The unexpected decrease in speed quotient at twice normal loudness may simply be due to variability in vocal fold control found in children with CP. Moreover, the children with CP may not have needed to make

as significant of a laryngeal adjustment at twice normal loudness because they made their largest adjustment in lung volume initiation at twice normal loud and in addition, they used a large amount of respiratory muscle effort at twice normal loudness levels.

Sapienza et al. (1998) highlighted that EGG signals can be difficult to interpret because (a) extreme variations in waveform shape can occur within and across subjects (most EGG measures assume a wave shape that approximates Figure 4; such an assumption may be safe in the absence of pathology but it may not be valid for many patients with atypical vocal fold behavior); (b) vertical phase differences can occur between the lower and upper margins of the vibrating folds; and, (c) mucus strands can occur across the glottis. Methodological error may have resulted from residual effects of high pass filtering. Further, weak signals may have resulted when individuals had excess tissue around their neck. Nevertheless, the current findings do indicate that both typically developing children and children with CP make some laryngeal adjustments when producing sentences that differ in vocal loudness.

Fundamental Frequency

Maximum Sustained Phonation

Again, based on findings from Stathopoulos and Sapienza (1997), F_0 increased with loudness in both children and adults. Others also have shown that F_0 increased with vocal loudness (Hirano et al., 1969; Dromey & Ramig, 1998a; Baken & Orlikoff, 2000; Baker et al., 2001). Increases in F_0 are due to an increase in vocal fold tension, medial compression, or subglottic pressure (Hirano et al.,

1969). In children, F_0 appears to be was most affected by tracheal pressure because of the relatively small size of the vocal folds themselves (Stathopoulos & Sapienza, 1997). Therefore, it was predicted that fundamental frequency (in Hz and semitones) would increase for both the control group and the experimental group, during maximum phonation tasks produced at twice normal loudness levels. F_0 was measured in both Hz and semitones. The results of this study did not change when applying a normalization technique to the F_0 values. Perhaps the similarities in vocal tract size in the current population did not necessitate the normalization process. Nevertheless, the fact that the both F_0 and semitone data revealed the same results, allows for the interpretation that differences were not related to vocal tract size.

The results of the current study confirmed that both groups of children exhibited an increase in F_0 (in Hz and semitones) from normal loudness to twice normal loudness. On average, the experimental group produced maximum phonation tasks at higher fundamental frequencies than typically developing children for both loudness conditions. Overall higher frequencies in the experimental group for maximum phonation tasks may be a result of hyper-adducted tense vocal folds, common in children with cerebral palsy (Yorkston et al., 1999).

Sentences

For reasons stated previously, it was predicted that from half normal loudness to four times normal loudness, fundamental frequency (in Hz and semitones) would increase for both the control group and the experimental group

when producing sentences. Both groups of children exhibited increases in fundamental frequency (in Hz and semitones) with increases in vocal loudness. In the control group three children increased fundamental frequency for s “a”p at half normal loudness and one child with CP also increased fundamental frequency for s “a”p at half normal loudness, indicating an increase in vocal fold tension to maintain voicing near the subglottal pressure threshold. This was expected, based on previous findings regarding fundamental frequency and loudness (Hirano et al., 1969; Fisher & Swank, 1997; Dromey & Ramig, 1998a; Baken & Orlikoff, 2000; Baker et al., 2001). Again, children with CP in the current study produced sentences at higher overall fundamental frequencies than their typically developing counterparts. Jacques, Rastatter, & Sullivan (1985) observed that adult speakers with spastic cerebral palsy spoke at significantly higher mean fundamental frequencies, during sentence tasks, than typical adult speakers. In addition to the possibility that hyper-adducted vocal folds resulted in the production of higher fundamental frequencies, children with cerebral palsy also used significantly more respiratory muscular effort during sentence tasks, compared to typically developing children. Increased muscular effort may have increased vocal fold strain or increased tracheal pull (Titze & Riede, 2010).

There is some evidence to suggest that by increasing respiratory and laryngeal muscular effort in combination with high positive lung pressure, significant tension is realized by the vocal ligament. In their studies on elk calls (e.g., high and low frequency bugles), Titze and Riede (2010) demonstrated that by increasing tension of the vocal ligament, a significant increase in fundamental

frequency and noticeable (10 dB SPL) difference in sound pressure would result. In contrast, these researchers found that lower fundamental frequencies were produced with large lung pressures coupled with high glottal airflow which resulted in low vocal fold strain and lower dB SPL. Thus, it appeared that elk used a higher frequency to more efficiently increase dB SPL. Perhaps, children with CP used higher frequencies as a compensatory strategy at the level of the larynx to more effectively increase dB SPL for loudness conditions.

No systematic changes in speed quotient occurred with changes in pitch (Woo, 1996). In the control group, both speed quotient and fundamental frequency tended to increase as loudness increased from normal loudness to twice/four times normal loudness, but as supported by the observed increase in speed quotient and decrease in fundamental frequency from normal loudness to half normal loudness; the two measures are not necessarily related.

On average, the control group increased speed quotient in the louder maximum sustained phonation condition, and increased speed quotient at half normal loudness as well as twice and four times normal loudness sentence productions. On average, the experimental group decreased speed quotient in the louder maximum sustained phonation condition, and increased speed quotient at half normal loudness and four times normal loudness. Overall both groups increased fundamental frequency as loudness increased in both maximum sustained phonation tasks and sentence tasks. Thus, both groups used a laryngeal adjustment when changing vocal loudness. It appears that both groups adjusted vocal fold tension, medial compression, or subglottic pressure (Hirano et al.,

1969). Moreover, both groups adjusted their speed and pattern of vocal fold closure (Woo, 1996), in order to achieve different vocal loudness levels.

Oroarticulatory System

Loud speech often involves a deeper inhalation, a greater degree of vocal fold closure, and larger articulatory excursions compared to speech produced at typical conversational levels (Dromey & Ramig, 1998b). Dromey and Ramig (1998b) compared the effects of changing sound pressure level (SPL) on respiratory, phonatory, and articulatory behavior during sentence production. Five men and 5 women repeated the sentence, "I sell a sapapple again," under 5 SPL conditions. Loud speech led to increases in lung volume initiation, lower lung volume terminations, higher fundamental frequencies, higher semitone standard deviations, and larger upper and lower lip displacements. Schulman (1989) also noted when sound pressure level increased, there was not only an increase in subglottal pressure, but there was also an increase in the displacement of oral articulators, which resulted in larger lip opening. A more open vocal tract allows a more efficient radiation of acoustic energy because increased mouth opening reduces acoustic radiation impedance during running speech activities (Dromey & Ramig, 1998b; Tasko & McClean, 2004; Hixon et al., 2008).

Maximum Sustained Phonation

From normal loudness to twice normal loudness, it was predicted that mouth opening would increase for the control group during maximum phonation tasks. The control group, on average, slightly decreased area of mouth opening from normal loudness to twice normal loudness during maximum phonation tasks.

Perhaps an increased area of mouth opening was not necessary, given the adjustments they made at the levels of the respiratory and laryngeal systems..

Children with cerebral palsy have difficulty with antagonist co-contraction of the depressor labii inferior and the orbicularis oris superior (Workinger, 2005). Moreover, children with cerebral palsy have reduced range of movement and coordination in speech articulators (Workinger, 2005). Thus, in the current study, it was predicted that mouth opening would not change for the experimental group during maximum phonation tasks produced at twice normal loudness levels.

Contrary to what was expected, children with cerebral palsy increased area of mouth opening from normal loudness to twice normal loudness during maximum phonation tasks. These children also showed a greater increase in mouth opening compared to the typically developing children, as loudness increased. Fine articulatory adjustments were not required during maximum phonation tasks. Perhaps, the simplicity of oral articulator movement required for a maximum phonation task did not tax the motor system and thus allowed the children with CP to coordinate a larger mouth opening to radiate acoustic energy. Increased variability in area of mouth opening, noted in the experimental group, may reflect that the children with CP simply had access to a greater range of performance, as indicated by their greater absolute area of mouth opening.

Sentences

From half normal loudness to four times normal loudness, it was predicted that the control group would increase area of mouth opening. Dromey & Ramig (1998b) noted that upper and lower lip displacements increased in sentences

spoken at louder than normal levels. They suggested that a more open vocal tract allowed for more efficient radiation of acoustic energy and that accordingly, larger articulatory excursions can directly contribute to higher SPL. As expected, the control group increased area of mouth opening as loudness increased.

Children with CP have more difficulty processing increased articulatory demands than typically developing children, which is reflected in greater oromotor variability (Chen, Chen, Hong, Yang, Yang, & Wu, 2010). Deficits in spatial and temporal control may be due to poor motor coordination (Chen et al., 2010). Children with CP also have abnormal oral movement patterns and postures (Workinger, 2005). The speed, range of movement, force, timing, and accuracy of movement in their speech articulators is reduced due to abnormal muscle tone, coordination, strength, and endurance of speech musculature (Workinger, 2005).

From half normal loudness to four times normal loudness, it was predicted that experimental group would not change area of mouth opening. Results were similar to what was expected for this group. On average, the children with CP were highly variable in area of mouth opening adjustments, and as such, a clear trend was not apparent. Two of four participants, with milder CP, exhibited an increase in mouth opening with loudness similar to their control counterparts. The other two participants did not show this pattern and were more severe in general. It could be inferred that participants, who were less severe across the speech mechanism had coordination, strength, and endurance in their speech musculature, allowing them to increase the area of mouth opening with loudness in a coordinated fashion for changes in vocal loudness.

Summary

The main findings from the current study were that typically developing children manipulated lung volume initiations (cm^3) and lung volume excursions (% VC), intercostal and oblique muscle activity (% MVE), speed quotient, fundamental frequency (in Hz and semitones), and area of mouth opening (mean difference from normal in $mm^{2)}$ to adjust loudness in both maximum sustained phonation tasks and sentence tasks. Children with cerebral palsy primarily modified oblique muscle activity (% MVE), fundamental frequency (in Hz and semitones), and area of mouth opening in maximum sustained phonation tasks to adjust for loudness. They mainly regulated lung volume initiations and terminations (cm^3), intercostal and oblique muscle activity (%MVE), speed quotient, and fundamental frequency (in Hz and semitones) to modify loudness in sentence tasks.

The participant with the mildest form of CP performed most similarly to her matched control pair, whereas the participants with the most severe form of CP produced loudness differently from their matched control pairs. One participant with a severe form of CP also indicated cognitive impairment. His data indicated the greatest difference from his match counterpart as well as the other participants in the current study. This suggests that severity of motor dysfunction as well as cognitive ability may influence the observed adjustments for vocal loudness.

It appears that typically developing children in the current study made biomechanically advantageous physiological adjustments in order to increase

loudness. Children with cerebral palsy appeared to rely almost entirely on intercostal muscle activity and increases in fundamental frequency to increase loudness. This is highly taxing on the respiratory musculature and vocal folds. Netsell et al. (1994) noted that some individuals can be taught to inspire to higher lung volume levels, and "let the air out slowly" when speaking, resulting in the maintenance of adequate subglottal air pressures for longer breath groups. This means that excessive and "potentially maladaptive" expiratory muscle forces would not be necessary (Netsell et al., 1994). Yorkston and colleagues (Yorkston, et al., 1999) advocate focusing on maximizing jaw movements in order to increase oral cavity volume and thus, loudness. In order to encourage healthy voice and speech outcomes, informed clinicians should use a model that emphasizes higher lung volume initiation and a larger area of mouth opening, when encouraging children with CP to increase loudness, such as in interventions like *LSVT®LOUD.* Further, it may be that children with cerebral palsy operate within a reduced performance envelope, and such they must be "pushed", through healthy vocal exercise and repetition, to increase the range of their performance envelope.

Limitations

Due to the exploratory nature of the present study, participant numbers were small and thus, the results of this study cannot be generalized to the population of typical children and children with CP. Moreover, the small number of participants did not allow gender and age effects to be accounted for. Further,

the participants with CP presented with highly variable profiles, falling anywhere between level 3 and 5 on the GMF Scale.

Another limitation of the current study was that subglottal pressure could not be measured during the maximum phonation and sentence tasks. Rather, subglottal pressure was estimated from oral pressure and was measured independently during a syllable repetition task (i.e. /pipipi/). It is difficult to estimate subglottal pressure noninvasively for soft phonation because of limitations to vocal motor control near the threshold of soft phonation (Fisher & Swank, 1997). The nose was occluded during all /pipipi/ trials to eliminate nasal airflow, but this may have created compensatory changes in glottal adduction and/or respiratory effort; thus, subglottal pressure estimates may have been altered (Fisher & Swank, 1997). The estimate of subglottal pressure used in this study created a measure under ideal conditions and does not account for potential velopharyngeal inadequacies that may occur during running speech in the present group of children with CP.

Although it is clear that subglottal pressure increased with loudness, it is not certain whether this increase was due to laryngeal activity or to respiratory drive. Laryngeal airway resistance was not measured in this study, and as such it is not possible to determine the relative contributions of the respiratory system and the laryngeal system to the increased subglottal pressure. Finnegan et al. (2000) observed that the primary role of the respiratory system in loudness control was to modulate tracheal pressure and that changes in laryngeal airway resistance played only a small role in loudness modulation in adults.

The interpretation of EGG signals may have been influenced by variable high pass filtering, variations in glottal waveform shape, and the quality of signal that was able to be retrieved (Sapienza et al., 1998). Therefore, experimental error could account for some of the observed variability in the present study.

Pitch was not controlled during loudness adjustments, making it difficult to fully interpret laryngeal measures. Vocal fold behavior varies with both intensity and frequency variations (Woo, 1996).

Finally, the area of mouth opening measure in the experimental group may have been influenced by difficulty managing movement artifacts due to head and trunk control problems and involuntary movements. Whereas tokens with a certain amount of movement artifacts were eliminated from the samples, experimental error may have over or underestimated absolute movements and resulting area measurements.

Future Research

Future studies may address the limitations of this study. Obviously a larger and more homogeneous sample would allow for statistical analysis of these data and greater generalizability. Measures of laryngeal airway resistance, controlling for pitch, and using EGG signals that require minimal high pass filtering would improve the experimental paradigm. A future study may also take measures of lung volume events, respiratory muscle activity, speed quotient, fundamental frequency, and area of mouth opening in children with cerebral palsy, before and following an intervention like *LSVT®LOUD* to determine if healthier speech and voice outcomes are established following treatment.

REFERENCES

Bailey, E., & Hoit, J. (2002). Speaking and breathing in high respiratory drive. *Journal of Speech, Language, and Hearing Research, 45*, 89-99.

Baken, R., & Orlikoff, R. (2000). *Clinical measurement of speech and voice* (2nd ed.) San Diego, CA: Singular.

Baker, K.K., Ramig, L.O., Sapir, S., Luschei, E. S., & Smith, M.E. (2001). Control of vocal loudness in young and old adults. *Journal of Speech, Language, and Hearing Research, 44*, 297-305.

Baumgartner, C.A., Sapir, S., & Ramig, L.O. (2001). Voice quality changes following phonatory-respiratory effort treatment (LSVT) versus respiratory effort treatment for individuals with Parkinson disease. *Journal of Voice, 15*(1), 105-114.

Boersma, Paul & Weenink, David (2010). Praat: doing phonetics by computer [Computer program]. Version 5.2.15, retrieved 01 May 2010 from http://www.praat.org/

Boliek, C., Hixon, T., Watson, P., & Morgan, W. (1996). Vocalization and breathing during the first year of life. *Journal of Voice, 10*, 1-22.

Boliek, C., Hixon, T., Watson, P., & Morgan, W. (1997). Vocalization and breathing during the second and third years of life. *Journal of Voice, 11*, 373-390.

Boliek, C.A., Hixon, T., Watson, P., & Jones, P. (2009). Speech breathing in healthy 4, 5, and 6 year old children. *Journal of Speech, Language and Hearing Research, 52,* 990-1007.

Burke, P. (1980). Serial growth changes in the lips. *British Journal of Orthodontics, 7*(1), 17-30.

Chen, C., Chen, H., Hong, W., Yang, F.G., Yang, L., & Wu, C. (2010). Oromotor variability in children with mild spastic cerebral palsy: a kinematic study of speech motor control. *Journal of NeuroEngineering and Rehabilitation*, *7*(54).

Dromey, C., & Ramig, L.O. (1998a). The effect of lung volume on selected phonatory and articulatory variables. *Journal of Speech, Language, and Hearing Research, 41*, 491-502.

Dromey, C., & Ramig, L.O. (1998b). Intentional changes in sound pressure level and rate: their impact on measures of respiration, phonation, and articulation. *Journal of Speech, Language, and Hearing Research, 41*, 1003-1018.

Finnegan, E.M., Lushei, E.S., & Hoffman, H.T. (2000). Modulations in respiratory and laryngeal activity associated with changes in vocal intensity during speech. *Journal of Speech, Language, and Hearing Speech, 43*, 934-950.

Fisher, K.V., & Swank, P.R. (1997). Estimating Phonation Threshold Pressure. *Journal of Speech, Language & Hearing Research*, *40*(5), 1122-9.

Fox, C.M., Ramig, L.O., Ciucci, M.R., Sapir, S., McFarland, D.H, & Farley, B.G. (2006). The science and practice of LSVT/LOUD: neural plasticity—principled approach to treating individuals with Parkinson disease and other neurological disorders. *Seminars in speech and language, 27*(4), 283-294.

Fox, C.M, & Boliek, C.A. (submitted). Intensive voice treatment [Lee Silverman Voice Treatment (LSVT)] for children with spastic cerebral palsy.

Goldstein, E. M. (2001). Spasticity management: An overview. *Journal of Child Neurology, 16,* 16-23.

Goffman, L., & Smith, A. (1999). Development and differentiation of speech movement patterns. *Human Perception and Performance, 25*, 1-12.

Green, J.R., Moore, C.A, & Reilly, K.J. (2002). The Sequential Development of Jaw and Lip Control for Speech. *Journal of Speech, Language, and Hearing Research, 45*, 66-79.

Gupta, V. B. (2001). Trends in etiology and epidemiology of cerebral palsy. In A. L. Scherzer (Ed.) *Early Diagnosis and Interventional Therapy in Cerebral Palsy: An Interdisciplinary Age-ocused Approach 3rd Edition* (pp. 27-47). New York: Marcel Dekker, Inc.

Heylen, L., Wuyts, F.L., Mertens, F., De Bodt, M., & Van de Heyning, P.H. (2002). Normative Voice Range Profiles of Male and Female Professional Voice Users. *Journal of Voice, 16*(1), 1-7.

Hirano, M., Ohala, J., & Vennard, W. (1969). The function of the laryngeal muscles in regulating fundamental frequency and intensity of phonation. *Journal of Speech and Hearing Research, 12*, 616-628.

Hixon, T. (1973). Respiratory Function in Speech. In F. Minifie, T. Hixon, & F. Williams (Eds). *Normal Aspects of Speech, Hearing, and Language*, 75-125. Englewood-Cliffs, NJ: Prentice-Hall.

Hixon, T.J., Goldman, M.D., Mead, J. (1973). Kinematics of the Chest Wall During Speech Production: Volume Displacements of the Rib Cage, Abdomen, and Lung. *Journal of Speech and Hearing Research*, *16*, 78-115.

Hixon, T.J., Mead, J., & Goldman, M.D. (1976). Dynamics of the Chest Wall During Speech Production: Function of the Thorax, Rib Cage, Diaphragm, and Abdomen. *Journal of Speech and Hearing Research*, *19*, 297-356.

Hixon, T.J., Weismer, G., & Hoit, J.D. (2008). *Preclinical speech science: anatomy physiology acoustics perception*. Plural Publishing: San Diego.

Hodge, M. (2009). *Motor Speech Disorders* [Lecture notes]. Edmonton, Canada: University of Alberta, Department of Speech Pathology and Audiology.

Hodge, M., & Daniels, J. (2004). *TOCS+: a software solution for efficient intelligibility measurement in young children with motor speech disorders*. Presented at the 12th Biennial Conference on Motor Speech, Albuquerque, N.M.

Hoit, J.D., Plassman, B.L., Lansing, R.W., & Hixon, T.J. (1988). Abdominal muscle activity during speech production. *American Physiological Society*.

Hoit, J.D., Hixon, T.J., Watson, P.J., & Morgan, W.J. (1990). Speech breathing in children and adolescents. *Journal of Speech and Hearing Research, 33*, 51-69.

Hollien, H., Green, R., & Massey, K. (1994). Longitudinal research on adolescent voice changes in males. *Journal of the Acoustical Society of America, 34*, 80-84.

Jacques, R.D., Rastatter, M., & Sullivan, J. (1985). Some effects of congenital spasticity on fundamental frequency. *Perceptual and Motor Skills*, *61*, 75-80.

Kahane, J., & Kahn, A. (1984). Weight measurements of infant and adult intrinsic laryngeal muscles. *Folia Phoniatrica, 36*, 129-133.

Kent, R. (1976a). Anatomical and neuromuscular maturation of the speech mechanism: Evidence from acoustic studies. *Journal of Speech and Hearing Research, 19*, 421-447.

Kent, R. (1997). *The speech sciences*. San Diego, CA: Singular.

Kleinow, J., Smith, A., & Ramig, L.O. (2001). Speech motor stability in IPD: effects of rate and loudness manipulations. *Journal of Speech, Language, and Hearing Research, 44,* 1041-1051.

Lab View. [Computer software]. National Instruments, Austin, TX.

Loeb, G.E., & Gans, C. (1986). *Electromyography for Experimentalists*. The University of Chicago Press: Chicago.

Ludlow, C.L. (2005). Central nervous system control of the laryngeal muscles in humans. *Respiratory Physiology & Neurobiology, 147*, 205-222.

Makiyama, K., Yoshihashi, H., Mogitate, M., & Kida, A. (2005). The role of adjustment of expiratory effort in the control of vocal intensity: clinical assessment of phonatory function. *Otolaryngology—Head & Neck Surgery, 132*(4), 641-646

MATLAB. (2010). The MathWorks. [Computer program] Version R2010a, Natick, MA.

McFarland, D.H., & Smith, A. (1992). Effects of vocal task and respiratory phase on prephonatory chest wall movement. *Journal of Speech and Hearing Research, 35* (5).

Mclean, M.D. & Tasko, S.M. (2003). Association of orofacial muscle activity and movement during changes in speech rate and intensity. *Journal of Speech, Language, & Hearing Research, 46*, 1387-1400.

Mutch, L., Alberman, E., Hagberg, B., Kodama, K., & Perat, M. (1992). Cerebral palsy epidemiology: Where are we now, and where are we going. *Journal of Developmental Medicine and Child Neurology, 34*, 547-555.

Netsell, R., Lotz, W.K., Peter, J.E., and Schulte, L. (1994). Developmental Patterns. *Journal of Voice, 8*(2), 123-131.

Palisano, R.J., Cameron, D., Rosenbaum, P.L., Walter, S.D., & Russel, D. (2006). Stability of the Gross Motor Function Classification System. *Developmental Medicine & Child Neurology, 48*, 424-428.

Powerlab [Computer software]. AD Instruments, Inc., Colorado Springs, CO.

Ramig LO., Countryman S., Thompson LL., & Horii Y. (1995). Comparison of two forms of intensive speech treatment for Parkinson disease. *Journal of Speech & Hearing Research, 38*(6),1232-51

Sapir, S., Spielman, J.L., Ramig, L.O., Story, B.H., & Fox, C. (2007). Effects of intensive voice treatment on vowel articulation in dysarthric individuals with idiopathic Parkinson disease: acoustic and perceptual findings. *Journal of Speech, Language, and Hearing Researc, 50*, 899-912.

Schulman, R. (1989). Articulatory dynamics of loud and normal speech. *Journal of the Acoustical Society of America, 85*, 295-312.

Sapienza, C.M., Stathopoulos, E.T., & Dromey, C. (1998). Approximations of open quotient and speed quotient from glottal airflow and EGG waveforms: effects of measurement criteria and sound pressure level. *Journal of Voice, 12*(1), 31-43

Solomon, N.P., Garlitz, S.J., & Milbrath, R.L (2000). Respiratory and Laryngeal Contributions to Maximum Phonation Duration. *Journal of Voice*, *14* (3), 331-340.

Stathopoulos, E.T. & Sapienza, C. (1993). Respiratory and laryngeal function of women and men during vocal intensity variation. *Journal of Speech & Hearing Research, 36*(1).

Stathopoulos, E.T. & Sapienza, C.M. (1997). Developmental changes in laryngeal and respiratory function with variations in sound pressure level. *Journal of Speech, Language, and Hearing Research, 40*, 595-614.

Stradling J, Chadwick G, Quirk C, Phillips T. Respiratory inductance plethysmography: calibration techniques, their validation and the effects of posture. *Bulletin de EuropeanPhysiopathologic Respiratoire* 1985;21:317-24.

Tang, J., & Stathopoulos, E.T. (1995). Vocal efficiency as a function of vocal intensity: a study of children, women, and men. *Acoustical Society of America, 97* (3), 1885-1892.

Tasko, S.M. & McClean, M.D. (2004). Variations in articulatory movement with changes in speech task. *Journal of Speech, Language, & Hearing Research, 47*, 85-100.

Tjaden, K., & Wilding, G.E. (2004). Rate and loudness manipulations in dysarthria: acoustic and perceptual findings. *Journal of Speech, Language, and Hearing Research, 47*, 766-782.

Vorperian, H.K., Kent, R.D., Lindstrom, M.J., Kalina, C.M., Gentry, L.R., and Yandell, B.S. (2005). Development of vocal tract length during early childhood: a magnetic resonance imaging study. *Journal of Acoustical Society of America, 117* (1), 338-350

Watson, H. (1979). The technology of respiratory plethysmography. In: F. Scott (Ed). *Proceedings of the third international symposium on ambulatory monitoring* (pp 537-558). London: Academic Press.

Webber, D. Motion Tracker 2D [Computer software]. University of Pittsburgh, Pittsburgh, PA.

Wit, J., Maassen, B., Gabreels, F., & Thoonen, G. (1993). Maximum performance tests in children with developmental spastic dysarthria. *Journal of Speech and Hearing Research, 36,* 452-459.

Wohlert, A.B. & Hammen, V.L. (2000). Lip muscle activity related to speech rate and loudness. *Journal of Speech, Language, and Hearing Research, 43*, 1229-1239.

Woo, Peak. (1996). Quantification of Videostrobolaryngoscopic Findings: Measurements of the Normal Glottal Cycle. *The Laryngoscope*, *106* (3), 1-27.

Workinger, M. (2005). *Cerebral palsy resource guide for speech language pathologists*. Thomson Delmar Learning.

Yorkston, K. M. (1996). Treatment efficacy: Dysarthria. *Journal of Speech and Hearing Research, 39*, S46-S57.

Yorkston, K.M., Beukelman, D.R., Strand, E.A., & Bell, K.R. (1999) Management of Motor Speech Disorders in Children and Adults. Austin: Pro-ed.

Zemlin, W.R. (1998). Speech and Hearing Science: Anatomy and Physiology (4th edition). Needham Heights, Massachusetts: Allyn & Bacon.

APPENDIX A

Control Group - Participant Recruitment Flyer

Participants needed for a speech study

Healthy boys and girls

Ages: 6 to 12 years

We are interested in understanding how children produce speech at different levels of loudness. We will use this information to help us develop better voice and speech treatment for children who have difficulty speaking because they have cerebral palsy.

If you are interested in learning more about our study, please contact:

Dr. Carol A. Boliek
Edmonton Oilers Community Foundation Speech Research Laboratory
Department of Speech Pathology and Audiology
University of Alberta
780-492-0841
carol.boliek@ualberta.ca

APPENDIX B

Control Group – Information Letter

Project Title:

Respiratory, laryngeal, and articulatory adjustments to changes in vocal loudness in typically developing children and children with spastic-type cerebral palsy

Project Investigators:

Carol A. Boliek, PhD
Erin Archibald, BSc

Purpose of the Study:

Speech problems in children with Cerebral Palsy (CP) can lead to other problems in areas like learning in school, developing good social skills, learning good job skills, and living independently. We do not know very much about how to treat speech problems in children with CP so we need to know more. Therefore, the main goal of this study is to provide speech-language pathologists with information that might help them improve oral communication in children who have CP. The purpose of this study is to learn how the speech mechanism [larynx ("voice box"), lungs, rib cage and stomach muscles, and mouth opening) change when producing sentences produced using different levels of loudness. Your child is being asked to participate in this study because s/he does not have any posture control or speech problems, has normal vision and hearing, and s/he matches the age and sex of one of the children in the study who has CP.

Procedure:

Before we begin the study we will measure your child's height and weight. During this study, we will be measuring breathing patterns and muscle activity of your child while talking. We also measure your child's breathing and muscle activity patterns while they are just listening to their mothers, fathers, or researchers, or looking at picture books. Your child will be asked to speak at a normal level, then at a louder level, then at an even louder level and finally at a soft level. They will be asked to repeat the sentence, "I sell a sapapple again," five times at each loudness level. If at any time, your child becomes tired or hungry, we will take a break and give you and your child as much time as you need. All parts of the experiment will be audio and video-taped so we can look at the information later on after you leave. These sessions will take place at Corbett Hall on the University of Alberta campus.

Breathing measurements. Your child's breathing patterns will be measured by placing cloth bands around the rib cage and stomach. In order to get an idea of how much air is going in and out of the lungs when the rib cage and stomach are moving, we will use a soft face-mask that captures air flowing in and out of your child's nose and mouth. Only a couple of breaths are needed to make this measurement. Another activity will be used to help us understand the relationship between the rib cage and stomach. We will ask older children to play a game where they will pull their tummy in and let it come back out. The investigators will ask younger children to drink water. This naturally causes their stomach to move in and out.

Muscle activity measurements. We will be placing small monitors on the skin at various spots along your child's rib cage, stomach and shoulder. These little monitors will pick up activity from your child's muscles while he/she is playing and talking. These monitors are gentle to the skin and are used routinely in clinics and hospitals. Once we are finished with the monitors, they are thrown away.

Vocal fold measurements. We will be placing a Velcro band around your child's neck just over the "Adam's apple." The band contains sensors which measure vocal fold movement (vocal folds open and close as we speak). This equipment senses changes in an electrical signal when the vocal folds are open compared to when they are closed.

Mouth area. Using washable eyeliner, we will be drawing eight dots on your child's face. A specialized computer program will track the movement of these dots, as your child produces sentences at different loudness levels. This computer program measures the area of your child's mouth opening during the speech tasks.

Video and audio recordings. There will be one small microphone, placed on your child's forehead and on placed on his or her shirt so we can record his or her speech. Two video cameras will be recording the session so we can be sure to eliminate any breathing and muscle activity that happens because of movement. The video camera in front will be recording mouth movement during the speech activities. Audio recordings will help us keep track of how loud your child is speaking and measure the pitch of his or her voice during speaking activities. All video and audio recordings will only be used for the study and will not be used for professional presentations or educational purposes unless we get separate permission from you and your child to do so.

Breathing, muscle activity measurements, vocal fold, and mouth area measurements will be taken all at the same time during your one visit to the speech laboratory. The test session will last about 1.5 hours. We will assist you with parking and pay for your parking fee when you come to the University.

Possible Benefits:

This study has no direct benefit to your child. However, this study will hopefully provide valuable information to speech language pathologists using treatment strategies that focus on encouraging children with CP to speak in a louder voice.

Possible Risks:

There are no known risks associated with participating in this study.

Confidentiality:

Only the people conducting this study will see the information obtained. The Health Research Ethics Board also has the right to access the study records if necessary. We will not give your name to anyone outside of the study. The information you and your child provide will be kept for at least five years after the study is completed. The researchers will store the information in a locked filing cabinet. Your child's name will not be attached to the information you provide your name will not be used in any presentations or publications.

Withdrawal:

Participation is voluntary and your child does not have to take part in this study. If you choose to participate, you may withdraw at any point in the study. You and your child do not need to give a reason.

Contact:

Please be sure to ask the investigators any questions you have now and any time throughout the study. If you have any further questions about this study later on, please contact Dr. Boliek (780-492-0841) or Erin Archibald (eda@ualberta.ca).

If you have any concerns about the conduction of the study, please call Dr. Joanne Volden, Associate Dean, Faculty of Rehabilitation Medicine, University of Alberta, at (780) 492-0651.

Thank you for your time.

APPENDIX C

Control Group – Assent Form

Project Title:

Respiratory, laryngeal, and articulatory adjustments to changes in vocal loudness in typically developing children and children with spastic-type cerebral palsy

Project Investigators:

Carol A. Boliek, PhD
Erin Archibald, BSc

Why have you been asked to do this?

You have been asked to help in a study because you are being matched to a child who has Cerebral Palsy. We want to see how your muscles, voice, and lungs work when you are speaking at different loudness levels.

What will I have to do?

First we will measure your height and weight. We are going to take pictures of how your rib cage and tummy muscles work while you are sitting and talking. We will place 4 sticky metal dots on your tummy and 1 sticky metal dot on your shoulder. These dots will measure how well your muscles are working. When these dots are on your body, we will get you to sit quietly, talk in a normal voice, talk in a louder voice, talk in an even louder voice, and talk in a soft voice. We also will use two video cameras to take pictures of you while you are speaking and two microphones which will record what you say. One microphone will be placed on your forehead and the other will be placed on your shirt.

At the same time we are looking at how your muscles work, we are also going to see how you are breathing. We will place a soft band around your rib cage and another band around your tummy. These will be worn under your shirt and will help us see how much air you use when you are talking. For a very short time, we will also get you to breathe through a small face mask for about 5 breaths.

At the same time we are looking at how you are breathing, we are also going to see how your vocal folds (voice box) work as you are talking at different loudness levels. We will place a Velcro band around your neck right where you can feel the little bump just below your jaw. Two round disks in the band will measure how your vocal folds open and close as you talk.

Using washable eyeliner, we will draw eight dots on your face. We will be using a camera and a computer program to watch the movement of the dots. We will measure how wide your mouth opens when you talk at different loudness levels.

How long will this take?

The total time for the University Visit will be about one and one/half hours. We will ask you to come to the University once.

Will it help?

By helping us out in this study we will learn about how to help children with Cerebral Palsy have better speech. This is important because then they can do better a school and talk to their friends.

Will it hurt?

Nothing we are asking you to do will hurt.

Can you quit?

You don't have to take part in the study at all and you can quit at any time. If you want to quit you should tell the researchers or your parents.

Who will know?

No one except your parents and the researchers will know you are taking part in the study unless you want to tell them. Your name and your information will not be seen by anyone except the researchers during the study.

Your signature:

We would like you to sign this form to show that you agree to take part. Your mom or dad will be asked to sign another form agreeing to for you to take part in the study.

Do you have any questions?

You can ask your mom or dad about anything you don't understand. You can also ask any of the researchers at any time during the study.

I agree to take part in the study.

_____ _____

Signature of Research Participant Date

APPENDIX D

Experimental Group – Information Letter

Project Title:

Respiratory, laryngeal, and articulatory adjustments to changes in vocal loudness in typically developing children and children with spastic-type cerebral palsy

Project Investigators:

Carol A. Boliek, PhD
Erin Archibald, BSc

Purpose of the Study:

Speech problems in children with Cerebral Palsy (CP) can lead to other problems in areas like learning in school, developing good social skills, learning good job skills, and living independently. We do not know very much about how to treat speech problems in children with CP so we need to know more. Therefore, the main goal of this study is to provide speech-language pathologists with information that might help them improve oral communication in children who have CP. The purpose of this study is to learn how the speech mechanism [larynx ("voice box"), lungs, rib cage and stomach muscles, and mouth opening) change when producing sentences produced using different levels of loudness. Your child is being asked to participate in this study because s/he has CP that involves his/her arms, legs, posture and speech.

Procedure:

Before we begin the study we will measure your child's height and weight. We will also do a quick speech test to help us understand the kinds of speech problems your child is having. This will take about 15 minutes. During this study, we will be measuring breathing patterns and muscle activity of your child while talking. We also measure your child's breathing and muscle activity patterns while they are just listening to their mothers, fathers, or researchers, or looking at picture books. Your child will be asked to speak at a normal level, then at a louder level, then at an even louder level and finally at a soft level. They will be asked to repeat the sentence, "I sell a sapapple again," five times at each loudness level. If at any time, your child becomes tired or hungry, we will take a break and give you and your child as much time as you need. All parts of the experiment will be audio and video-taped so we can look at the information later on after you leave. These sessions will take place at Corbett Hall on the University of Alberta campus.

Breathing measurements. Your child's breathing patterns will be measured by placing cloth bands around the rib cage and stomach. In order to get an idea of how much air is going in and out of the lungs when the rib cage and stomach are moving, we will use a soft face-mask that captures air flowing in and out of your child's nose and mouth. Only a couple of breaths are needed to make this measurement. Another activity will be used to help us understand the relationship between the rib cage and stomach. We will ask older children to play a game where they will pull their tummy in and let it come back out. The investigators will ask younger children to drink water. This naturally causes their stomach to move in and out.

Muscle activity measurements. We will be placing small monitors on the skin at various spots along your child's rib cage, stomach and shoulder. These little monitors will pick up activity from your child's muscles while he/she is playing and talking. These monitors are gentle to the skin and are used routinely in clinics and hospitals. Once we are finished with the monitors, they are thrown away.

Vocal fold measurements. We will be placing a Velcro band around your child's neck just over the "adam's apple." The band contains sensors which measure vocal fold movement (vocal folds open and close as we speak). This equipment senses changes in an electrical signal when the vocal folds are open compared to when they are closed.

Mouth area. Using washable eyeliner, we will be drawing eight dots on your child's face. A specialized computer program will track the movement of these dots, as your child produces sentences at different loudness levels. This computer program measures the area of your child's mouth opening during the speech tasks.

Video and audio recordings. There will be one small microphone, placed on your child's forehead and on placed on his or her shirt so we can record his or her speech. Two video cameras will be recording the session so we can be sure to eliminate any breathing and muscle activity that happens because of movement. The video camera in front will be recording mouth movement during the speech activities. Audio recordings will help us keep track of how loud your child is speaking and measure the pitch of his or her voice during speaking activities. All video and audio recordings will only be used for the study and will not be used for professional presentations or educational purposes unless we get separate permission from you and your child to do so.

Breathing, muscle activity measurements, vocal fold, and mouth area measurements will be taken all at the same time during your one visit to the speech laboratory. The test session will last about 2.0 hours. We will assist you with parking and pay for your parking fee when you come to the University.

Possible Benefits:

This study has no direct benefit to your child. However, this study will hopefully provide valuable information to speech language pathologists using treatment strategies that focus on encouraging children with CP to speak in a louder voice.

Possible Risks:

There are no known risks associated with participating in this study.

Confidentiality:

Only the people conducting this study will see the information obtained. The Health Research Ethics Board also has the right to access the study records if necessary. We will not give your name to anyone outside of the study. The information you and your child provide will be kept for at least five years after the study is completed. The researchers will store the information in a locked filing cabinet. Your child's name will not be attached to the information you provide your name will not be used in any presentations or publications.

Withdrawal:

Participation is voluntary and your child does not have to take part in this study. If you choose to participate, you may withdraw at any point in the study. You and your child do not need to give a reason.

Contact:

Please be sure to ask the investigators any questions you have now and any time throughout the study. If you have any further questions about this study later on, please contact Dr. Boliek (780-492-0841) or Erin Archibald (eda@ualberta.ca).

If you have any concerns about the conduction of the study, please call Dr. Joanne Volden, Associate Dean, Faculty of Rehabilitation Medicine, University of Alberta, at (780) 492-0651.

Thank you for your time.

APPENDIX E

Experimental Group – Assent Form

Project Title:

Respiratory, laryngeal, and articulatory adjustments to changes in vocal loudness in typically developing children and children with spastic-type cerebral palsy

Project Investigators:

Carol A. Boliek, PhD & Erin Archibald, BSc

Why have you been asked to do this?

You have been asked to help in a study because you have Cerebral Palsy. We want to see how your muscles, voice, and lungs work when you are speaking at different loudness levels.

What will I have to do?

First we will measure your height and weight and have you give us a sample of your speech. This will take about 15 minutes. We are going to take pictures of how your rib cage and tummy muscles work while you are sitting and talking. We will place 4 sticky metal dots on your tummy and 1 sticky metal dot on your shoulder. These dots will measure how well your muscles are working. When these dots are on your body, we will get you to sit quietly, talk in a normal voice, talk in a louder voice, talk in an even louder voice, and talk in a soft voice. We also will use two video cameras to take pictures of you while you are speaking and two microphones which will record what you say. One microphone will be placed on your forehead and the other will be placed on your shirt.

At the same time we are looking at how your muscles work, we are also going to see how you are breathing. We will place a soft band around your rib cage and another band around your tummy. These will be worn under your shirt and will help us see how much air you use when you are talking. For a very short time, we will also get you to breathe through a small face mask for about 5 breaths.

At the same time we are looking at how you are breathing, we are also going to see how your vocal folds (voice box) work as you are talking at different loudness levels. We will place a Velcro band around your neck right where you can feel the little bump just below your jaw. Two round disks in the band will measure how your vocal folds open and close as you talk.

Using washable eyeliner, we will draw eight dots on your face. We will be using a camera and a computer program to watch the movement of the dots. We will measure how wide your mouth opens when you talk at different loudness levels.

How long will this take?

The total time for the University Visit will be about two hours. We will ask you to come to the University once.

Will it help?

By helping us out in this study we will learn about how to help children with Cerebral Palsy have better speech. This is important because with help they might be able to talk better, do more activities in school and talk to their friends so they can understand them.

Will it hurt?

Nothing we are asking you to do will hurt.

Can you quit?

You don't have to take part in the study at all and you can quit at any time. If you want to quit you should tell the researchers or your parents.

Who will know?

No one except your parents and the researchers will know you are taking part in the study unless you want to tell them. Your name and your information will not be seen by anyone except the researchers during the study.

Your signature:

We would like you to sign this form to show that you agree to take part or you can tell us that you agree if you can't sign your name. Your mom or dad will be asked to sign another form agreeing to for you to take part in the study.

Do you have any questions?
You can ask your mom or dad about anything you don't understand. You can also ask any of the researchers at any time during the study.

I agree to take part in the study.

_____ _____

Signature of Research Participant Date

APPENDIX F

Parent Consent Form

Title of Project:

Respiratory, laryngeal, and articulatory adjustments to changes in vocal loudness in typically developing children and children with spastic-type cerebral palsy

Principal Investigator: *Carol A. Boliek, PhD* Phone Number: *492-0841*
Co-Investigator: *Erin Archibald, BSc* **Email:** eda@ualberta.ca

Do you understand that your child has been asked to participate in a research study? Yes □ No □

Have you read and received a copy of the attached Information Sheet? Yes □ No □

Do you understand the benefits and risks involved in taking part in this research study? Yes □ No □

Have you had an opportunity to ask questions and discuss this study? Yes □ No □

Do you understand that you are free to withdraw your child from the study at any time, without having to give a reason and without affecting your child's future medical care or therapeutic treatment? Yes □ No □

Do you understand that each of the sessions will be videotaped for later data analyses? Yes □ No □

Who explained this study to you? _____
Child's Name _____

I agree for my child to take part in this study: YES □ NO □

Signature of Parent or Guardian _____ Date _____
(Printed Name) _____
Signature of Witness _____ Date _____
Signature of Investigator or Designee _____ Date _____

THE INFORMATION SHEET MUST BE ATTACHED TO THIS CONSENT FORM AND A COPY GIVEN TO THE RESEARCH SUBJECT

APPENDIX G

Gross Motor Function Scale

LEVEL I - Walks without Limitations
LEVEL II - Walks with Limitations
LEVEL III - Walks Using a Hand-Held Mobility Device
LEVEL IV - Self-Mobility with Limitations; May Use Powered Mobility
LEVEL V - Transported in a Manual Wheelchair

APPENDIX H

Groupings Based on Level of Function for Children with Motor Speech Disorders

Group 1: So severely impaired that need augmentative communication to support communication of wants and needs, interaction, and language development

Group 2: Need augmentative communication for interaction and to support language learning; can vocalize and speak well enough to meet basic needs

Group 3: Speak well enough to handle much of communication of wants and needs and a fair amount of interaction; often need augmentative communication to support language learning (new words and combination of words into grammatical utterances)

Group 4: Tend to communicate everything verbally despite obvious MSD. May have an augmentative communication system as a back up to help in repairing communication breakdowns

Group 5: Detectable MSD but speech is intelligible; atypical prosody and/or voice quality and/or slight articulatory imprecision are present

APPENDIX I

Respiratory Subsystem Individual Maximum Sustained Phonation Data

			Control Group		Experimental Group	
			Conditions		Conditions	
Variables:	**Subject**		NL	2X NL	NL	2X NL
Lung Volume Initiation (cc)	F1001	*M*	258.20	361.47	1006.45	967.79
		(sd)	(67.75)	(35.18)	(190.05)	(294.37)
	F1201	*M*	2221.92	2427.89	163.46	143.45
		(sd)	(312.43)	(108.28)	(128.33)	(57.18)
	M0801	*M*	275.18	218.05	182.13	197.67
		(sd)	(18.26)	(0.11)	(69.05)	(64.07)
	M1201	*M*	738.80	841.64	222.13	436.30
		(sd)	(68.79)	(151.74)	(57.97)	(138.54)
Lung Volume Termination (cc)	F1001	*M*	-196.13	-155.60	8.76	33.58
		(sd)	(26.29)	(95.02)	(220.62)	(205.43)
	F1201	*M*	-733.73	-652.17	-236.06	-269.19
		(sd)	(142.08)	(62.72)	(42.28)	(40.30)
	M0801	*M*	-191.26	-239.47	-16.35	-59.68
		(sd)	(85.47)	(124.00)	(46.16)	(69.20)
	M1201	*M*	-213.83	-309.52	-2.57	-98.43
		(sd)	(116.56)	(234.56)	(66.86)	(104.98)
Lung Volume Excursion (%VC)	F1001	*M*	17.08	19.44	42.61	39.90
		(sd)	(3.32)	(2.78)	(2.09)	(7.28)
	F1201	*M*	107.76	112.15	15.59	16.10
		(sd)	(11.32)	(5.17)	(5.63)	(1.24)
	M0801	*M*	22.39	21.96	11.65	15.11
		(sd)	(4.85)	(5.95)	(3.24)	(2.12)
	M1201	*M*	36.48	44.08	6.94	16.52
		(sd)	(3.25)	(4.16)	(0.98)	(3.55)
Intercostal Muscle Activity (%MVE)	F1001	*M*	46.74	53.48	30.09	44.57
		(sd)	(35.50)	(6.25)	(8.98)	(15.28)
	F1201	*M*	48.92	43.94	87.29	63.26
		(sd)	(12.20)	(7.51)	(8.74)	(17.80)
	M0801	*M*	50.15	57.88	30.19	40.36
		(sd)	(6.81)	(17.98)	(28.93)	(6.18)
	M1201	*M*	41.15	59.01	45.93	49.40
		(sd)	(3.96)	(24.85)	(33.13)	(13.09)
Oblique Muscle Activity (%MVE)	F1001	*M*	52.77	46.29	26.02	26.52
		(sd)	(18.77)	(5.69)	(7.87)	(11.74)
	F1201	*M*	29.09	57.07	34.97	39.47
		(sd)	(11.41)	(28.64)	(3.46)	(10.91)
	M0801	*M*	25.23	26.98	44.72	55.13
		(sd)	(6.50)	(14.48)	(4.01)	(9.27)
	M1201	*M*	43.36	44.92	2.95	40.37
		(sd)	(10.17)	(9.25)	(0.69)	(10.64)

APPENDIX J

Respiratory Subsystem Individual Sentence Data

			Control Group				Experimental Group			
			Conditions				Conditions			
Variables:	**Subject**		.5X NL	NL	2X NL	4X NL	.5X NL	NL	2X NL	4X NL
Lung	F1001	*M*	153.87	142.05	195.35	202.00	129.79	394.58	557.95	439.95
Volume		(sd)	(46.35)	(17.85)	(58.39)	(17.34)	(129.12)	(90.26)	(160.65)	(134.80)
Initiation	F1201	*M*	469.16	476.90	778.02	864.74	69.89	89.99	90.58	83.98
(cc)		(sd)	(39.94)	(74.85)	(45.72)	(291.55)	(51.98)	(37.68)	(39.98)	(79.49)
	M0801	*M*	106.50	211.18	249.10	286.62	132.07	61.73	79.60	-47.26
		(sd)	(47.84)	(41.52)	(49.36)	(55.20)	(73.90)	(108.54)	(136.85)	(116.51)
	M1201	*M*	234.07	283.63	331.69	448.62	258.58	171.26	286.52	83.41
		(sd)	(57.45)	(47.15)	(46.58)	(135.99)	(116.20)	(197.71)	(177.93)	(181.33)
Lung	F1001	*M*	7.55	23.03	-1.07	-4.59	-216.44	43.64	49.94	59.62
Volume		(sd)	(10.80)	(16.49)	(11.59)	(9.76)	(44.44)	(90.50)	(123.03)	(83.76)
Termination	F1201	*M*	-37.41	-20.38	20.49	-4.70	-37.01	7.68	-24.34	-90.04
(cc)		(sd)	(24.80)	(44.18)	(116.30)	(77.93)	(38.27)	(43.68)	(39.14)	(31.00)
	M0801	*M*	-73.86	-16.04	-58.36	-36.47	5.81	-68.49	-132.75	-228.25
		(sd)	(50.03)	(24.89)	(67.06)	(59.95)	(86.61)	(114.10)	(93.65)	(86.48)
	M1201	*M*	-68.34	-43.17	-139.12	-69.00	-54.03	-63.36	-2.94	-121.79
		(sd)	(149.15)	(31.51)	(63.01)	(118.57)	(126.45)	(139.63)	(121.20)	(161.49)
Lung	F1001	*M*	5.50	4.47	7.38	7.77	14.79	14.99	21.70	16.24
Volume		(sd)	(1.68)	(0.55)	(2.15)	(0.76)	(5.37)	(2.21)	(4.19)	(3.36)
Excursion	F1201	*M*	18.45	18.11	27.58	31.66	4.17	3.21	4.49	6.79
(%VC)		(sd)	(1.73)	(1.61)	(4.52)	(7.87)	(0.56)	(1.71)	(0.26)	(2.35)
	M0801	*M*	8.66	10.91	14.76	15.51	7.41	7.65	12.47	10.63
		(sd)	(1.11)	(1.74)	(1.24)	(3.90)	(3.35)	(2.03)	(5.23)	(2.94)
	M1201	*M*	11.58	21.18	18.03	19.82	9.66	7.25	8.94	6.34
		(sd)	(4.14)	(1.27)	(3.50)	(3.06)	(2.98)	(3.36)	(6.16)	(5.15)
Intercostal	F1001	*M*	18.15	14.12	18.57	26.89	26.98	32.69	31.19	47.95
Muscle		(sd)	(8.57)	(5.68)	(2.91)	(19.52)	(2.66)	(12.44)	(10.70)	(15.21)
Activity	F1201	*M*	3.75	5.57	5.64	7.40	41.52	31.90	42.38	50.71
(%MVE)		(sd)	(1.49)	(1.10)	(1.20)	(1.87)	(5.36)	(12.41)	(13.62)	(8.75)
	M0801	*M*	17.24	20.92	27.58	38.81	16.52	37.43	46.01	77.53
		(sd)	(4.18)	(8.32)	(5.34)	(4.76)	(8.68)	(15.08)	(11.77)	(14.67)
	M1201	*M*	13.89	13.57	13.04	34.85	46.00	75.22	46.40	58.73
		(sd)	(4.75)	(2.04)	(4.29)	(15.48)	(5.57)	(13.31)	(25.44)	(12.88)
Oblique	F1001	*M*	22.55	16.10	15.88	22.47	24.60	15.53	17.85	28.92
Muscle		(sd)	(10.00)	(5.47)	(4.03)	(14.70)	(5.95)	(2.85)	(4.73)	(5.79)
Activity	F1201	*M*	18.12	5.50	8.79	5.95	13.71	20.51	20.87	37.40
(%MVE)		(sd)	(16.35)	(1.58)	(7.98)	(1.00)	(3.96)	(9.68)	(4.34)	(5.28)
	M0801	*M*	24.24	13.50	22.32	31.56	12.41	40.79	42.31	69.22
		(sd)	(22.69)	(3.00)	(7.45)	(6.65)	(0.11)	(19.79)	(6.13)	(19.27)
	M1201	*M*	18.12	11.16	14.98	36.09	4.12	5.21	2.44	1.04
		(sd)	(16.35)	(15.27)	(6.48)	(14.22)	(5.83)	(6.41)	(2.16)	(0.11)

APPENDIX K

Laryngeal Subsystem Individual Maximum Sustained Phonation Data

			Control Group		Experimental Group	
			Conditions		Conditions	
Variables:	**Subject**		NL	2X NL	NL	2X NL
Speed Quotient	F1001	*M*	0.54	0.44	0.31	0.40
		(sd)	(0.15)	(0.14)	(0.06)	(0.04)
	F1201	*M*	0.47	1.08	1.93	1.09
		(sd)	(0.17)	(0.68)	(0.95)	(0.79)
	M0801	*M*	1.12	3.42	0.50	0.48
		(sd)	(0.37)	(1.10)	(0.20)	(0.17)
	M1201	*M*	0.25	0.48	0.43	0.66
		(sd)	(0.09)	(0.32)	(0.17)	(0.33)
Fundamental Frequency (Hz)	F1001	*M*	189.38	213.11	239.11	265.74
		(sd)	(8.11)	(16.53)	(9.49)	(14.43)
	F1201	*M*	224.96	294.62	323.47	348.64
		(sd)	(5.58)	(4.53)	(2.90)	(41.45)
	M0801	*M*	241.49	311.15	297.99	389.00
		(sd)	(69.39)	(3.36)	(20.30)	(39.22)
	M1201	*M*	230.61	260.67	259.42	334.46
		(sd)	(9.51)	(15.18)	(26.81)	(31.70)
Fundamental Frequency (semitones)	F1001	*M*	1.00	3.04	1.00	2.83
		(sd)	(0.74)	(1.37)	(0.69)	(0.93)
	F1201	*M*	1.00	5.67	1.00	2.30
		(sd)	(0.43)	(0.27)	(0.16)	(2.07)
	M0801	*M*	1.00	5.39	1.00	5.61
		(sd)	(6.00)	(0.19)	(0.45)	(1.78)
	M1201	*M*	1.00	3.12	1.00	5.40
		(sd)	(0.72)	(1.00)	(1.71)	(1.59)

APPENDIX L-1

Laryngeal Subsystem Individual Sentence Data

Speed Quotient

			Control Group				Experimental Group			
			Conditions				Conditions			
Variables:	**Subject**		.5X NL	NL	2X NL	4X NL	.5X NL	NL	2X NL	4X NL
Speed	F1001	*M*	0.54	0.77	1.08	0.53	0.48	0.43	0.28	0.42
Quotient		(sd)	(0.12)	(0.52)	(0.62)	(0.11)	(0.18)	(0.13)	(0.17)	(0.16)
"I"	F1201	*M*	0.72	0.44	0.37	0.51	1.10	0.79	0.66	1.09
		(sd)	(0.41)	(0.04)	(0.16)	(0.30)	(0.32)	(0.16)	(0.18)	(0.57)
	M0801	*M*	0.84	0.79	1.83	1.42	0.81	1.01	0.57	0.84
		(sd)	(1.06)	(0.60)	(1.00)	(0.72)	(0.25)	(0.72)	(0.27)	(0.35)
	M1201	*M*	0.97	0.46	1.47	1.24	0.45	1.11	0.70	0.54
		(sd)	(0.46)	(0.29)	(0.59)	(0.36)	(0.17)	(0.77)	(0.19)	(0.24)
Speed	F1001	*M*	0.82	0.52	0.89	1.63	0.42	0.42	0.38	0.78
Quotient		(sd)	(0.44)	(0.20)	(0.66)	(0.83)	(0.11)	(0.06)	(0.24)	(0.30)
s"a"p	F1201	*M*	0.44	0.24	0.32	0.31	0.69	0.70	0.56	1.40
		(sd)	(0.12)	(0.14)	(0.13)	(0.09)	(0.20)	(0.17)	(0.13)	(0.78)
	M0801	*M*	1.22	1.10	1.43	1.38	0.95	1.26	0.80	1.80
		(sd)	(0.65)	(0.69)	(0.24)	(0.38)	(0.89)	(0.93)	(0.49)	(0.77)
	M1201	*M*	0.42	0.30	0.86	1.10	0.63	0.56	0.47	0.44
		(sd)	(0.12)	(0.08)	(0.65)	(0.43)	(0.20)	(0.14)	(0.15)	(0.06)

APPENDIX L-2

Laryngeal Subsystem Individual Sentence Data

Fundamental Frequency

			Control Group				Experimental Group			
			Conditions				Conditions			
Variables:	**Subject**		.5X NL	NL	2X NL	4X NL	.5X NL	NL	2X NL	4X NL
Fundamental	F1001	*M*	212.05	244.61	265.27	268.44	230.97	262.95	290.80	359.94
Frequency		(sd)	(11.13)	(8.78)	(8.60)	(16.41)	(4.75)	(10.22)	(6.83)	(17.00)
"l" (Hz)	F1201	*M*	239.69	231.18	290.17	371.20	285.60	282.94	288.62	320.24
		(sd)	(16.97)	(18.52)	(14.63)	(44.94)	(5.80)	(6.91)	(9.93)	(18.54)
	M0801	*M*	190.91	214.77	199.36	336.93	239.03	295.45	365.99	381.12
		(sd)	(69.31)	(75.86)	(86.91)	(7.93)	(149.11)	(34.09)	(27.17)	(134.64)
	M1201	*M*	235.46	238.85	289.64	364.68	215.41	256.01	276.73	294.61
		(sd)	(6.65)	(13.65)	(23.13)	(34.05)	(57.34)	(80.36)	(21.93)	(11.55)
Fundamental	F1001	*M*	197.48	190.90	239.81	236.82	189.02	240.96	275.64	357.62
Frequency		(sd)	(3.63)	(48.24)	(9.55)	(7.69)	(48.47)	(17.03)	(13.59)	(14.37)
s"a"p (Hz)	F1201	*M*	218.10	189.64	277.46	347.79	278.91	268.74	248.64	332.81
		(sd)	(7.75)	(49.28)	(5.34)	(28.98)	(4.58)	(4.49)	(65.37)	(34.24)
	M0801	*M*	236.73	212.93	209.64	286.43	307.00	343.13	362.25	450.83
		(sd)	(7.67)	(77.12)	(108.64)	(104.97)	(51.56)	(38.86)	(24.10)	(18.58)
	M1201	*M*	197.06	230.87	246.92	329.29	250.51	271.34	218.62	278.69
		(sd)	(48.60)	(12.63)	(58.88)	(125.47)	(29.41)	(23.58)	(80.07)	(25.63)
Fundamental	F1001	*M*	-1.47	1.00	2.40	2.61	-1.24	1.00	2.74	6.44
Frequency "l"		(sd)	(0.93)	(0.63)	(0.57)	(1.07)	(0.35)	(0.68)	(0.40)	(0.82)
(semitones)	F1201	*M*	1.63	1.00	4.93	9.20	1.16	1.00	1.34	3.14
		(sd)	(1.28)	(1.42)	(0.90)	(1.97)	(0.35)	(0.42)	(0.59)	(1.00)
	M0801	*M*	-1.04	1.00	-0.29	8.80	-2.67	1.00	4.71	5.41
		(sd)	(7.33)	(7.04)	(6.99)	(0.41)	(11.59)	(2.12)	(1.26)	(8.59)
	M1201	*M*	0.75	1.00	4.34	8.33	-1.99	1.00	2.35	3.43
		(sd)	(0.49)	(0.98)	(1.38)	(1.63)	(5.84)	(6.61)	(1.38)	(0.67)
Fundamental	F1001	*M*	1.59	1.00	4.95	4.73	-3.20	1.00	3.33	7.84
Frequency		(sd)	(0.32)	(5.44)	(0.69)	(0.57)	(5.56)	(1.23)	(0.84)	(0.69)
s"a"p	F1201	*M*	3.43	1.00	7.59	11.50	1.64	1.00	-0.35	4.70
(semitones)		(sd)	(0.63)	(5.66)	(0.33)	(1.43)	(0.28)	(0.29)	(5.73)	(1.74)
	M0801	*M*	2.83	1.00	0.73	6.13	-0.93	1.00	1.94	5.73
		(sd)	(0.56)	(7.31)	(8.24)	(7.41)	(2.80)	(2.09)	(1.18)	(0.72)
	M1201	*M*	-1.74	1.00	2.16	7.15	-0.38	1.00	-2.74	1.46
		(sd)	(5.30)	(0.95)	(4.98)	(9.78)	(2.12)	(1.52)	(7.09)	(1.55)

APPENDIX M

Oroarticulatory Subsystem Individual Maximum Sustained Phonation Data

			Control Group		Experimental Group	
			Conditions		Conditions	
Variables:	**Subject**		NL	2X NL	NL	2X NL
Area of Mouth Opening (mm^2)	F1001	*M*	0.00	409.25	0.00	-143.59
		(sd)	(239.74)	(162.03)	(150.27)	(62.52)
	F1201	*M*	0.00	-79.37	0.00	-323.20
		(sd)	(142.58)	(227.33)	(87.82)	(226.55)
	M0801	*M*	0.00	-277.88	0.00	-1135.97
		(sd)	(50.13)	(130.47)	(795.35)	(1242.06)
	M1201	*M*	0.00	226.76	0.00	232.28
		(sd)	(81.45)	(329.30)	(865.55)	(510.38)

APPENDIX N

Oroarticulatory Subsystem Individual Sentence Data

			Control Group				Experimental Group			
			Conditions				Conditions			
Variables:	**Subject**		.5X NL	NL	2X NL	4X NL	.5X NL	NL	2X NL	4X NL
Area of Mouth Opening (mm^2)	F1001	*M*	181.78	0.00	134.53	-230.82	232.05	0.00	-337.73	-748.05
		(sd)	(123.76)	(255.86)	(395.31)	(167.90)	(131.78)	(39.70)	(60.59)	(354.21)
	F1201	*M*	63.93	0.00	-299.31	-396.82	41.77	0.00	-62.40	-220.07
		(sd)	(61.15)	(60.57)	(83.74)	(46.98)	(51.12)	(60.47)	(52.70)	(79.13)
	M0801	*M*	2.09	0.00	-95.38	-193.30	-526.55	0.00	424.93	925.40
		(sd)	(104.24)	(45.83)	(72.82)	(58.04)	(320.98)	(1611.87)	(331.32)	(261.28)
	M1201	*M*	108.06	0.00	-159.04	-286.04	957.97	0.00	214.50	365.74
		(sd)	(45.72)	(20.40)	(35.61)	(60.64)	(166.60)	(796.04)	(658.61)	(601.45)