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Real-time continuous visual biofeedback in the treatment of speech breathing disorders following childhood traumatic brain injury: report of one case

BRUCE E. MURDOCH, GAIL PITT, DEBORAH G. THEODOROS and ELIZABETH C. WARD

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Summary

The efficacy of traditional and physiological biofeedback methods for modifying abnormal speech breathing patterns was investigated in a child with persistent dysarthria following severe traumatic brain injury (TBI). An A-B-A-B single-subject experimental research design was utilized to provide the subject with two exclusive periods of therapy for speech breathing, based on traditional therapy techniques and physiological biofeedback methods, respectively. Traditional therapy techniques included establishing optimal posture for speech breathing, explanation of the movement of the respiratory muscles, and a hierarchy of non-speech and speech tasks focusing on establishing an appropriate level of sub-glottal air pressure, and improving the subject's control of inhalation and exhalation. The biofeedback phase of therapy utilized variable inductance plethysmography (or Respitrace) to provide real-time, continuous visual biofeedback of ribcage circumference during breathing. As in traditional therapy, a hierarchy of non-speech and speech tasks were devised to improve the subject's control of his respiratory pattern. Throughout the project, the subject's respiratory support for speech was assessed both instrumentally and perceptually. Instrumental assessment included kinematic and spirometric measures, and perceptual assessment included the Frenchay Dysarthria Assessment, Assessment of Intelligibility of Dysarthric Speech, and analysis of a speech sample. The results of the study demonstrated that real-time continuous visual biofeedback techniques for modifying speech breathing patterns were not only effective, but superior to the traditional therapy techniques for modifying abnormal speech breathing patterns in a child with persistent dysarthria following severe TBI. These results show that physiological biofeedback techniques are potentially useful clinical tools for the remediation of speech breathing impairment in the paediatric dysarthric population.

Introduction

With recent improvements in post-trauma medical care, an increasing number of children with severe traumatic brain injury (TBI) are surviving [1]. Due to the diffuse nature and severity of brain damage in these children, they are likely to present with persistent neurological sequelae. Research indicates that dysarthria, a neuromotor speech disorder, is one of the most persistent neurological sequelae of particularly severe TBI, often remaining beyond the acute stage of recovery and the return of motor and language function [1–9]. Little research, however, has addressed the efficacy of the rehabilitation of dysarthria in this population [1].

The primary mechanism of brain damage in TBI is diffuse cerebral injury, produced by shearing at the time of impact [4, 10]. As a consequence of the diffuse injury, dysarthria in TBI patients is likely to be variable in terms of type, severity, and the sub-systems of speech production involved, depending on the site and extent of brain damage. Yorkston *et al.* [11] suggested that the dysarthria is unlikely to present in a pure form, indicating the difficulty in determining the underlying neurological impairment in these subjects.

One component of the speech production mechanism, however, that has been found to be disordered in the severely TBI adult population is breath support for speech [8, 12]. There have been no studies to date documenting the degree of involvement of the respiratory sub-system in children with dysarthria following TBI,

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Authors: Bruce E. Murdoch (author for correspondence), Gail Pitt, Deborah G. Theodoros and Elizabeth C. Ward, Motor Speech Research Unit, The Department of Speech Pathology & Audiology, The University of Queensland, Brisbane 4072, Australia.

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however, some studies have been conducted in the adult TBI population by Murdoch *et al.* [12] and Theodoros *et al.* [8]. Theodoros *et al.* [8] studied the perceptual speech characteristics of dysarthric speakers following severe TBI and reported that, of the 20 adults in their study, deviation in breath support for speech was perceived by trained judges in 85% . There were also high incidences of phrase length deviation and phonatory disturbance (e.g. variation of loudness, loudness level, maintenance of loudness) for which breath support could be the underlying cause.

Murdoch *et al.* [12] performed kinematic and spirometric assessments on 20 adult TBI subjects and 20 non-neurological impaired controls. They reported that the TBI subjects exhibited decreased vital capacities and forced expiratory volumes, and impaired two-part coordination of the chest wall during speech production (i.e. coordination of the ribcage and abdomen during inspiration and expiration).

These findings suggest a pattern of speech breathing impairment in severe TBI adults presenting with persistent dysarthria. Since the production of a good quality voice, correct phoneme production, and good intonation, stressing, rhythm and phrasing rely on adequate supply and control of expiration [13, 14], any impairment in breath support for speech should be a priority in speech rehabilitation. There have been no studies, however, detailing the degree of involvement of the respiratory sub-system of the speech production mechanism in children with dysarthria following TBI. The evidence in the literature for persisting dysarthria in the paediatric TBI population [2, 3, 6, 15, 16] and reports from some researchers that dysarthria in adults and children is clinically similar [2, 6] indicate the need for similar studies in the paediatric TBI population.

Traditionally, rehabilitation of dysarthria involved the development of treatment programmes based primarily on assessment of the perceptual dimensions of motor speech production, from which the clinician inferred the presence of physiological impairment. In the last few decades, however, the need for a more objective approach has seen the development of the physiological approach to rehabilitation of dysarthria. The physiological approach, based on a neurobiologic view of speech production, involves assessing the components or sub-systems of the speech production mechanism individually, and in controlled combinations, to determine the underlying patho-physiology. The primary purpose of physiological examination is to determine the extent and type of dysfunction in each of the components or sub-systems of the speech production mechanism utilizing objective, instrumental measurement techniques. Based on the results of this comprehensive physiological examination, therapeutic targets can be identified and prioritized based on the severity, neuropathology, and dependency of a component or sub-system of the speech production mechanism on the functioning of another component [13].

Traditional behavioural methods for the treatment of speech breathing in dysarthria have been well documented [11, 14, 17, 18]. Traditional therapy techniques utilize visual, auditory, kinaesthetic, and tactile feedback to improve the strength and coordination of the speech breathing musculature, increase vital capacity, and most importantly to establish a controlled exhalation [14].

Although traditional, behavioural techniques have been proven effective in the treatment of some dysarthric subjects [18, 19], they are limited by the nature of the feedback they provide. Recent research into the effect of feedback on perceptual-motor learning indicates superior performance for objective feedback that is both immediate and continuous. The feedback provided in traditional therapy, however, is largely subjective, often delayed, and non-continuous.

The feedback available in traditional techniques, therefore, allows the possibility of misinterpretation of the underlying motor performance in subjects with speech breathing impairment, and does not provide the most efficient information for perceptual-motor learning. In the rehabilitation of speech breathing impairment in subjects with dysarthria, therefore, a need exists for objective, immediate, and continuous feedback of motor performance of the respiratory mechanism.

Several physiological biofeedback techniques have been used in the rehabilitation of respiration in the adult dysarthric population. Some researchers have used pressure transducers coupled with oscilloscopes to provide visual feedback of subglottal air pressure to dysarthric subjects [20–23]. These studies have shown improved respiratory support for speech in several subjects with dysarthria following CVA or TBI.

Improving respiratory support for speech, however, is not always facilitated by biofeedback of sub-glottal air pressure. Research has shown that subjects with dysarthria (especially mixed dysarthria) following severe TBI have difficulty using their respiratory musculature most effectively. Physiologic assessment of 20 subjects with dysarthria following severe TBI indicated that this population had problems coordinating ribcage and abdomen movement during speech [12]. Consequently, more recent rehabilitation for respiration in dysarthric subjects has focused on providing biofeedback of the excursion of the ribcage and abdominal muscles during speech and non-speech tasks.

Murdoch *et al.* [24] used strain gauge belt pneumographs coupled with an oscilloscope to provide visual biofeedback of ribcage and abdominal excursions to two subjects with dysarthria following severe TBI. Thompson-Ward *et al.* [25] used the same system to provide biofeedback to a subject with dysarthria following a CVA. In both studies the subjects received visual biofeedback of their abdominal and ribcage displacement on *x* and *y* axes respectively. All subjects were able to use biofeedback to alter their paradoxical breathing patterns to attain coordinated ribcage–abdominal speech breathing.

Yorkston *et al.* [11] used a more advanced system of measuring ribcage and abdominal excursion called Respiratory Inductive Plethysmography (or 'Respitrace') in a subject with Amyotrophic lateral sclerosis. Using this system, Yorkston *et al.* [11] trained their subject in two sessions to increase their abdominal contribution and decrease paradoxical abdominal movement during inspiration. This resulted in greater vocal loudness and less fatigue during speech.

All of the above biofeedback research has involved adult subjects with dysarthria, little research in this area having been carried out using paediatric cases. The few pilot studies that have been published, however, suggest that biofeedback techniques may be used successfully to rehabilitate various speech impairments in children [26–31]. There have been no published studies investigating the efficacy of physiological biofeedback techniques in the rehabilitation of speech breathing in children with dysarthria following TBI.

The aim of the present study, therefore, was to compare traditional methods and physiological biofeedback methods for modifying abnormal speech breathing patterns in a child with dysarthria following severe TBI, with a view to improving respiratory support for speech.

Methods

SUBJECT

The subject was a 12-year-old male with persistent dysarthria subsequent to a severe TBI incurred approximately 2.5 years prior to his participation in the study. Selection criteria included: a clinical diagnosis of severe TBI (GCS < 8), as determined by a qualified neurologist; diagnosis of dysarthria established by a qualified speech pathologist; subject and parental enthusiasm for intervention; stabilized and persistent dysarthria determined by a post-onset time of at least 12 months; and a negative history of dysarthria or neurological impairment prior to TBI.

The subject, who was right handed, had sustained a severe TBI in a motor vehicle accident as a bicyclist. His Glasgow Coma Score (GCS) was 3–4 on admission to hospital. Computerized tomography results revealed a major intra-cerebral haematoma in the left parietal lobe. A right frontal external ventricular drain was inserted to monitor intra-cranial pressure. The subject was in the intensive care unit for approximately 3 weeks and remained in hospital for 3 months.

The subject was discharged, at the parents' request, 3 months post-TBI. Following discharge he has received weekly physiotherapy, occupational therapy, and speech therapy until the time of his participation in the present study. Speech therapy consisted of both articulation and language programmes. At the time of inclusion in the present study, the subject had a persistent right hemiparesis and was wheel-chair dependent for mobility.

PROCEDURE

Assessment of speech production

Initially the subject was administered a comprehensive physiological and perceptual assessment of his speech production mechanism in order to establish the specific nature of his impairments and identify appropriate treatment targets.

Procedure for physiological assessment

The respiratory, laryngeal, velo-pharyngeal, and articulatory function of the subject was assessed instrumentally using a comprehensive battery of physiological techniques. The subject's respiratory function was assessed using spirometric techniques described by Murdoch *et al.* [12], kinematic techniques used by Murdoch *et al.* [24], and aerodynamic techniques outlined by Theodoros and Murdoch [32]. A hand held Minjhardt dry spirometer was used to measure vital capacity and forced expiratory volume 1 second. Variable inductance plethysmography (Respitrace) was used to determine the relative contribution of the ribcage and abdomen, lung volume initiation and termination levels, the incidence of slope changes and paradoxical chest wall movements during performance of a number of speech and non-speech tasks. In addition, mean syllables per breath, speaking rate, and voice onset and offset latencies were also determined using a throat microphone in combination with the Respitrace system during performance of a range of speech tasks. An Aerophone

II 6800 (Kay Elemetrics) airflow measurement system was used to evaluate sub-glottal air pressure.

The subject's laryngeal function was assessed using electroglottography (Kay Elemetrics laryngograph with waveform display system, Model 6091) to measure vocal fold vibration, and the Aerophone II (Kay Elemetrics, Model 6800) to measure laryngeal airflow. For a full description of these procedures see Theordoros and Murdoch [32]. Vocal fold vibration measurements included fundamental frequency (F_0), duty cycle, closing time, and adduction-abduction rate. Laryngeal airflow measurements included sound pressure level, glottal resistance, and phonatory flow rate. The subject's velopharyngeal function was assessed using a nasal accelerometer, according to the procedure outlined by Theodoros *et al.* [33], for measurement of nasality (the Horii Oral Nasal Coupling Index).

The subject's articulatory function was assessed using strain-gauge and pressure transduction systems to measure tongue and lip strength, endurance and rate of repetitive movements. The rubber-bulb pressure transducer used to assess tongue function was identical to that used by Murdoch *et al.* [34] and the miniaturized pressure transducer based on semi-conductor strain-gauge technology used to assess lip function was similar to that described by Hinton and Luschei [35]. For complete details of the instrumentation and procedures used to test articulatory function see Horton *et al.* [36].

The results of physiological assessment were compared to those of a control subject, matched for age and sex, and used to obtain a physiological profile of the subject's motor speech mechanism revealing impairments in all sub-systems, but most noticeably, a severe impairment of the subject's ability to control his expiratory airflow at all levels of his speech production mechanism.

Procedure for perceptual assessment

Perceptual assessment of the subject's speech production mechanism involved the administration of the Frenchay Dysarthria Assessment [37], to determine the type and severity of dysarthria, and the Assessment of Intelligibility of Dysarthric Speech [38] for levels of word and sentence intelligibility. These assessments were scored according to the instruction manual. In addition the subject was required to read a standard passage, 'The Grandfather Passage' [39], to obtain a tape recorded speech sample for perceptual analysis of respiration, phonation, resonance, articulation, and prosody, as used by FitzGerald *et al.* [40].

The results of perceptual assessment were used to construct a perceptual profile of the subject which demonstrated impairment in all sub-systems of the speech production mechanism, with the respiratory and laryngeal sub-systems perceived as the most severely impaired. The subject's intelligibility was rated as being profoundly impaired.

Identification of therapy goals

The subject's physiological and perceptual profiles revealed a pattern of motor speech impairment consistent with a mixed spastic-ataxic-flaccid dysarthria. On the basis of the subject's performance on the battery of physiological and perceptual tests, it was determined that the respiratory sub-system of the speech production mechanism was a major contributor to the overall speech deficit and, therefore, would be the initial focus of therapy. Specifically, increasing the subject's control of inhalation and exhalation, and improving his coordination of phonation and exhalation, were established as the main goals of therapy.

Research design

The effects of traditional and physiological biofeedback therapy on the breath support for speech of a subject with dysarthria following severe TBI was determined using an A-B-A-B single-subject experimental research design, involving baseline assessments (A_1); traditional therapy (B_1); a withdrawal phase (A_2); and physiological biofeedback therapy (B_2).

BASELINE PHASE (A_1)

The baseline phase consisted of six instrumental assessments of speech breathing obtained over 2 days, and two perceptual assessments administered at the beginning and end of the baseline phase. Physiological instrumental assessment of speech breathing involved both kinematic and spirometric assessments, of approximately 30 minutes duration.

Kinematic assessment

Kinematic assessment involved recording the changes in circumference of the ribcage and abdomen during speech and non-speech tasks. Variable inductance plethysmography was used to measure ribcage and abdominal circumference. This system utilizes a pair of elastic straps containing embedded coils of electrical wire which are positioned around the subject's chest and abdomen. The inductance of the wire changes with the circumference of the chest wall and, therefore, is the analogue of chest wall displacement. The signal passes through an oscillator positioned on the subject and demodulating circuitry to produce a single voltage output corresponding to the displacement of the ribcage and abdomen.

Signals from the ribcage and abdomen inductive transducers were amplified by a DC amplifier and passed simultaneously to two separate recording and storage instruments. Outputs from the ribcage and abdomen inductive transducers were displayed on a computer screen (IBM-486) via a physiological data acquisition system (ASYSTANT PLUS) to yield a relative volume chart, with the ribcage on the *y*-axis and the abdomen on the *x*-axis. The outputs from the ribcage and abdominal inductive transducers were independently recorded in one channel of a four-channel Y-T oscillographic recorder (Bioscience Washington Model MD4). The X-Y outputs were recorded on floppy disc for later analysis. During the reading task of the kinematic assessment the subject's speech was recorded onto audiotape using a high-quality tape recorder (Marantz Model CP430) and microphone (Sony ECM-30 Electret Condenser Microphone).

A miniature accelerometer (Knowles Electronics Model BU-1771) was used to detect vocal fold vibrations during speech simultaneously to ribcage and abdomen movement recordings. It was positioned on the lamina of the thyroid cartilage and attached with double-sided adhesive tape. The accelerometer consisted of a ceramic vibration transducer complete with an amplifier stage. The frequency response of the accelerometer ranged from 20–4000 Hz. The output from the accelerometer was recorded on one channel of the oscillographic recorder simultaneously with the outputs from the inductive transducers to allow comparison of voice onset and offset with respiratory movements.

Kinematic measures were made with the subject seated in his wheelchair. The subject sat with his back to the visual display unit of the computer. The two elasticized inductive transducer straps were positioned around the ribcage and abdomen and fastened anteriorly with velcro fittings. The inductive transducer strap for recording circumferential changes of the ribcage was positioned midway between the supra-sternal notch and the xiphoid process. The strap for recording circumferential changes of the abdomen was placed around the abdomen at the level of the umbilicus and below the level of the coastal margin to avoid ribcage movement contamination. The subject was required to avoid changes in body positioning during assessment recordings and the elasticized straps were monitored visually during the recordings to ensure strap slippage did not occur.

Measurements were recorded during non-speech and speech tasks. Recordings of quiet and deep breathing were made while the subject breathed with no equipment at the airway. The subject's manipulable range of lung volumes was defined by vital capacity manoeuvres. For the vital capacity (VC) manoeuvre, the subject inspired fully from the resting-end expiratory level (REL) and then expired fully whilst wearing a noseclip and breathing into a spirometer (Mijnhardt Vicatest-P1).

Speech tasks for kinematic evaluation included sustained vowel productions, syllable repetition tasks, a counting task, reading of a declarative passage, and spontaneous conversation. Measurements were taken during the prolongation of the vowels /a/, /i/ and /u/, where the subject was instructed to inspire to total lung capacity and sustain the production of the vowel on a single expiration. The syllable repetition tasks included /pa/, /ta/ and /pataka/. The specific instructions for this task were 'Take a deep breath and say /pa/ until you run out of breath'. The task was demonstrated at a rate of approximately three syllables per second. The counting task required the subject to 'Take a deep breath and count for as long as you can' at a rate of approximately one number per second. The subject was required to read 'The Grandfather Passage' [39] with the instructions 'Read this paragraph out aloud so that a person could hear you across the room'. The passage was provided in large print and held at a comfortable reading distance from the subject. The final speech task consisted of recording spontaneous conversation with the subject. The subject was unaware of the recordings during this period of assessment.

Spirometric assessment

Spirometric assessment was conducted using a Mijnhardt Vicatest-P1 spirometer, comprising a digital volume transducer coupled with a microprocessor, which calculated the values for the subject's vital capacity (VC) and forced expiratory volume in 1 second (FEV_1). For VC, the subject was instructed to 'Take as deep a breath as you can, then let it all out until there is nothing left in your lungs'. For FEV_1 the subject was instructed to 'Take as deep a breath as you can, then let it all out as fast as you can'. Both spirometric tasks were performed with a noseclip in place and a firm lip seal around the cardboard tubing to avoid air leakage.

Perceptual assessment

The Frenchay Dysarthria Assessment (FDA) [37] was administered as a standardized measure of the type and severity of dysarthria indicating functioning of the different sub-systems of the speech production mechanism. The FDA was administered at the beginning and end of the baseline phase.

The subject was required to read a standard passage, 'The Grandfather Passage' [39] to obtain a sample of his speech. The subject was instructed to speak in his natural manner, using a normal speaking rate, with a loudness level appropriate for speaking to someone across the room. The speech sample was recorded onto audiotape using a Marantz Portable Cassette Recorder (Model no. PMD222). The microphone was located 20 cm from the subject's mouth.

The Assessment of Intelligibility of Dysarthric Speech (ASSIDS) [38] assessment provides an index of severity of dysarthric speech by quantifying both single word and sentence intelligibility of adult dysarthric speakers. The ASSIDS was administered according to the procedure specified in the test manual. The test involved reading or repeating 50 randomly selected single words and repeating 22 randomly selected sentences, ranging in length from five to 15 words. All speech assessments were administered under standard conditions in quiet surroundings, and were approximately 45 minutes in duration.

TRADITIONAL THERAPY (B_1)

The subject received eight 30–45 minute sessions of traditional therapy for speech breathing across 2 weeks. The aim of therapy was to increase the subject's respiratory support for speech by establishing a controlled inhalation and controlled steady exhalation. A number of traditional therapy techniques were utilized in the attempt to achieve this aim.

Instruction in improved posture and deeper, controlled inhalations (not maximal inhalation) during non-speech tasks were used to increase steadily the volume of inhalation. Each session began with instruction in good posture for optimal breathing. Establishing adequate control of sub-glottal air pressure for speech and techniques to increase the control of exhalation were the main focus of this phase.

A 'home-made glass and straw' U-tube water manometer, as described by Yorkston *et al.* [11] was used to increase the subject's control of subglottal air pressure, providing visual feedback of performance. The subject was required to generate 5 cm H_2O for increasing periods of time, and the duration was recorded for each attempt during all sessions. Once the subject had reached the criterion of generation 5 cm H_2O for 5 seconds across 10 consecutive trials, the subject was provided with a description and demonstration of the breathing pattern for speech (i.e. quick inhalation, followed by a slow, controlled exhalation, as described by Yorkston *et al.* [11]).

An hierarchy of speech tasks was devised, with set criteria for progressing through each level. These included vowel (/a/, /i/, /u/) prolongation, syllable repetition (/pa/, /pi/, /ta/), serial speech tasks (e.g. counting, reciting alphabet), and reciting of nursery rhymes. Due to the severity of the subject's speech breathing impairment, however, the criterion for syllable repetition was not met and, therefore, the majority of sessions focused on the non-speech tasks and the vowel prolongation speech task. Feedback was provided by the therapist and included information concerning the duration of tasks (in seconds), and subjective feedback of the depth and speed of inhalation, voice onset latency (i.e. the time delay between the beginning of exhalation and the beginning of phonation), and control of exhalation.

In order to monitor the effect of therapy on speech breathing, therapy sessions on days 2, 4, 5, 6 and 8 were followed by a 30 minute instrumental assessment of speech breathing and a recording of a standard passage for speech analysis. Spirometric recordings were taken during instrumental assessments on days 2, 4, 5, 6 and 8. The other perceptual assessments, the FDA and the ASSIDS, were administered prior to therapy on day 1 and following therapy on day 8.

WITHDRAWAL PHASE (A_2)

Following the 2 weeks of traditional therapy, a period of 10 weeks elapsed during which the subject received no therapy. Six assessments were administered during this time in weeks 1, 3, 4, 5, 7 and 9. Each assessment included both instrumental and perceptual evaluations and was approximately 1 hour in duration.

BIOFEEDBACK THERAPY (B_2)

Following the 10 week withdrawal period, the subject received eight sessions of biofeedback therapy for speech breathing over 2 weeks using the 'Respitrace' system. The aim of biofeedback therapy was to improve the subject's respiratory support for speech by increasing his control of inhalation and exhalation utilizing real-time visual biofeedback of chest wall movements during breathing, as displayed on the Y-T strip chart recorder

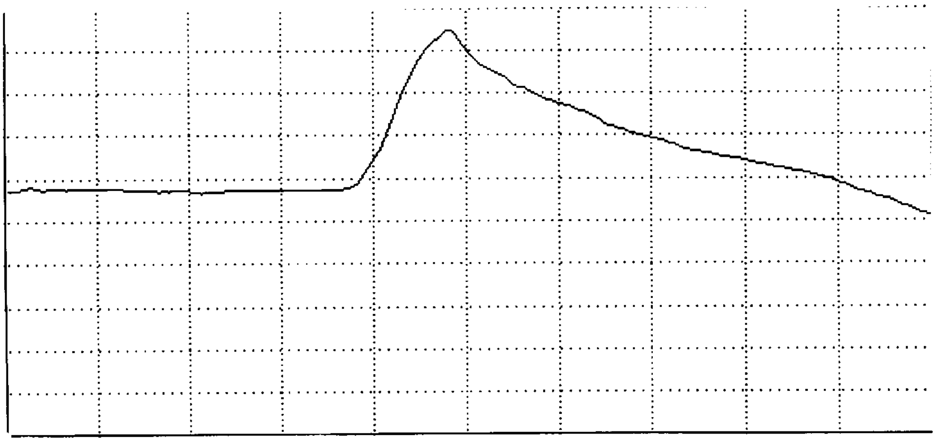

Figure 1 Schematic representation of the Y-T strip chart recorder visual biofeedback target trace.

mode of the ASYSTANT PLUS program. Visual biofeedback of ribcage movement during breathing was chosen as the most suitable feedback dimension due to the subject's inability to coordinate his abdominal musculature.

Each session was approximately 30 minutes in length and began by establishing good posture for optimal breathing. The subject was required to match a target trace provided at the top of the computer screen whilst performing various non-speech and speech tasks. The target was chosen because it represented normal use of the ribcage during maximal breathing tasks (see figure 1). A hierarchy of tasks was devised including quiet breathing, deep breathing, prolonged vowels (/a/ /i/, /u/), syllable repetition (/pa/, /pi/), serial speech tasks (e.g. counting, alphabet), and reading of phrases and sentences of increasing length (e.g. 3–5 syllables per breath). As in the traditional therapy phase, however, due to the severity of the subject's speech breathing impairment, he was unable to reach the criterion for syllable repetition. The majority of sessions, therefore, were focused on deep breathing and vowel prolongation.

Establishing appropriate coordination between phonation and respiration, as measured by voice onset latency, was a major focus of therapy during this period. The visual biofeedback of ribcage circumference enable the subject to 'see' the point at which inspiration ended and expiration began and, therefore, attempt to coordinate phonation with this point. During the biofeedback sessions the therapist operated the computer, explained the visual biofeedback to the subject, and instructed the subject to make the necessary adjustments to his breathing pattern and voice onset coordination with expiration.

Prior to therapy on days 1, 3, 4, 6, 8 and 9, and after therapy on day 10, instrumental assessments were administered and a standard passage of reading was recorded. Spirometric recordings were taken during instrumental assessment on days 1, 3, 6, 8, 9 and 10. The other perceptual assessments were administered prior to therapy on day 1 and after therapy on day 10.

DATA ANALYSIS

Kinematic analysis

The outcome measures chosen for kinematic analysis were phonation time, voice onset latency, and the degree of abdominal and ribcage paradoxing, calculated on the vowel prolongation speech task. The variables were chosen because they were considered to best reflect the abnormal aspects of speech breathing identified in this subject.

Phonation time and voice onset latency

A major focus of both therapy phases was to improve the subject's expiratory control for speech by increasing the duration of phonation and decreasing the amount of air wastage prior to the onset of phonation. Phonation time and voice onset latency, therefore, were calculated from analysis of vowel prolongation tasks.

For the vowel prolongation speech task, measurements were taken from the three Y-T oscilloscopic recordings obtained during the prolongation of /a/, and each of the single recordings obtained during the prolongation of /i/ and /u/. The prolongation of /a/ was selected to be analysed, as the recordings obtained during this task were considered to be representative of all

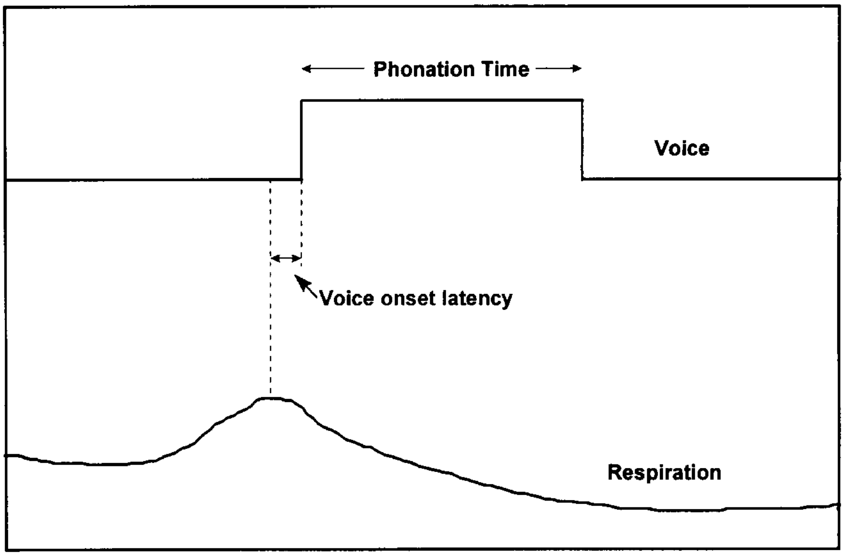

Figure 2 Schematic representation of the Y-T strip chart recorder trace showing method of calculation of phonation time and voice onset latency.

other vowel prolongation tasks. In total, there were 72 Y-T oscilloscopic recordings obtained during prolongation of /a/. Ten of these were excluded from analysis due to breaks in phonation. The remaining 62 recordings were then analysed.

The four-channel Y-T oscilloscopic recordings were analysed for length of phonation and voice onset latency for the vowel prolongation speech task. The duration of phonation was determined by simply measuring the width of vowel prolongations on the channel recording accelerometer output (see figure 2). As the chart paper was set to a constant speed of 1 mm per second, the width of the recording in millimetres was equivalent to the duration in seconds of vowel prolongation. Voice onset latency was determined by comparing the time difference between the beginning of the expiratory phase, measured on the channel recording ribcage excursion, with the beginning of phonation, measured on the channel recording accelerometer output. Again, the time delay between the beginning of expiration and the onset of phonation was measured in millimetres, which was equivalent to the same number of seconds (see figure 2).

Chest wall coordination

Each of the 72 X-Y expiratory relative volume traces for the vowel prolongation speech task were analysed for the frequency of ribcage and abdominal paradoxing. Three traces were excluded from analysis due to subject movement during the recordings. In accordance with definitions of Hodge and Putnam-Rochet [41], paradoxical movements were defined as instances during speech production, where displacement of the ribcage or abdomen was in an inspiratory direction. The prolongation of /a/ was again considered to be representative of all vowel prolongation tasks. In addition to the frequency counts, a measure of the percentage of paradoxing was calculated to determine the total amount of paradoxing per relative volume trace for vowel prolongation. Each relative volume trace was rated 0, 25, 50, 75, or 100% paradoxical (for both abdomen and ribcage independently). The number of traces per phase with a rating of 50, 75, or 100% paradoxical were then divided by the total number of traces recorded during the phase and multiplied by 100 to obtain a measure of the percentage of traces per phase that were $\geq 50\%$ paradoxical for both abdomen and ribcage.

Spirometric analysis

The values obtained for spirometric parameters of vital capacity (VC) and forced expiratory volume at 1 second (FEV_1) were compared to predicted values based on the subject's age, height and sex using the formulae weighted for age and/or height suggested by Boren *et al.* [42] and Kory *et al.* [43].

Perceptual analysis

The results obtained for the FDA were transformed from a 5-point a–e scale of *normal function–no function* to a 9-point scale in order to present them graphically.

The results of the respiratory parameters, and two of the laryngeal parameters (time and volume), were tabulated and recorded graphically for ease of interpretation.

Each of the 18 speech samples was rated by two qualified speech–language pathologist judges on three of the speech dimensions used by FitzGerald *et al.* [40]—phrase length, breath support for speech, and overall intelligibility. Both judges listened independently to a recording of the speech sample. In all, two tapes of the speech samples were made, with random order of presentation of the samples. The judges were unfamiliar with the subject and had no knowledge as to the stage from which each speech sample was taken. To confound the judges, four samples from another dysarthric speaker were included as distracters. Both judges were given a description of the speech dimensions being rated, and a 1–4 descriptive scale on which to rate the severity of each dimension. Unlimited time was allowed for the judges to listen to the tapes of the speech samples and rate the dimensions.

The inter-judge reliability was estimated for each of the three speech dimensions rated using Spearman Rho correlations for ranked data. The mean correlation was 0.652 ($p < 0.001$) for the breath support dimension, 0.674 ($p < 0.05$) for the phase length dimension, and 0.776 ($p < 0.000$) for the intelligibility dimension.

Four speech samples were re-rated by both judges on the three speech dimensions to obtain a measure of intra-judge reliability. Spearman Rho correlation coefficients were calculated to determine the intra-judge reliability for both judges on all three dimensions. The mean correlations were 0.79 ($p < 0.003$) and 1.000 ($p < 0.000$) for judges 1 and 2 respectively. Where the judges' ratings differed for any dimension, a consensus rating was obtained from the two judges and used in the analysis of the results.

The subject's responses on the ASSIDS were tape-recorded and then transcribed by a judge unfamiliar with the subject. A multiple choice answer sheet of 12 words per item was provided for scoring of word intelligibility. The order of presentation of test samples on the tapes was random, to ensure the judge was unaware of the stage to which each sample belonged. The transcriptions were scored according to the test manual and percentage intelligibility for single words and sentences were determined for the subject.

Results and discussion

During the baseline phase both instrumental and perceptual findings indicated severe impairment of breath support for speech. Kinematic analysis revealed incoordination of the chest wall, and incoordination of phonation and expiration. Spirometric values were well below normal, and all perceptual assessments revealed severe respiratory dysfunction.

The most significant finding of the traditional therapy phase was a reduction in ribcage paradoxing. However, no changes were evidenced in the degree of abdominal paradoxing, and no significant improvements were found for other kinematic measures. Spirometric parameters increased slightly, but were variable and remained well below normal levels. Perceptual findings remained at baseline levels.

During the withdrawal phase the subject's performance on all assessment tasks was expected to return to baseline levels. The withdrawal phase, however, was characterized by considerable variability across a number of parameters. The degree of ribcage paradoxing was the only instrumental parameter to return to baseline levels. All other measures were higher than baseline levels, with greater ranges than either baseline or traditional therapy, indicating a high degree of variability. Perceptual measures were also noted to be variable, with respiratory parameters of the FDA increasing, but speech analysis parameters remaining at baseline levels.

The most significant physiological changes were produced during the biofeedback phase. With the use of visual biofeedback of ribcage circumference, the subject improved on all kinematic measures. More importantly, he increased his consistency of performance on these parameters, indicating greater control of his respiratory patterns. Spirometric parameters, which were not targeted during therapy, remained below normal, and perceptual assessments demonstrated mixed results.

KINEMATIC ANALYSIS

Phonation time and voice onset latency

As expected from the subject's physiological profile, baseline assessments of vowel prolongation demonstrated markedly reduced phonation times ($M = 2.41$ seconds, range = 1–3 seconds) when compared to males of the same age ($M = 17.74$ seconds, SD = 4.14) [44] and increased and variable voice onset latencies ($M = 1.22$ seconds, range = 0–2 seconds), indicating inefficient use of exhalation.

Traditional therapy aimed to improve respiratory support for speech by increasing control of inhalation and exhalation. Instruction in increasing the coordination of respiration and phonation by decreasing the voice onset latency was used to partially achieve this aim. As a result of traditional therapy, the subject's voice onset latencies were reduced (M = 1.14 seconds, range = 0.5–2 seconds) and phonation times increased (M = 2.57 seconds, range = 1.5–4 seconds). However, both of these results were small and not convincing evidence of improvement in the subject's control of his breathing pattern for speech.

The subjective nature of the feedback available in traditional therapy techniques provides a possible explanation for the lack of significant improvement in these parameters. Recent research has shown that perceptual-motor learning is facilitated by objective feedback that is both immediate and continuous [20]. However, the feedback provided in the traditional phase was largely subjective, delayed, and non-continuous. The results from the present study indicate that the subject was unable to use feedback provided at the end of one task, to alter his speech breathing pattern during the following task.

During the withdrawal phase, instead of returning to pre-treatment levels, the subject's voice onset latencies were higher than baseline levels, with a mean of 1.32 seconds (range = 0–2). While phonation times were higher than traditional therapy averages, with a mean of 3.18 seconds (range = 1.5–5.5). Both of these parameters demonstrated high degrees of variability, as exemplified in figures 3 and 4, respectively.

The aim of the biofeedback phase was to improve the subject's respiratory support for speech by increasing his control of inhalation and exhalation, utilizing visual biofeedback techniques. With the use of objective, continuous, real-time visual biofeedback of ribcage excursion, the subject was able to produce average phonation time of 4.4 seconds (range = 3.5–5).

During biofeedback therapy, the subject received visual biofeedback of the exact point at which inhalation ends and exhalation begins. Using this information, he was able to adjust his speech breathing pattern to increase the consistency of his voice onset times by attempting to coordinate the beginning of phonation with this point. Although the subject found this task the hardest to achieve, during biofeedback he was able to reduce his average voice onset latencies to 1 second (range = 0–2). More importantly, he demonstrated an increase in the consistency of his response, as exemplified in figure 3. The reduction of voice onset latency demonstrated the subject's ability to improve his coordination of expiration and phonation. In addition, by reducing air wastage through increasing coordination, he was able to sustain phonation for longer. In contrast, throughout the baseline, traditional therapy, and withdrawal phases the subject produced average voice onset latencies greater than 1 second, and demonstrated a high degree of variability.

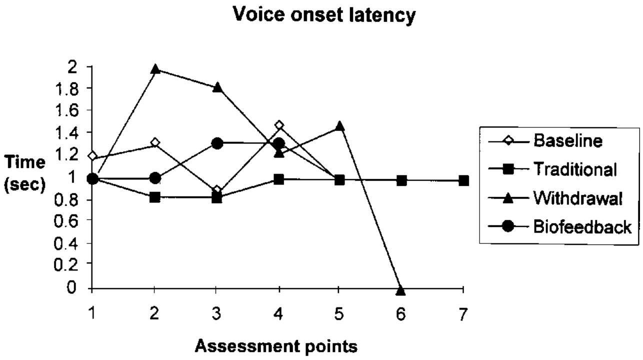

Figure 3 Changes in voice onset latency for vowel prolongations (higher values indicate more severe impairment).

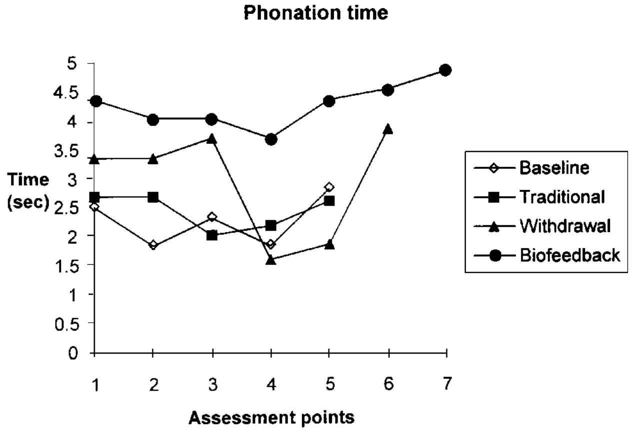

Figure 4 Changes in phonation time for vowel prolongation.

The above results indicate that, with the use of continuous, real-time visual biofeedback of ribcage circumference, the subject was able to modify his abnormal breathing pattern to attain a more natural breathing pattern for speech. The simple nature of the Y-T strip chart visual biofeedback enabled the subject to focus on specific parameters of his inhalation and exhalation. Example traces demonstrating the subject's improved ability to control his inspiration and expiration during the biofeedback phase are shown in figures 5, 6 and 7. Figure 5A, was recorded during session 1 of biofeedback therapy when the subject was instructed to focus only on inhaling deeply and quickly. The figure demonstrates an unsteady inspiratory pattern, as evidenced by the irregular ascending line. In contrast, figure 5B was recorded during session 4 of biofeedback therapy when the subject was given the same instructions. The ascending trace (i.e. increasing ribcage circumference) is smooth and almost vertical, demonstrating a quick, controlled inspiration.

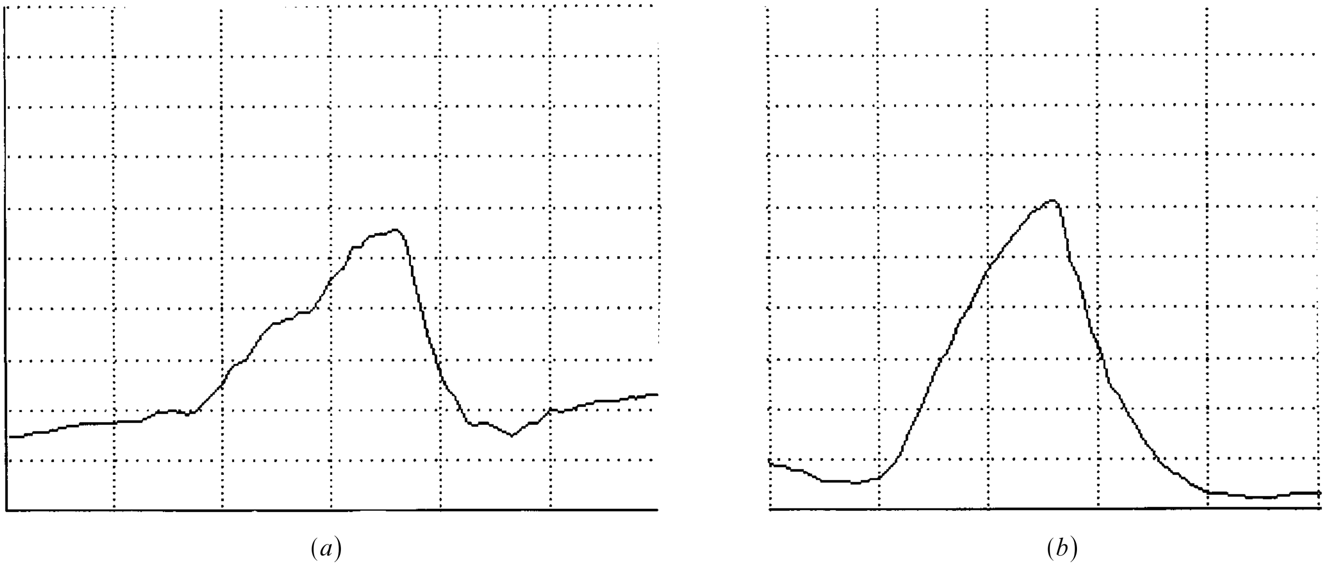

Figure 5 Y-T strip chart trace recorded during (*a*) Session 1, and (*b*) Session 4, of the biofeedback therapy phase demonstrating improvement in the subject's ability to increase speed and depth of inhalation.

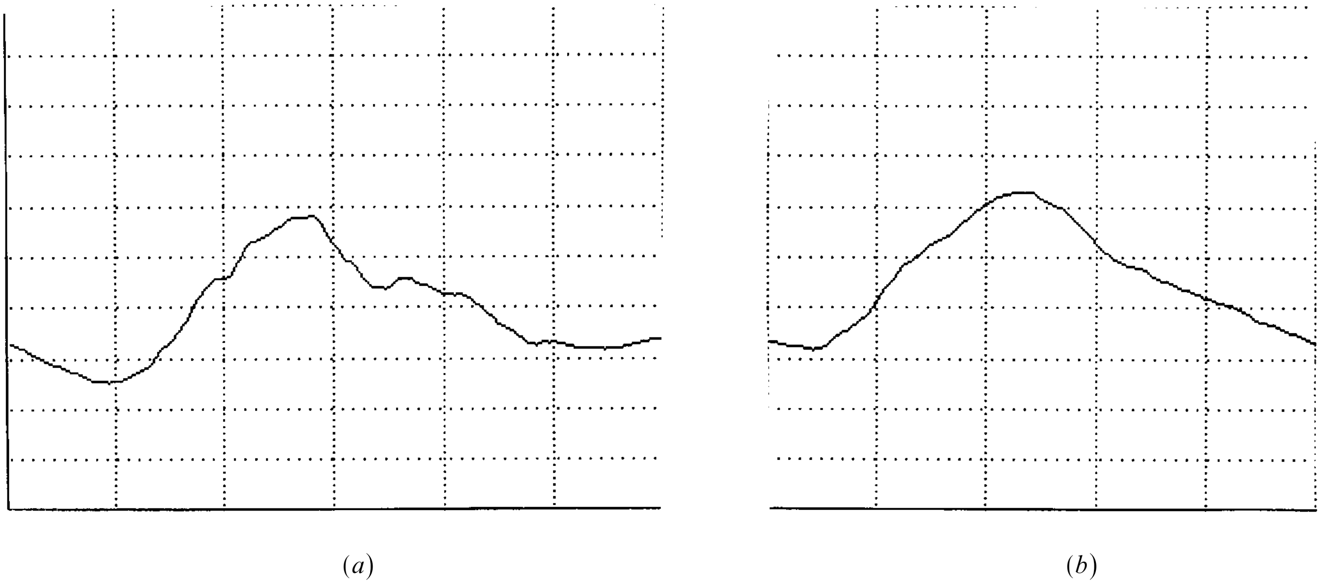

Figure 6 Y-T strip chart trace recorded during (*a*) Session 1, and (*b*) Session 5, of the biofeedback therapy phase demonstrating the subject's ability to increase the control and duration of exhalation.

Figure 6A was recorded during session 1 of biofeedback therapy when the subject was instructed to focus only on duration and control of exhalation. The descending trace (i.e. decreasing ribcage circumference) is irregular, indicating poor control of the expiratory airstream. In contrast, figure 6B, which was recorded during session 4 of biofeedback therapy when the subject was given the same instructions, demonstrates a controlled and prolonged exhalation, as evidenced by the long and smooth descending trace. These traces demonstrate the subject's ability to modify his respiratory pattern with visual biofeedback of ribcage circumference.

During the final two sessions of biofeedback therapy, connected speech tasks were introduced to determine whether the techniques the subject had been using for controlled exhalation and vowel prolongation had improved his respiratory support during connected speech. In the serial speech task of alphabet repetition,

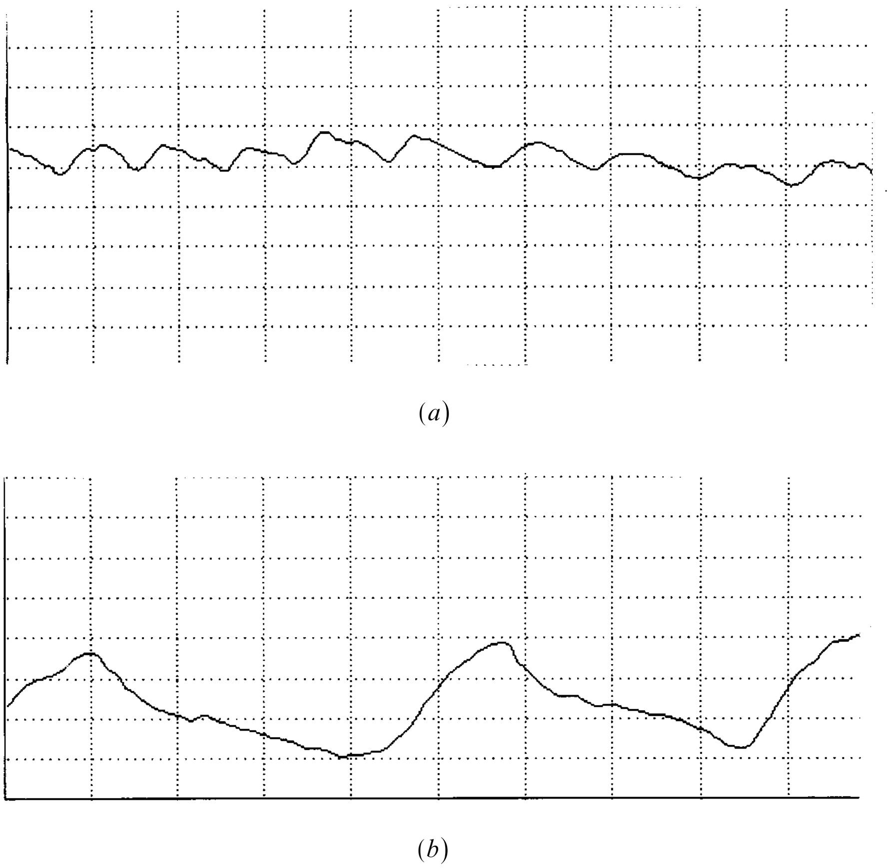

Figure 7 Y-T strip chart trace recorded during alphabet recitation in (*a*) Session 4 of the biofeedback therapy phase, without the use of visual biofeedback, and (*b*) Session 9, with the use of real-time continuous visual biofeedback of ribcage circumference.

the subject was able to alter his depth of inhalation and control of exhalation to improve his respiratory support for speech during this task. Figure 7A demonstrates the subject's ribcage circumference recording during alphabet recitation without the use of visual biofeedback, while figure 7B demonstrates the improved respiratory pattern of the subject during alphabet recitation with real-time, continuous visual biofeedback of ribcage excursion. These figures demonstrate completely different breath patterns during completion of the same task over the same period of time.

Without biofeedback (figure 7A), the subject's inhalations were shallow and he required more than 20 breaths to recite the alphabet. In contrast when provided with biofeedback (figure 7B), the subject increased his depth of inspiration and required only six breaths to complete the task, indicating a more natural speech breathing pattern. In addition, the subject demonstrated a more consistent breath pattern with biofeedback than without biofeedback, as indicated by the similarity in shape of each recorded breath in figure 7B. These recordings illustrate the subject's ability to improve his control of respiration using visual biofeedback.

Following the serial speech task, the subject was instructed to repeat simple four syllable phrases using the visual biofeedback and the techniques for controlled inhalation and exhalation already discussed. Figure 8 is an example of the subject's use of visual biofeedback to improve his control of inhalation and exhalation, and expiratory-phonatory coordination to produce three phrases of four syllables each. The figure demonstrates a deep and controlled inspiratory pattern, as evidenced by the height and smoothness of the ascending trace, and a controlled, steady expiratory pattern, as evidenced by the smoothness and length of the descending trace.

Chest wall coordination

The subject exhibited both abdominal and ribcage paradoxing throughout the baseline phase indicative of an impairment in his ability to coordinate chest wall movements. In particular, during this phase his expira-

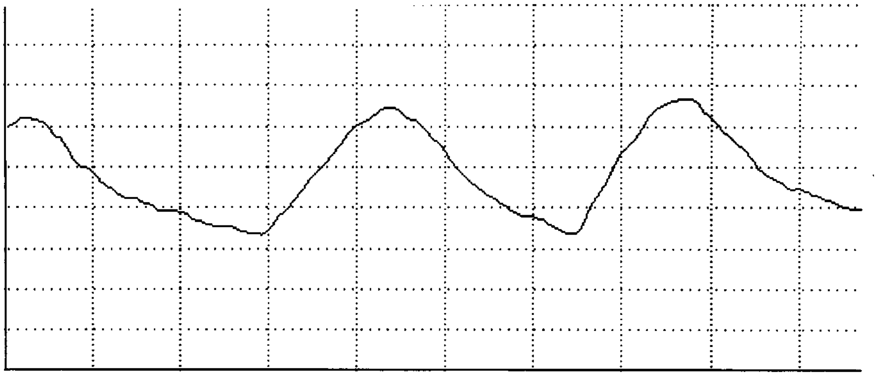

Figure 8 Y-T strip chart trace recorded during phrase repetition in Session 10 of the biofeedback therapy phase with the use of visual biofeedback.

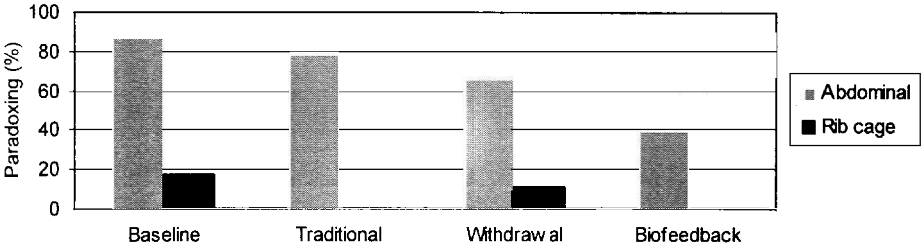

Figure 9 Percentage of relative volume charts for vowel prolongation per phase that were $\geq 50\%$ paradoxical.

tory traces were dominated by abdominal paradoxing, which in some cases occurred for the complete duration of expiration. Murdoch *et al.* [12] suggest that coordination of the chest wall is necessary to produce the steady and constant flow of air required to maintain sub-glottal air pressures necessary for normal speech production. The subject in the present study was adopting an inspiratory-type breathing pattern with regard to his abdominal movements during expiration and, therefore, reducing his control of the expiratory airstream.

A similar pattern of impairment was found by Hixon [45] in a young ataxic woman. Hixon hypothesized that the woman's paralysed abdominal muscles could not resist the downward pressure caused by the decreasing size of the ribcage during expiration. Thus, during expiration, her abdominal circumference increased as her ribcage circumference decreased, causing incoordination of the chest wall and, therefore, decreased control of the expiratory airstream. A similar explanation may account for the chest wall incoordination experienced by the subject in the present study.

There was little difference between the frequency of abdominal paradoxing in the traditional therapy phase ($M = 1.53$, range = 0–3) and the baseline ($M = 1.39$, range = 1–3), however, traditional therapy produced fewer instances of ribcage paradoxing ($M = 0.27$, range = 0–1) than baseline ($M = 0.83$, range = 0–2). Traditional therapy also produced fewer completely abdominally paradoxical traces (3/11) than baseline.

During traditional therapy, the subject continued to demonstrate incoordination of the speech breathing musculature with 80% of trace displaying $\geq 50\%$ abdominal paradoxing, only a slight reduction from baseline levels (88%). Ribcage paradoxing was reduced, however, from 17% of traces displaying $\geq 50\%$ paradoxing in baseline to 0% in traditional therapy (see figure 9). These results indicated that the traditional therapy techniques, not directly targeting coordination of chest wall movements, had little effect on the amount of abdominal paradoxing displayed by this subject but were effective in reducing ribcage paradoxing.

Improved ribcage movement, without an equivalent improvement in abdominal movement, could be attributed to a more severe level of abdominal impairment, as found by Hixon [45] in a young ataxic woman—with moderately to severely impaired ribcage musculature and paralysed abdominal musculature—who demonstrated abdominal paradoxing. In the current study, the subject's infrequent use of his abdominal musculature due to reduced mobility (i.e. wheelchair-bound), could also account for improvements in ribcage movement with increased awareness of breathing patterns, without equivalent improvement in abdominal movement.

During the withdrawal phase, the subject continued to demonstrate incoordination of the speech breathing musculature, with 66% of traces displaying $\geq 50\%$ abdominal paradoxing, and 11% displaying $\geq 50\%$ ribcage paradoxing (see figure 9). The frequency of abdominal paradoxing in the withdrawal phase ($M = 1, 5$, range = 0–4) was similar to traditional therapy levels. The frequency of ribcage paradoxing ($M = 0.72$, range = 0–3), however, had returned to baseline levels. Both parameters demonstrated greater ranges than the baseline or therapy phases, indicating greater variability.

During the biofeedback therapy phase the frequency of abdominal paradoxing ($M = 1.45$, range = 0–3) was similar to baseline and traditional therapy levels. The frequency of ribcage paradoxing ($M = 0.45$, range = 0–1) was lower than baseline levels, and similar to traditional therapy levels. The number of paradoxes per trace, however, did not reflect the fact that a greater percentage of each trace was non-paradoxical in the biofeedback therapy phase than in either the baseline or traditional therapy phase. Only 40% of traces in the biofeedback phase were $\geq 50\%$ abdominally paradoxical as compared to 88% of traces in the baseline phase and 80% in the traditional therapy phase. In addition, the biofeedback phase did not produce any traces with $\geq 50\%$ ribcage paradoxing, compared to 17% in the baseline phase (see figure 9). The marked decrease in abdominal paradoxing was an unexpected finding, considering that during biofeedback therapy the subject did not receive biofeedback of abdominal excursion. The decrease in abdominal paradoxing, therefore, appeared to be a coincidental effect of the subject's improved respiratory pattern utilizing feedback of ribcage excursion.

The physiologic instrumental results indicated that with the use of visual biofeedback of ribcage excursion the subject was able to modify his abnormal breathing patterns to attain a more natural breathing pattern for speech, as evidenced in his increased phonation time, decreased voice onset latencies, and, to a lesser extent, in the decrease in degree of paradoxing, compared to both baseline and traditional therapy levels. These results indicated that biofeedback techniques were superior to traditional therapy techniques in establishing the required physiological changes for the subject in the present study. Similar success in the use of visual biofeedback of chest wall movement for the rehabilitation of abnormal speech breathing in subjects with acquired dysarthria has also been reported elsewhere [11, 24, 25].

Spirometric analysis

Baseline assessments revealed markedly reduced and variable results for both vital capacity (VC) ($M = 19\%$, range = 11–26%) and forced expiratory volume in 1 second (FEV_1) ($M = 20.7\%$, range = 15–27%), when compared to males of the same age and height. Reduced VC has been similarly reported in a study of 20 severe TBI adults with dysarthria [12], and also in subjects with other neuromuscular disorders such as motor neurone disease [45], cerebellar disease [47], and Parkinson's disease [48].

Murdoch *et al.* [12] reported that the reduction in VC and FEV_1 in their TBI subjects did not appear to be attributed to atrophy of the respiratory musculature (as proposed for subjects with motor neurone disease) or decreased excursion of the chest wall due to associated rigidity (as proposed for subjects with Parkinson's disease). They reported that the TBI subjects in their study presented with a similar disruption in the two-part coordination of the chest wall as subjects with cerebellar disease involving the respiratory muscles. Murdoch *et al.* [12] proposed that the chest wall incoordination, leading to decreased excursion of the respiratory apparatus, possibly contributed to the reduction in VC and FEV_1 in the TBI subjects. A similar explanation may account for the substantial reduction in these parameters for the subject in the present study.

Research has also shown that normal subjects initiate connected speech at lung volumes in the middle range of their VC (i.e. 50–60%) [49, 50]. The subject in the present study had a markedly reduced VC compared to normal and was, therefore, only able to initiate speech at very low lung volumes. The subject's spirometric results were consistent with perceptual findings of severely reduced breath support for speech and shortening of phrase length, and indicative of the subject's impaired respiratory volume and control of the expiratory airflow.

Spirometric parameters were not directly targeted during therapy, however, they were monitored throughout the project to determine whether the traditional and biofeedback therapy techniques had any affect on these parameters. Unfortunately, no improvements were noted for VC of FEV_1 in either of the therapy phases.

Perceptual analysis

Baseline perceptual assessment of speech using the FDA indicated severely impaired respiration both at rest and during speech. Assessment of the laryngeal parameters of phonation time and volume, both of which can be affected by respiratory dysfunction [11, 25], also indicated severe impairment. These results were consistent with the instrumental findings of respiratory dysfunction. Instrumental results indicating incoordination of chest wall movement and reduced lung capacity, could account for the perception of impaired speech breathing, and decreased loudness control and phonation times. The subject's incoordination of phonation and expiration, indicating inefficient use of expiratory airflow, could also account for the perception of reduced breath support for speech and decreased phonation times.

Perceptual analysis of 'The Grandfather Passage' speech samples recorded during the baseline phase indicated severely impaired breath support for speech, phrase length, and overall intelligibility, supporting the FDA and instrumental findings of severe respiratory dysfunction. Respiratory incoordination, and incoordination of phonation with the expiratory airstream, could together account for the subject's reduced phrase length and the perception of reduced breath support for speech. Baseline assessments using the ASSIDS indicated severely impaired intelligibility at word level, and unintelligible sentence repetition. These findings were consistent with the judges' scores for overall intelligibility on the speech sample analyses.

During traditional therapy, the results of perceptual assessment were consistent with the unremarkable progress in respiratory function as determined instrumentally, and the results of perceptual assessment in the withdrawal phase were as variable as the instrumental results. Instrumental findings of improved respiratory support for speech in the biofeedback phase were not convincingly supported by the perceptual assessment results. These results were not surprising, however, considering the subject's respiratory support for speech was severely impaired, and appeared to be exacerbated by decreased control of the expiratory airstream in and above the larynx. Severely impaired articulatory function also contributed to the perception of decreased phase length and intelligibility.

Conclusions

The results of the present study demonstrated that real-time continuous visual biofeedback techniques for modifying speech breathing patterns were not only effective, but superior to traditional therapy techniques in the rehabilitation of a child with persistent dysarthria following severe TBI. It is concluded that physiological biofeedback techniques are potentially useful clinical tools for the remediation of speech breathing impairment in the paediatric dysarthric population and may have application in the treatment of other aspects of impaired speech manifest in children following TBI. While more research is required to provide efficacy data for existing biofeedback instrumentation, there is an urgent need to develop a wider range of effective biofeedback techniques that have direct clinical application. As demonstrated by the present study, there is also a need to develop biofeedback techniques specifically tailored to the paediatric population, which involve stimulating and interesting graphic representations of physiological changes. The multiple sub-system impairment of the subject in the present study also demonstrated that future biofeedback research should target several sub-systems of the speech production mechanism, especially those with the greatest effect on intelligibility, in multiple baseline experimental designs.

References

1. Ylvisaker, M. S. and Urbanczyk, B.: The efficacy of speech-language pathology intervention: Traumatic brain injury. *Seminars in Speech and Language,* **11**: 215–226, 1990.
2. Alajouanine, T. and Lhermitte, F.: Acquired aphasia in children. *Brain,* **88**: 653–662, 1965.
3. Brink, J. D., Garrett, A. L., Hale, W. R. *et al.*: Recovery of motor and intellectual function in children sustaining severe head injuries. *Developmental Medicine and Child Neurology,* **12**: 565–571, 1970.
4. Levin, H. S.: Aphasia in closed head injury. In: M. T. Sarno (editor) *Acquired Aphasia* (New York: Academic Press), pp. 427–463, 1981.
5. Najenson, T., Sazbon, L., Fiselzon, J. *et al.*: Recovery of communicative functions after prolonged traumatic coma. *Scandinavian Journal of Rehabilitative Medicine,* **10**: 15–21, 1978.
6. Sarno, M. T.: Nature of verbal impairment after closed head injury. *Journal of Nervous and Mental Disease,* **168**: 685–692, 1980.
7. Sarno, M. T., Buonaguro, A. and Levita, E.: Characteristics of verbal impairment in closed head injured patients. *Archives of Physical and Medical Rehabilitation,* **67**: 400–405, 1986.
8. Theodoros, D. G., Murdoch, B. E. and Chenery, H. J.: Perceptual speech characteristics of dysarthric speakers following severe closed head injury. *Brain Injury,* **8**: 101–124, 1994.
9. Thomsen, I. V.: Evaluation and outcome of aphasia in patients with severe closed head trauma. *Journal of Neurology, Neurosurgery and Psychiatry,* **38**: 713–718, 1975.
10. Adams, J. H., Mitchell, D. E., Graham, D. I. *et al.*: Diffuse brain damage of immediate impact type: Its relationship to 'primary brain-stem damage' in head injury. *Brain,* **100**: 489–502, 1977.
11. Yorkston, K., Beukelman, D. and Bell, K.: *Clinical management of dysarthric speakers*, 2nd ed. (Boston: College-Hill Press), 1991.
12. Murdoch, B. E., Theodoros, D. G., Stokes, P. D. *et al.*: Abnormal patterns of speech breathing in dysarthria following severe closed head injury. *Brain Injury,* **7**: 295–308, 1993.
13. Netsell, R.: *A neurobiologic view of speech production and the dysarthrias* (Boston: College-Hill Press), 1986.
14. Robertson, S. J. and Thomson, F.: *Working with dysarthrics: A practical guide to therapy for dysarthria* (Winslow: Winslow Press), 1986.
15. Guttman, E.: Aphasia in children. *Brain,* **65**: 205–219, 1942.

16. Hecaen, H.: Acquired aphasia in children and the ontogenesis of hemispheric functional specialisation. *Brain and Language,* **3**: 114–134, 1976.
17. Netsell, R. and Rosenbek, J.: Treating the dysarthrias. In: J. Darby (editor) *Speech and language evaluation in neurology: Adult disorders* (New York: Grune & Stratton), pp. 363–392, 1985.
18. Rosenbek, J. C. and LaPointe, L. L.: The dysarthrias: description, diagnosis and treatment. In: D. F. Jones (editor), *Clinical management of neurogenic communicative disorders* 2nd ed. (Boston: Little, Brown & Co), pp. 251–310, 1991.
19. Workinger, M. and Netsell, R.: Restoration of intelligible speech 13 years post-head injury. *Brain Injury,* **6**: 183–187, 1992.
20. Rubow, R. T.: Role of feedback, reinforcement, and compliance on training and transfer in biofeedback-based rehabilitation of motor speech disorders. In: M. McNeil, J. Rosenbek and A. Aronson (editors) *The dysarthrias: Physiology, acoustics, perception, management* (Boston: College-Hill Press), pp. 207–230.
21. Netsell, R. and Daniel, B.: A physiologic approach to rehabilitation for adults with dysarthria. *Archives of Physical Medicine and Rehabilitation,* **60**: 502–508, 1979.
22. Rubow, R. T. and Flynn, M.: Physiologic biofeedback techniques in the treatment of speech disorders. *Proceedings of the Biofeedback Society of America,* Colorado Springs, Colorado, 1980.
23. Simpson, M. B., Till, J. A. and Goff, A. M.: Long term treatment of severe dysarthria: A case study. *Journal of Speech and Hearing Disorders,* **53**: 433–440, 1988.
24. Murdoch, B. E., Sterling, D. K., Theodoros, D. G. *et al.*: Physiological rehabilitation of disordered speech breathing in dysarthric speakers following severe closed head injury. *Paper presented at the Australian Society for the Study of Brain Impairment Conference*, Hobart, 1994.
25. Thompson-Ward, E. C., Murdoch, B. E. and Stokes, P. D.: Biofeedback rehabilitation of speech breathing for an individual with dysarthria: a case study. *Journal of Medical Speech/Language Pathology,* **5**: 277–288, 1997.
26. Gallegos, K., Medina, R. and Espinoza, E.: Electromyographic feedback in the treatment of bilateral facial paralysis: A case study. *Journal of Behavioural Medicine,* **15**: 533–539, 1992.
27. Gibbon, F., Dent, H. and Hardcastle, W.: Diagnosis and treatment of abnormal alveolar stops in a speech-disordered child using electropalatography. *Clinical Linguistics & Phonetics,* **7**: 247–267, 1993.
28. Hardcastle, W. J., Gibbon, F. E. and Jones, W.: Visual display of tongue-palate contact: Electropalatography in the assessment and remediation of speech disorders. *British Journal of Disorders of Communication,* **26**: 41–74, 1991.
29. Kunzel, H.: First application of a biofeedback device for the treatment of velopharyngeal incompetence. *Folia Phoniatrica,* **34**: 92–100, 1982.
30. Shelton, R., Beaumont, K., Trier, W. *et al.*: Videoendoscopic feedback in training velopharyngeal closure. *Cleft Palate Journal,* **15**: 6–12, 1978.
31. Witzel, M., Tobe, J. and Salyer, K.: The use of nasopharyngoscopy biofeedback therapy in the correction of inconsistent velopharyngeal closure. *International Journal of Paediatric Otorhinolaryngology,* **15**: 137–142, 1988.
32. Theodoros, D. G. and Murdoch, B. E.: Laryngeal dysfunction in dysarthric speakers following severe closed head injury. *Brain Injury,* **8**: 667–684, 1994.
33. Theodoros, D. G., Murdoch, G. E., Stokes, P. D. *et al.*: Hypernasality in dysarthric speakers following severe closed head injury. *Brain Injury,* **7**: 59–69, 1993.
34. Murdoch, B. E., Attard, M. and Ozanne, A.: Impaired tongue strength and endurance in developmental verbal dyspraxia: a physiological analysis. *European Journal of Communication Disorders,* **30**: 51–64, 1995.
35. Hinton, V. A. and Luschei, E. S.: Validation of a modern miniature transducer for measurement of inter-labial contact pressure during speech. *Journal of Speech and Hearing Research,* **35**: 245–251, 1992.
36. Horton, S. K., Murdoch, B. E., Theodoros, D. G. *et al.*: Motor speech impairment in a case of childhood basilar artery stroke: treatment directions derived from physiological and perceptual assessment. *Pediatric Rehabilitation,* **1**: 163–177, 1997.
37. Enderby, P.: *Frenchay Dysarthria Assessment* (San Diego: College-Hill Press), 1983.
38. Yorkston, K. and Beukelman, K.: *Assessment of intelligibility of dysarthric speech* (Austin: Pro-ed), 1981.
39. Darley, F. L., Aronson, A. E. and Brown, J. R.: *Motor speech disorders* (Philadelphia: WB Saunders), 1975.
40. FitzGerald, F. J., Murdoch, B. E. and Chenery, H. J.: Multiple sclerosis: associated speech and language disorders. *Australian Journal of Human Communication Disorders,* **15**: 15–33, 1987.
41. Hodge, M. M. and Putnam-Rochet, A. P.: Characteristics of speech breathing in young women. *Journal of Speech and Hearing Research,* **32**: 466–480, 1989.
42. Boren, H. G., Kory, R. C. and Synder, J. C.: The Veterans Administration-Army cooperative study of pulmonary function II. The lung volume and its subdivisions in normal men. *American Journal of Medicine,* **41**: 96–114, 1966.
43. Kory, R. C., Callahan, R. and Boren, H. G.: The Veterans Administrative-Army cooperative study of pulmonary function I. Clinical spirometry in normal men. *American Journal of Medicine,* **30**: 243–258, 1961.
44. Kent, R., Kent, D. and Rosenbek, J. C.: Maximum performance tests of speech production. *Journal of Speech and Hearing Research,* **52**: 367–387, 1987.
45. Hixon, T.: Speech breathing kinematics and mechanism inferences therefrom. In: S. Grillner, J. Lindbolm, J. Lubker and A. Persson (editors) *Speech Motor Control* (New Jersey: Prentice-Hall), pp. 75–93, 1982.
46. Putnam, A. H. B. and Hixon, T. J.: Respiratory kinematics in speakers with motor neurone disease. In: M. R. McNeil, J. C. Rosenbek and A. E. Aronson (editors) *The Dysarthrias; Physiology, Acoustics, Perception, Management* (San Diego: College-Hill Press), pp. 37–67, 1984.
47. Murdoch, B. E., Chenery, H. J., Stokes, P. D. *et al.*: Respiratory kinematics in speakers with cerebellar disease. *Journal of Speech and Hearing Research,* **34**: : 768–780, 1991.
48. Murdoch, B. E., Chenery, H. J., Bowler, S. *et al.*: Respiratory function in Parkinson's patients exhibiting a perceptible speech deficit: A kinematic and spirometric analysis. *Journal of Speech and Hearing Disorders,* **54**: 610–626, 1989.
49. Hixon, T., Goldman, M. and Mead, J.: 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, 1973.
50. Manifold, J. A. Y. and Murdoch, B. E.: Speech breathing in young adults: effect of body type. *Journal of Speech and Hearing Research,* **36**: 657–671, 1993.