Journal of Communication Disorders 44 (2011) 331–335

Contents lists available at ScienceDirect

Journal of Communication Disorders

Effects of exposure to sulfur mustard on speech aerodynamics

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ARTICLE INFO

Article history:
Received 10 March 2010
Received in revised form 16 November 2010
Accepted 5 January 2011
Available online 19 January 2011

ABSTRACT

Sulfur mustard is an alkylating agent with highly cytotoxic properties even at low exposure. It was used widely against both military and civilian population by Iraqi forces in the Iraq–Iran war (1983–1988). Although various aspects of mustard gas effects on patients with chemical injury have been relatively well characterized, its effects on speech are still evolving. We evaluated aerodynamics of speech in male patients following sulfur mustard inhalation. In a case-control study patients with chemical injuries ($n = 19$) along with age and sex-matched healthy control group ($n = 20$) were selected. Aerodynamic analyses were performed by using the Glasgow Airflow Measurement System (known as ST1 dysphonia). Results indicated that except mean flow rate, there were statistically significant differences in vital capacity, phonation time, phonation volume, vocal velocity index, total expired volume and phonation quotient of patients between experimental and control groups ($P < 0.05$). This study demonstrated mustard gas can impair different parameters of speech aerodynamics.

Learning outcomes: As a result of this activity, the reader will be able to describe: (1) the evaluation of air flow in relation to speech system dysfunction and efficiency; (2) the effect of sulfur mustard known as mustard gas on respiratory physiology.

1. Introduction

The vocal tract is an aerodynamic sound generator and resonator system. Variations in the flow air through this tract reflect changes in the "manner" of consonant and vowel articulation. Evaluation of air flow then can provide considerable insight into speech system dysfunction and efficiency (Amerman & Williams, 1979). Adequate airflow during expiration is a prerequisite for the effective vibration of vocal cords during the generation of voice (Dogan, Eryuksel, Kocak, Celikel, & Sehitoglu, 2007).

Inhalation of the incomplete products of combustion such as steam and hot gases causes respiratory tract ventilation injury. Chemical burns are caused by potent acids, alkalies, phenols, cresols, mustard gas or phosphorus. Consequences following these injuries are aphonia, inability to initiate a swallow, and wet and/or breathy vocal quality (Pore & Reed, 1997).

Sulfur mustard known as mustard gas is one of the major chemical warfare agents developed and used during World War I (Kehe & Szinicz, 2005). During the Iraq–Iran war, approximately 100,000 Iranian people were exposed to sulfur mustard used by Iraq army and many victims are still suffered from Aghanouri et al. (2004). The acute and long-term effects of mustard agent in different organs are well-known (Akhavan, Ajalloueyan, Ghanei, & Moharamzad, 2009). Exposure of sensitive respiratory structures to sulfur mustard can lead to persistent lung disease. The delayed effects of deterioration in lung function are the major cause of morbidity following exposure to sulfur mustard (Rowell, Kehe, Balszuweit, &

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doi:10.1016/j.jcomdis.2011.01.003

Thiemann, 2009). Khateri, Ghanei, Keshavarz, Soroush, & Haines (2003) found 42.5% of their cohort of 34,000 patients suffered from lung lesions. The major symptoms are of chronic cough, dyspnoea, and sputum production (Ghanei & Harandi, 2007). Additional symptoms can include chest pain, gastro-esophageal reflux pain, and haemoptysis. Chronic persistant bronchitis was seen in 59% of 197 veterans with a single heavy exposure (Emad & Rezaian, 1997), and in these patients the symptoms can be particularly debilitating, and lead to marked general physical deterioration. A number of pathological processes including asthma, emphysema, chronic bronchitis, pulmonary fibrosis, and bronchiolitis have been described in the respiratory tract of those suffering from sulfur mustard exposure. In a comprehensive study on 3500 patients who were suffered from delayed effects of lung poisons, some speech disorders and laryngeal diseases were reported (Lohs, 1975). One study showed some chemical injuries had different degrees of dysphonia including hoarseness and harshness (Akhavan et al., 2009).

Speech aerodynamics measures have never been obtained from individuals with chemical injuries. However, there are some findings about effects of respiratory disease on breathing speech patterns. One study investigated volumes and breathing patterns during speech in healthy and asthmatic individuals. In that study lung volume changes were measured with a Respitrace. Asthmatics used a greater percentage of their reduced vital capacity. The inspiratory flow rates were slower but expiratory rates were faster. Asthmatics spent a greater proportion of the total respiratory cycle time on inspiration and expired greater volume of air without sound (Loudon, Lee, & Holcomb, 1988). Another study on patients with asthma, emphysema or sarcoidosis revealed that all patients have had a more rapid respiratory rate and increased the proportion of time spent on inspiration (Lee, Loudon, Jacobson, & Stuebing, 1993). The purpose of this study was to evaluate speech aerodynamics in patients who had been exposed to sulfur mustard during Iraq–Iran war.

2. Method

2.1. Participants

A controlled comparative study was conducted using a convenience sample of 19 men with chemical injury and 20 healthy men. The mean (±SD) of age was 40 ± 5.22 years and 41.32 ± 4.63 years in the healthy and patients groups, respectively. Participants with chemical injuries were non-smoking and 30–50 years old, with a documented history of exposure to mustard gas. Based on interviews conducted, the patients had no history of phonatory or pulmonary disease before the chemical exposure. Healthy participants were matched for age and sex, and also had no smoking history and no history of phonatory and pulmonary disease. An informed consent was obtained from all participants.

2.2. Measures

The Glasgow Airflow Measurement System (known as ST1 dysphonia) developed by G.M. Instruments Ltd. and Department of Clinical Physics and Bioengineering (Glasgow, UK), was used to measure the various parameters (McCurrach, Evans, Smith, Gordon, & Cooke, 1991). The system consists of a flowmeter and microphone which is interfaced to a computer which analyzes the data as it is acquired. The data and results are displayed on a monitor and can be sent to a printer.

2.3. Procedures

Tests were performed by a speech-language pathologist in the voice and speech laboratory of the University of Social Welfare and Rehabilitation Sciences, Tehran, Iran. Seven dependent variables were collected. Once the participant was settled, he was instructed to take a deep breath in and then breathe out into the mask in a slow and controlled manner. This procedure was repeated three times for each subject and the recording with the longest duration was considered the vital capacity (VC), a measure of the maximum lung capacity.

Then the subject was instructed to produce a vowel at a comfortable pitch and loudness, and to sustain it as long as was comfortable. Maximum phonation time (MPT) is the maximum time (in seconds) for which voice can be prolonged. It is common for MPT to be used as an indicator of the physiologic support for speech (Baken & Orlikoff, 2000). Phonation volume (PV) is defined as the maximum amount of air which is available for maximally sustained phonation (Yanagihara & Koike, 1967). The mean flow rate over the course of a speech sound, phonation or utterance is considered as mean flow rate (MFR). It is valuable when assessing the general characteristics of speech and vocal function (Baken & Orlikoff, 2000). The measurement of airflow usually represents the mean flow over several glottal cycles (Colton, Casper, & Leonard, 2006). Total expired volume (TEV) which is the phonation volume plus the volume of air which is expired after phonation (Gordon, Morton, & Simpson, 1978). Vocal velocity index (VVI) is defined as the ratio of the mean airflow rate during sustained phonation of/a/to vital capacity (Koike & Hirano, 1968). Finally, Phonation quotient (PQ) is determined by dividing VC by MPT. It is computed to reduce the possible bias of supportive respiratory capabilities compensating for poor vocal fold closure (Dejonckere, 2000).

2.4. Statistical analysis

Statistical analyses were performed using the SPSS (version 11.0, SPSS Inc., Chicago, IL). The independent *t*-test was used to compare two groups concerning seven dependent variables related to the aerodynamics of speech.

Table 1
Mean (±SD) of variables of speech aerodynamics among chemical injuries and control groups.

Values	Chemical injuries ($n = 19$) Height:175.11 ± 5.39 cm [5′7″]	Control ($n = 20$) Height:174 ± 13.48 cm [5′7″]	*P*-value	*df*	*t*
Vital capacity (l)	1.77 ± 0.89	3.22 ± 0.91	0.00*	37	4.99
Maximum phonation time (s)	11.55 ± 8.16	25.41 ± 5.32	0.00*	37	6.31
Phonation volume (l)	1.54 ± 1.20	3.05 ± 1.29	0.001*	37	3.60
Mean flow rate (ml/s)	137.56 ± 89.42	124.67 ± 89.42	0.615	37	−.50
Vocal velocity index	91.57 ± 74.48	37.65 ± 15.85	0.004*	37	−3.08
Total expired volume (l)	1.56 ± 1.20	3.58 ± 0.83	0.00*	37	5.42
Phonation quotient (ml/s)	230.24 ± 201.9	133.74 ± 51.26	0.046*	37	−2.07

* *P*-value less than 0.05 was considered significant.

3. Results

The comparison of aerodynamics of speech parameters in study groups is shown in Table 1. Results of the statistical analysis revealed significant differences between the two groups for Vital Capacity ($t = 4.99$, $df = 37$, $P = 0.00$), Maximum Phonation Time ($t = 6.31$, $df = 37$, $P = 0.00$), Phonation Volume ($t = 3.60$, $df = 37$, $P = 0.001$), Vocal Velocity Index ($t = -3.08$, $df = 37$, $P = 0.004$), Total Expired Volume ($t = 5.42$, $df = 37$, $P = 0.00$), and Phonation Quotient ($t = -2.07$, $df = 37$, $P = 0.046$). No significant differences were observed between chemical group and control group in Mean Flow Rate.

The mean of VC, MPT, PV and TEV was decreased in patients rather than healthy subjects. Also, the mean of VVI and PQ was increased in chemical injuries rather than the healthy controls. Except MFR, there were statistically significant differences between two groups in all remainder values ($P < 0.05$).

4. Discussion

Overall, the results of this study show that the subjects chemically injured by mustard gas exhibit pulmonary rather than phonatory deficits. That is, the results of our measures indicated that chemically injured subjects had a decreased respiratory capacity to support breathing for speech. Each dependent variable will be discussed in terms of how it supports this conclusion.

Vital capacity (VC) was greatly reduced in our chemically injured subjects compared to our healthy control subjects. We expected that result because VC mainly reflects lung function. VC is the maximum volume of air that can be expired (Gordon et al., 1978). Respiratory problems are the greatest cause of long-term disability among patients with combat exposure to sulfur mustard gas (Ghanei & Adibi, 2007). A severe reduction in vital capacity could potentially affect utterance length and phrasing during speech (McNeil, 1997). We did not measure utterance length or phrasing in this study, but it would be desirable to add those measures to future studies in this population.

Maximum phonation time (MPT) was also greatly reduced in our chemically injured patients compared to our healthy control subjects. We expected that result, too, because MPT reflects, in part, the efficiency of the respiratory system (Colton et al., 2006). McNeil (1997) has pointed out that reduced MPT may be the result of respiratory insufficiency. For example, Dogan et al. (2007) found that the average MPT was lower in asthma patients compared to controls. On the other hand, it must be noted with care that MPT has also been viewed as a way to provide information on some supraglottal control (Kent, Kent, & Rosenbeck, 1987) and phonatory system efficiency (Colton et al., 2006). The MPT measure alone cannot separate pulmonary from phonatory contributions, but taken together with the reduced VC measure in our chemically injured subjects, the two measures point more convincingly to problems at the pulmonary level.

Phonation quotient (PQ) is expressed in milliliters per second; it reflects air usage during phonation and thus both lung and laryngeal functions (Dogan et al., 2007). It was expected that the chemically injured group would show a greater PQ value compared to the control group because PQ is determined by dividing VC by MPT and the chemically injured group exhibited a statistically lower MPT value than did the control group. Values of PQ in adults and children are typically between 120 and 190 ml/s (Haynes & Pindzola, 2004). Our chemically injured subjects showed a mean value of 230.24 ml/s. Our results are similar to those of Dogan et al. (2007) who found that asthma patients exhibited a greater average PQ value than did healthy subjects. Again, the PQ measure alone cannot separate pulmonary from phonatory contributions, but taken together with the reduced VC measure in our chemically injured subjects, the measures are consistent in pointing to problems at the pulmonary level.

Phonation volume (PV) is related to measures of the MPT and mean phonatory airflow. Thus, the PV is generally affected by the same factors (especially vital capacity) that influence maximum phonation (Baken & Orlikoff, 2000). Low vital capacity and low phonation volume indicate poor lung function of organic or neuromuscular origin (Gordon et al., 1978). It was expected that the chemically injured group would show a lower PV value compared to the control group because VC and MPT values were lower in our chemically injured group compared to our healthy control subjects.

Total expired volume (TEV) was also greatly reduced in our chemically injured patients compared to our healthy control subjects. We expected that result, because TEV is the phonation volume plus the volume of air which is expired after phonation, and the chemically injured group exhibited a statistically lower PV value than did the control group.

The flow rate measured during vocalization provides a reflection of the speaker's ability to regulate the mean resistance of the glottal airflow. During production of a vowel, the upper airway resistance is very small compared to the resistance of

the glottis. Since airflow is a reciprocal function of resistance, measuring it during sustained phonation of a vowel should provide insight into glottal function (Baken & Orlikoff, 2000). In terms of a review on aerodynamic date available in the Japanese literature, Hirano (1981) concluded that the normal mean phonatory flow rates between 90 and 140 ml/s can be expected. In our study, no significant differences were observed between chemically injured group and control group.

Vocal velocity index (VVI) is defined as the ratio of the mean airflow rate during sustained phonation of/a/to vital capacity (Koike & Hirano, 1968). VVI was greatly increased in our chemically injured patients compared to our healthy control subjects. It was expected, because the chemically injured group exhibited a statistically lower VC value than did the control group. Baken and Orlikoff (2000) reported that VVI values greater than 44 or less than 14.3 should be considered abnormal. Our chemically injured subject showed a mean value of 91.57.

4.1. Limitations of the present study

Several factors have to be considered when interpreting the results of this study. First, only 19 patients participated in this study. The results from only 19 patients seem difficult to generalize to the population of chemical patients. Because of lack of cooperation of patients it was not possible to include more patients in this study. Secondly, it was not possible to add acoustic analysis and/or to estimate subglottal pressure in this study. The instrument that we have used in this study was not able to evaluate these components. If these evaluations were completed, we could better judge about the results.

5. Conclusion

We found that sulfur mustard as a potent chemical warfare agent could impair values of speech aerodynamics. These effects can be measured and evaluated as the disturbed phonatory criteria in patients with chemical injuries. We believe these criteria could be considered as a valued guide for speech and language pathologists for better understanding of the related physiopathology and for improving therapeutic plans during treatment of the chemical victims.

To our knowledge, this is the first study to evaluate speech aerodynamics in sulfur mustard-exposed patients with respiratory involvement. However, complementary studies are needed to reveal different aspects of such disorder in this filed.

Acknowledgments

Our thanks go to Research Center for Chemical Injuries and to Baqiyatallah University of Medical Sciences for their generous supports. The authors wish to thank Monir Mazaheri and Hamid Asayesh for their help in editing of this article.

Appendix A. Continuing education questions

1. What is mustard gas?
   (a) An alkylating agent.
   (b) A chemical warfare agent.
   (c) An agent with highly cytotoxic properties.
   (d) All of items.
2. What is the major effect of mustard gas?
   (a) It primarily affects the respiratory system.
   (b) All organs of the body.
   (c) It primarily affects the laryngeal system.
   (d) It primarily affects the skin.
3. Which one of the speech aerodynamics was evaluated in this study?
   (a) Nasal air pressure
   (b) Oral air pressure
   (c) Maximum phonation time
   (d) Nasal airflow
4. Mustard gas has...
   (a) Acute effects only.
   (b) Long-term effects only.
   (c) Acute and and later developing effects.
   (d) A number of effects that have not been identified at this time.
5. Considering the findings of this study and the current body of knowledge about the effects of mustard gas, which of the following areas for future research appears to be least needed?
   (a) Evaluating various acoustic parameters in chemical patients.
   (b) Measuring additional speech aerodynamics parameters.
   (c) Assess other physiologic speech parameters in chemical patients.
   (d) All of the above.

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