Bratisl Lek Listy 2011; 112 (3)
102 – 108

PERSPECTIVES

Urge to cough and its application to the behavioural treatment of cough

Vertigan AE[1], Gibson PG[2]

Speech Pathology, John Hunter Hospital, University of Newcastle, Australia. **anne.vertigan@hnehealth.nsw.gov.au**

Abstract: The Urge-To-Cough has recently been recognised as a significant factor in understanding the pathophysiology of cough. The principles in the urge to cough model are relevant to the behavioural management of laryngeal conditions including chronic non-specific cough, cough due to paradoxical vocal fold movement, aspiration and reflux. This paper analyses the pathophysiology and behavioural management of these conditions according to the urge to cough model *(Tab. 3, Ref. 28)*. Full Text in free PDF *www.bmj.sk.*
Key words: cough, laryngeal conditions, non-specific cough, cough model.

The urge to cough has recently been recognised as a significant factor in understanding of the pathophysiology of cough. The urge to cough is the cognitive process related to the need to respond to the cough stimulus (1). Capsaicin inhalation has been found to provoke an urge to cough and is associated with activation of various areas in the cerebral cortex including the primary motor cortex and primary sensory cortex (2). Davenport (2008), described a six stage model of the Urge to Cough which has a similar neural mechanism to other visceral systems. The principles in this Urge to Cough model are relevant to several laryngeal conditions that have traditionally been managed via behavioural treatments. These conditions include chronic non specific cough (CNSC), chronic refractory cough (CRC) such as cough persisting after treatment for gastroesophageal reflux disease (GER), paradoxical vocal fold movement/vocal cord dysfunction (PVFM/VCD), and oropharyngeal dysphagia. The evidence base supporting behavioural interventions for these conditions requires further development and greater understanding of the pathophysiology behind symptom improvement is required. The purpose of this paper is to apply the Urge to Cough model to the pathophysiology and treatment of functional laryngeal disorders to ensure consistency between the clinical approach and theoretical understanding of underlying physiology.

Cough can be broadly classified as *necessary cough* where there is a physical need to cough in order to maintain airway patency or clearance. Examples of necessary cough include cough in response to aspiration, vocal fold spasm, or excessive secretions in the lower airway. *Unnecessary cough* is cough that occurs in response to irritation where there is no physiological need to cough and can also be termed *behavioural cough.* Examples of unnecessary cough include cough in response to perfume, GER, or a sensation of laryngeal irritation, which is common in CNSC.

Urge to cough model

The urge to cough model contains six stages: a stimulus, the urge to cough, a desire for action, a motor action, feedback and reward (1). These stages are summarized in Table 1. The stimulus for cough (stage 1) triggers a neural event. Receptors that trigger cough are located in the upper and lower respiratory tract and are activated by chemical, mechanical and thermal stimuli. Afferents project to a brainstem cough centre that also projects to higher cortical structures. This leads to the urge to cough (stage 2). The urge to cough is a neural urge activation that converts physical stimuli into a cognitive physiological urge. This urge is translated to desire for action (stage 3) which produces conscious motor action (stage 4) toward a particular goal to either suppress or elicit a cough. Cough reflex sensitivity testing with capsaicin in normal subjects elicited activation of the somatosensory, premotor, prefrontal, limbic and cerebellar regions (2) (University of Newcastle). There was a significant correlation between the magnitude of signal change and the magnitude of urge to cough ratings in several cerebral areas.

The descending neural drive for motor action is a physical response to achieve the urge-desire goal which may be a cough,

[1]Speech Pathology, John Hunter Hospital, University of Newcastle, and [2]Department of Respiratory & Sleep Medicine, John Hunter Hospital and Woolcock Institute of Medical Research, University of Newcastle, Australia

Address for correspondence: A.E. Vertigan, Dr, MBA, PhD, Speech Pathology, John Hunter Hospital, Locked Bag 1, Hunter Region Mail Centre, NSW 2310, Australia.
Phone: +61249213726

Acknowledgements: This work was supported by the NHMRC Centre for Clinical Research Excellence in Respiratory & Sleep medicine. Prof Gibson is an NHMRC practitioner fellow. Dr Vertigan is holds a post doctoral research fellowship. The authors are very grateful to Dr Paul Davenport for advice in the development of this manuscript.

The paper is dedicated to the memory of Professor Juraj Korpas.

Tab. 1. Summary of Davenport Urge to Cough Model and its application to the cough.

Stage	Description	Application to Management of cough
1. Stimulus	Trigger for neural event	Understand exposure to cough triggers Determine whether exposure to triggers results in cough or urge to cough
2. Urge	Physical need to respond to stimulus	Warning before cough Rate urge to cough during cough challenge tests
3. Desire	Transition of urge into central neural targeted goal	Rate urge to cough during cough challenge tests
4. Action	Physical response that satisfies urge-desire	Deliberate coughing Cough reflex sensitivity testing Description of cough Strategies used in attempt to control cough
5. Evidence	Feedback to neural systems	Perception of effectiveness of cough and other strategies
6. Reward	Sensory system that determines if urge is satisfied	Rating of sensation after coughing

Tab. 2. Application of urge to cough to clinical scenarios.

Stage	1. Chronic non-specific cough	2. Cough due to PVFM	3. Cough due to aspiration of food or fluids	4. Cough due to persisting reflux
1. Stimulus	No tussigenic stimulus Low cough threshold	Involuntary vocal fold adduction during episode of PVFM	Food reaches level of vocal folds	Reflux event
2. Urge	No physical need Cough in response to laryngeal parasthesia	Vocal folds need to abduct to maintain airway Person perceives difficulty inspiring air	Food needs to be cleared to maintain airway patency	Need to prevent aspiration of gastric contents if they reach level of airway or cough stimulated by cough reflex arc.
3. Desire	Person perceives need to clear irritation from airway	Desire to cough to open airway	Person perceives need to cough	Patient perceives need to clear irritation
4. Action	Cough	Cough → Vocal folds abduct and airway reimains patent	Cough → Food dislodged from vocal folds	Cough which clears gastric contents from glottis The cough may stimulate more reflux
5. Evidence	Feedback that irritation is reduced	Feedback that airway patency has been restored	Feedback that materail has been dislodged	Feedback that the laryngeal irritation is relieved
6. Reward	Urge satisfied	Urge satisfied. No breathing discomfort. Action reinforced	Urge satisfied. No sensation of food residue. Action is reinforced	Urge satisfied. No irritation. Action reinforced

PVFM – vocal cord dysfunction

or an attempt to modify or suppress the cough. Descending cortical motor neurones activate the brainstem cough neural network, which generates the cough (stage 4). The cough action stimulates a feedback system (stage 5). This feedback provides the central nervous system with evidence of the motor action, for example the pattern of action and effectiveness of action. Feedback comes from cough activated sensory receptors and central neural receptors. Feedback projects to the limbic system which mediates a sense of reward (stage 6). The feedback provides cognitive information to the individual that the right action has occurred and rewards the continuance of the action. The Urge to Cough model can be applied to several different clinical conditions which are summarised in Table 2.

Chronic non specific cough

Chronic Non-Specific Cough (CNSC) accounts for approximately 10 % of cases of chronic cough. CNSC occurs when there is no known cause for the cough and when paradoxical vocal fold movement, asthma, ACE inhibitors, post nasal drip syn-

Tab. 3. Application of urge-to-cough model to therapeutic approaches.

	CNSC	PVFM	Aspiration	GER	LPR	LPRA
Stimulus	Vocal hygiene, trigger avoidance, anticipatory CSS	Vocal hygiene, trigger avoidance, aticipatory PVFM release breathing	Swalloving therapy to prevent aspiration	Vocal hygiene, trigger avoidance, pharmaceutical and lifestyle strategies for reflux	Vocal hygiene, trigger avoidance, pharmaceutical and lifestyle strategies for reflux	Pharmaceutical and lifestyle strategies for reflux
Urge	Reduce stimulus to cough. Anticipatory CSS	Recognize urge to breathe rather than urge to cough				
Desire	Psychoeducational counselling; education	Psychoeducational counselling; education	Encourage cough	Psychoeducational counselling; education	Psychoeducational counselling; education	
Motor	CSS	Relaxed throat brething	Adduction exercises; Deliberate coughing	Cough suppression strategies	Cough suppression strategies	Encourage cough
Feedback	Perceives reduction in irritation	Sense improvement in breathing	Improved respiration	Perceives reduction in irritation	Perceives reduction in irritation	Perceives reduction in irritation
Reinforcement	Education to reinforce success of strategies	Positive reinforcement of response that both prevents and relieve episodes		Education to reinforce success of strategies	Education to reinforce success of strategies	

CNSC – chronic non specific cough, PVFM – paradoxical vocal fold movement, GER – gastroesophageal reflux, LPR – laryngopharyngeal reflux without aspiration, LPRA – laryngopharyngeal reflux with aspiration, CSS – cough suppression strategy

drome, lung pathology, smoking and GER have all been investigated and managed (3, 4, 5).

Cough in CNSC is thought to be due to hyperresponsiveness of the upper airway so that cough is triggered by a low level of sensory input (stage 1: stimulus). Cough may also be triggered by an abnormal sensation in the upper airway. Typically this laryngeal parasthesia is described as an *itch, scratch,* or *need to clear something from the throat.* Other relevant stimuli are those that become tussogenic in CNSC, such as perfume, exercise, vocalisation and cold air. The stimulus causes activation of the airway nerves but with no airway compromise or functional need to clear the airway. This stimulus creates an urge to cough even when there is nothing to clear from the airway (stage 2: urge). In other words, the urge to cough is hypersensitive. The individual perceives the need to clear irritation from the airway (stage 3: desire) and coughs in response to this perceived need (stage 4: motor). The cough is typically dry and non productive. In some cases the individual may cough deliberately to clear the irritation from the throat or may cough repeatedly in attempt to clear very small quantities of secretions. In these cases it could be argued that the urge to cough is similar to forced cough. This cough behaviour can increase sensitivity and irritation in the airway and lead to phonotrauma (6). The individual receives feedback that the cough has been relieved (stage 5: feedback) and that the irritation has been temporarily relieved. This behaviour may, however, form a cycle where irritation leads to coughing, which creates further irritation that subsequently leads to further coughing.

The Urge to Cough model can be applied to assessment of individuals with chronic cough as outlined in Table 1. For example stage 1 (stimulus) could be examined by understanding exposure to various cough triggers and whether those triggers result in a cough or an urge to cough. In stage 2 (urge) the urge to cough might be reduced by reducing the stimulus to cough which may subsequently reduce hyperresponsiveness of the upper airway. Understanding the degree and type of warning before cough episodes provides information about the individual's urge and desire to cough (stages 2 and 3). Furthermore the urge to cough can be rated during cough challenge tests. It could be argued that the desire for cough equates to the warning that an individual may experience before cough. This occurs commonly, although 55 % of individuals with CC report that they perceive no warning before the cough (6).

Motor aspects (stage 4) can be examined by determining whether or not the individual coughs deliberately in response to the urge to cough. Twenty five percent of individuals with cough report that they cough deliberately in response to irritation (7). Cough reflex sensitivity testing, description of the cough, cough pattern analysis and the strategies individuals use in attempt to control their cough can evaluate the motor component of the cough. The feedback component (stage 5) could be assessed by determining the individual's perception of effectiveness of their cough or other strategies used to control or suppress the cough. Finally the reward (stage 6) could be assessed by asking individuals to describe how they feel after a cough.

In addition to assessment, the urge to cough model can be applied to behavioural intervention for cough (Tab. 3). Speech pathology intervention has been found to be effective for individuals with chronic cough that has persisted despite medical

management (8). In a randomised trial there was an 88 % improvement in cough following speech pathology intervention compared with a 14 % improvement in the placebo group. Speech pathology intervention encompasses four phases including education, vocal hygiene training, cough suppression strategies, and psychoeducational counselling (8, 9). The education component encompasses information about the lack of physiological benefit from coughing, negative side effects of repeated coughing and the rationale for behavioural management. Vocal hygiene training involves strategies to improve hydration and reduce exposure to irritating substances. Strategies to suppress the cough include distraction techniques, Cough Suppression Swallow technique, Cough Control Breathing technique and the relaxed throat breathing technique. Psychoeducational counselling aims to assist the individual to internalise responsibility for cough behaviour and set realistic goals for treatment.

This treatment can be analysed according to the Urge to Cough model. Management of stage 1 (stimulus) focuses on reducing the stimulus for cough through temporary reduction in exposure to known triggers where possible while they learn to control symptoms in controlled conditions. Throughout the course of treatment they gradually reduce avoidance of triggers. Vocal hygiene training may also reduce the laryngeal irritation by improving hydration. It is hypothesised that poor vocal hygiene contributes to increased cough stimulus and increased urge to cough. Dehydration has been shown to increase phonation threshold pressure and increase the risk of vocal fold injury (10, 11). Promoting nasal rather than oral breathing may prevent pharyngeal drying and cooling. Shivansker et al found increased vocal effort following oral versus nasal breathing (12). In stage 2 (urge) the urge to cough might be reduced by reducing the stimulus to cough which may subsequently reduce hyperresponsiveness of the upper airway. The risk of vocal fold injury increases with increased vocal load (13). It is hypothesised that phonation can contribute to the urge to cough in individuals with CC due to increased hyperresponsiveness and hyperadduction of the vocal folds. The desire to cough (stage 3) in response to the urge can be modified by education and psychoeducational counselling with emphasis on the fact that cough is occurring in response to irritation and that there is no physiological need to cough. These strategies are designed to modify the individual's cognitive interpretation of the urge signals. Individuals are taught that despite irritation, there is no need to cough and that they can respond to the cough urge a different way. Anticipatory use of cough suppression strategies can also be applied after exposure (stimulus) or perception of the urge to cough in order to reduce cough. Management of stage 4 (motor) involves substituting the cough with various cough suppression strategies such as the Cough Suppression Swallow, and Breathing Techniques for Cough. Individuals are taught to substitute a cough suppression technique for a cough when they perceive the need to cough. In contrast with coughing, these techniques do not cause additional irritation and vocal fold trauma, hence do not contribute to further stimulation of the cough. When successfully implemented, these techniques can satisfy the urge to cough. It is necessary to ensure individuals practice these behavioural techniques when symptom free to ensure proficiency and subsequently enable them to implement the technique automatically when they perceive the urge to cough. In stage 5 (feedback), the individual receives feedback in that the urge to cough has been relieved by an activity other than coughing or a sustained urge to cough. Stage 6 (reinforcement) comprises positive reinforcement that the irritation has been relieved without the discomfort of coughing. Behavioural intervention in these instances may need to focus on increasing awareness of impending cough. In therapy for CC, psychoeducational counselling may be required to correct faulty conceptions about the cough.

Cough due to Paradoxical Vocal Fold Movement

Paradoxical Vocal Fold Movement (PVFM), also known as Vocal Cord Dysfunction(VCD), is a condition whereby the vocal folds adduct episodically and involuntarily during inspiration leading to symptoms of dyspnoea, stridor, dysphonia and cough (14). PVFM is associated with chronic cough and approximately 50 % of patients with chronic cough also have PVFM (15). There is an overlap in the symptomatology between PVFM and CC (7). PVFM accounts for approximately 10 % of CC.

There is also an overlap in the triggers for CC and PVFM (16–20). It has been hypothesised (21) that cough occurs in response to episodes of PVFM in attempt to open the vocal folds (stage 1: stimulus). In this case the episode of PVFM is the stimulus for cough. The vocal fold spasm triggers an urge to cough which is required in order to maintain airway patency (stage 2: urge). The individual senses difficulty breathing and interprets this as an urge to cough to open the airway (stage 3: desire). The individual coughs which forces abduction of the vocal folds and establishes airway patency (stage 4: motor). They receive feedback that airway patency has been achieved by a reduced urge to breathe and that respiration has returned to normal (stage 5: feedback). Although the cough response might be reinforced, it may actually increase laryngeal irritation and the risk of phonotrauma (6). The cycle is reinforced as the cough response is temporarily effective in relieving dyspnoea. There are likely non-cough neural responses elicited by PVFM, including a sense of suffocation, urge to breathe, urge to gag, and a sensation of anxiety and panic when increased difficulty of breathing occurs (14, 21). The behavioural interventions described below target these sensations as well.

Behavioural intervention for PVFM targets the urge to cough cycle in a number of different ways (Tab. 3). Firstly it targets the stimulus component by identifying and reducing exposure to known triggers to PVFM (stage 1). Secondly the desire to cough is addressed through education and increasing awareness of vocal fold spasms. Patients are taught that when they perceive the need to cough they are actually perceiving a difficulty breathing due to vocal fold spasm (stage 3). Individuals are also taught to increase awareness of precipitating sensations for the PVFM episodes. Specific techniques such as Relaxed Throat Breathing, PVFM Release and emergency strategies for relieving PVFM

are taught to voluntarily abduct the vocal folds during an episode (18) (stage 4: motor). These motor responses address the neural urge stimuli (urge to breathe and urge to cough) by releasing PVFM without employing potentially phonotraumatic behaviours such as cough. Many individuals describe 'air hunger' during an episode of PVFM (14) as they perceive inadequate inspiration. Therapy relies on patients being aware of the warning signs (urge and desire) and implementing the therapy exercises at the very first sign of breathing discomfort. When successfully able to avert an episode of PVFM, patients are asked to sense the improvement in their breathing (stage 5: feedback). This sequence sets up a positive reinforcement (stage 6) of a response that prevents PVFM episodes rather than merely relieves the episodes. This response can often relieve the anxiety that accompanies a PVFM episode.

Cough due to oropharyngeal dysphagia

Cough can also occur in individuals with oropharyngeal dysphagia. The four phases of swallowing are the oral preparatory phase, the oral propulsive phase, the pharyngeal phase and the oesophageal phase. In the oral preparatory phase, food is masticated and prepared into a cohesive bolus. During this phase the velum and base of tongue seal to prevent material falling into the pharynx. In the oral propulsive phase food is propelled posteriorly in the oral cavity by sequential contraction of the tongue along the hard palate. In the pharyngeal phase the bolus moves through the pharynx and through the upper oesophageal sphincter. During this phase velopharyngeal closure occurs to prevent material entering the nasal cavity. Airway protection to prevent penetration of food and fluids into the laryngeal vestibule and trachea occurs through closure of true and false vocal folds, hyolaryngeal excursion and epiglottic closure. The oesophageal phase involves movement of the bolus through the oesophagus and through the lower oesophageal sphincter. Dysphagia can affect any of the phases of swallowing. Oropharyngeal dysphagia can result from a number of different medical conditions such as traumatic brain injury and stroke. It can lead to serious medical complications such as aspiration pneumonia, dehydration and malnutrition.

The urge to cough model can be used to understand the pathogenesis of cough due to oropharyngeal dysphagia. Oropharyngeal dysphagia causes material to penetrate the laryngeal vestibule during swallowing and trigger cough (stage 1: stimulus). The response triggered is likely to represent an expiration reflex, rather than a triphasic cough response. If sensation is intact the penetration may trigger an urge to cough (stage 2: urge). The individual perceives the need to cough (stage 3: desire). The cough/expiration reflex occurs and is effective in clearing penetrated material from the airway (stage 4: motor). The patient receives feedback that food has been dislodged from the airway thus satisfying the urge.

This sequence can be further compromised in individuals with neurological impairment. Firstly, if sensation is not intact, the patient may not perceive a need to cough when laryngeal penetration occurs and material will continue to enter the trachea without a reflexive cough (stage 2: impaired urge). This phenomenon is known as silent aspiration (22). Secondly, coexisting cognitive problems may impair the perception of the urge and the desire to cough (stage 3: impaired desire). Thirdly, impaired motor function due to apraxia or weakness of the respiratory and laryngeal muscles may render the cough response inadequate to clear material from the laryngeal vestibule (stage 4: impaired motor function).

In clinical practice it is presumed that a cough is physiologically required to protect the airway. However it has been argued that it is the expiratory reflex rather than a cough reflex that protects the airway. The expiratory reflex is defined as a strong expiratory effort that is not preceded by an inspiration or accompanied by closure and opening of the glottis that results from chemical and mechanical stimulation of the larynx (23). The expiratory reflex starts with expiration and the function is to prevent aspiration. In contrast, a cough reflex starts with inspiration the function is to draw air into the lungs to promote a more efficient subsequent expulsion of mucous and airway debris. Despite these clear and significant differences there is limited differentiation between these two reflexes in the clinical management of patients with oropharyngeal dysphagia.

The primary aim of speech pathology intervention in an individual with aspiration due to oropharyngeal dysphagia is to prevent aspiration though therapeutic manoeuvres, compensatory postural strategies or modification of food and fluid textures (22). These strategies increase airway protection during swallowing. This aspect of the therapy targets stage 1 (stimulus). The presence of a cough/expiration reflex generally indicates laryngeal penetration and provides feedback to patient and therapist that the safety of the swallow has been compromised. If the patient is known to have silent aspiration, for example as demonstrated by modified barium swallow or fibreoptic endoscopic examination, they may be asked to cough deliberately in order to clear material. However voluntary cough has different motor actions from reflexive cough (24). If cough motor function is impaired (stage 4), laryngeal adduction exercises and physiotherapy exercises might be beneficial to improve the strength of the cough response. As voluntary cough starts with inspiration and therefore could cause aspiration, patients are taught the supraglottic swallow technique whereby they inspire air and breath hold before placing the bolus in the oral cavity, then swallow and then cough.

Reduced cough reflex sensitivity (or reduced urge) in individuals with dysphagia can lead to aspiration. Laryngeal and pharyngeal sensation can be assessed using the Fibreoptic Endoscopic Evaluation with Sensory Testing (FEEST) technique. This technique delivers short calibrated pulses of air to the laryngopharyngeal mucosa in attempt to stimulate the laryngeal adductor reflex (25). In practice, however, assessment of individuals with dysphagia typically focuses on the motor aspects of swallowing function with limited access to assessments for oral, pharyngeal and laryngeal sensation or cough/expiration reflex sensitivity. Knowledge of cough sensory sensitivity in these individuals

would enable a more specific application of treatment techniques and would ensure greater validity of presence or absence of reflexive cough during swallowing assessments.

Some individuals with CNSC and PVFM complain that eating triggers coughing. We argue that this phenomenon is distinct from oropharyngeal dysphagia. Careful case history assessment and cranial nerve examination may ascertain that cough and PVFM episodes are triggered by hyperresponsiveness of surrounding pharyngeal tissue such as when dry crumbly food touches the pharyngeal wall or during the oral phase when chewing solid food, rather than by aspiration. These individuals could be considered to have globus pharyngeus and extrathoracic airway hyperresponsiveness rather than primary oropharyngeal dysphagia. Of course dysphagia needs to be ruled out through careful cranial nerve examination and swallowing examination. It is our experience that it is safe to implement behavioural management of cough and PVFM prior to performing extensive swallowing investigations. It would be useful to clarify whether there is increased sensitivity of the expiration reflex in CNCS and PVFM.

Cough due to persisting gastroesophageal reflux

Gastroesophageal reflux is a common cause of CC (26). Although pharmaceutical and surgical treatment for reflux is usually beneficial, cough may persist after treatment (4). This is known as Chronic Refractory Cough (CRC). Reflux disease can be broadly classified into distal GER, laryngopharyngeal reflux without aspiration (LPR) or laryngopharyngeal reflux with aspiration (LPRA). In distal GER there is a reflex arc whereby stimulation of the distal oesophagus by gastric contents triggers a cough reflex reaction at the laryngeal level (27, 28). LPR occurs where there is laryngeal irritation by direct stimulation with gastric contents. LPRA occurs where there is micro aspiration of gastric contents. It could be argued that the pathogenesis of cough is different in distal GER, LPR and LPRA.

In LPR, a reflux event occurs with gastric contents reaching the level of the glottis (stimulus). The individual perceives the need to clear irritation from the throat (desire) and coughs thereby clearing gastric contents from the trachea (motor). They receive feedback via the central nervous system that the irritation has been cleared and that the urge has been satisfied (feedback). The individual then receives positive reinforcement for their cough response to the irritation (reward). If the LPR causes laryngeal inflammation (known as reflux laryngitis) inflammatory mediators could then act as the stimulus triggering further urge to cough and neural events.

In LPRA, the individual needs to prevent aspiration of gastric contents, hence has a biological urge to cough (urge). In LPRA there is a physiological need to cough and behavioural intervention to suppress the cough would not be advised. Intervention would primarily focus on reducing the stimulus through pharmaceutical treatment and lifestyle modification. Although many patients are given advice on lifestyle modification for reflux such as raising the head of the bed, avoiding certain foods, eating smaller meals, avoiding eating before lying down, it is our experience that they require a more structured intervention program to ensure adherence to this advice.

In distal GER and LPR, gastric contents do not penetrate the trachea (stage 1; stimulus). Although there is little chance of aspiration, the reflex arc may trigger an urge to cough (urge). The individual may perceive irritation in the larynx and wish to relieve the irritation by coughing (desire) but there is no physical need to cough. When the person coughs (motor) however there is no benefit to the airway. In fact the act of coughing may actually lower the tone in the lower oesophageal sphincter and increase the risk of subsequent reflux episodes. Coughing can also increase laryngeal irritation and thus act as a further stimulus to cough and increase the urge to cough thus perpetuating the cycle. The patient then receives feedback that the cough has occurred and that the irritation is temporarily relieved (feedback) and this feedback subsequently reinforces their response of coughing in response to irritation (reward). Management of stage 1 (stimulus) focuses on reducing the stimulus to cough, ensuring compliance with pharmaceutical treatment and lifestyle modification for reflux.

Conclusion

Behavioural intervention for cough should aim to address all six components of the Urge to Cough Model. This model strengthens the theoretical basis behind behavioural management of laryngeal conditions involving cough. It has the advantage of emphasising sensory components of conditions which previously had a predominantly motor focus. This model has the potential to expand and test further therapeutical strategies for cough.

References

1. Davenport P. Urge to cough: What can it teach us about cough. Lung 2008; 186 (S1): 107–111.

2. Mazzone SB, McLennan L, McGovern AE, Egan GF, Farrell MJ. Representation of capsaicin evoked urge to cough in human brain using functional MRI. Amer J Resp Crit Care Med 2007; 176 (4): 327–332.

3. Pratter M, Brightling C, Boulet L, Irwin R. An empiric integrative approach to the management of cough: ACCP evidence-based clinical practice guidelines. Chest 2006; 129 (Suppl 1): 222S–231S.

4. McGarvey L, Forsythe P, Heaney L, MacMahon J, Ennis M. Idiopathic chronic cough: A real disease or a failure of diagnosis. Cough 2005; 1 (9).

5. Haque R, Usmani O, Barnes P. Chronic idiopathic cough: A discrete clinical entity? Chest 2005; 127 (5): 1710–1713.

6. Colton J, Casper J, Leonard R. Understanding voice problems: A physiological perspective for diagnosis and treatment (3rd ed.) 3rd ed. Baltimore: Lippincott Williams & Wilkins 2006.

7. Vertigan A, Theodoros D, Gibson P, Winkworth A. Voice and upper airway symptoms in people with chronic cough and paradoxical vocal fold movement. J Voice 2007; 21 (3): 361–363.

8. Vertigan A, Theodoros D, Gibson P, Winkworth A. Efficacy of speech pathology management for chronic cough: A randomised, single blind, placebo controlled trial of treatment efficacy. Thorax 2006; 61: 1065–1069.

9. Vertigan A, Theodoros D, Winkworth A, Gibson P. Chronic cough: A tutorial for speech language pathologists. J Med Speech Lang Path 2007; 15: 189–206.

10. Verdolini-Marston K, Sandage M, Titze I. Effect of hydration treatments on laryngeal nodules and polyps and related voice measures. J Voice 1994; 8: 30–47.

11. Verdolini K, Titze I, Druker D. Changes in phonation threshold pressure with induced conditions of hydration. J Voice 1990; 4: 142–151.

12. Sivasankar M, Fisher K. Oral breathing increases Pth and vocal effort by superficial drying of vocal fold mucosa. J Voice 2008; 16 (2): 172–181.

13. Laukkanen A-M, Jarvinen K, Artkoski M, et al. Changes in voice and subjective sensations during a 45-min vocal loading test in female subjects with vocal training. Folia Phoniat Logopaed 2004; 56 (6): 335–346.

14. Brugman S. What's this thing called vocal cord dysfunction? Primary Crit Care Update 2006; 20 (26).

15. Ryan N, Gibson P. Cough reflex hypersensitivity and upper airway hyperresponsiveness in vocal cord dysfunction with chronic cough. Respirology 2006; 11 (Suppl 2): A48.

16. Ringsberg K, Segesten K, Akerlind I. Walking around in circles; The life situation of patients with asthma-like symptoms but negative asthma tests. Scand J Caring Sci 1997; 11: 103–112.

17. Morrison M, Rammage L, Emami A. The irritable larynx syndrome. J Voice 1999; 13 (3): 447–455.

18. Mathers-Schmidt B. Paradoxical vocal fold motion: A tutorial on a complex disorder and the Speech-Language pathologists role. Amer J Speech Lang Path 2001; 10: 111–125.

19. Altman K, Simpson C, Amin M, Abaza M, Balkissoon R, Casiano R. Cough and paradoxical vocal fold motion. Otolaryngol Head Neck Surg 2002; 127 (6): 501–511.

20. Morice A, Fontana GA, Sovijarvi ARA, et al. The diagnosis and management of chronic cough. Eur Resp J 2004; 24 (3): 481–492.

21. Blager F. Paradoxical vocal fold movement: Diagnosis and management. Curr Opinion Otolaryngol Head Neck Surg 2000; 8: 180–183.

22. Logemann JA. Evaluation and treatment of swallowing disorders. 2nd ed. Austin Texus: Pro-Ed; 1998.

23. Tatar M, Hanacek J, Widdicome J. The exspiration reflex from the trachea and bronchi. Eur Resp J 2008; 31: 385–390.

24. Hammond CS. Cough and aspiration of food and liquids due to oropharyngeal dysphagia. Lung 2008; 186 (Suppl 1): S35–S40.

25. Johnson P, Belafsky P, Potsma G. Topical nasal anesthesia and laryngopharyngeal sensory testing: A prospective, double-blind crossover study. Ann Otol, Rhinol, Laryngol 2003; 112: 14–16.

26. Irwin RS. Chronic cough due to gastroesophageal reflux disease: ACCP evidence based clinical practice guidelines. Chest 2006; 129: 80S–94S.

27. Ing A, Ngu M, Breslin A. Pathogenesis of chronic persistent cough associated with gastroesophageal reflux. Amer J Resp Crit Care Med 1994; 149: 160–167.

28. Rolla G, Colagrande P, Magnano M, et al. Extrathoracic airway dysfunction in cough associated with gastroesophageal reflux. J Allergy Clin Immunol 1998; 120 (1): 1–11.

Received June 26, 2009.
Accepted October 27, 2010.