The dyspnea experience: Nociceptive properties and a model for research and practice

Dyspnea has been defined as the unpleasant sensation of difficult breathing and the reaction to that sensation. Dyspnea research, however, has largely used a unidimensional, sensory model of dyspnea devoid of the affective and motivational dimensions that uniquely characterize this sensation in clinical populations. Dyspnea might be more comprehensively viewed as a nociceptive phenomenon which, like pain, has affective dimensions expressed as distress in response to aversiveness. A multidimensional, ecologic model of the dyspnea experience is presented that incorporates nociceptive sensation properties and is suggestive of new directions for dyspnea research uniquely relevant to nursing science.

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UNDER NORMAL conditions, breathing is primarily an automatic function that occurs without conscious awareness. In the presence of cardiopulmonary and selected neurologic disorders, however, breathing may be sensed as uncomfortable and stressful. This experience, termed dyspnea, is defined as the unpleasant sensation of difficult breathing and the human responses to that sensation.[1,2] In particular dyspnea accounts for a high proportion of the disability, impaired life quality, and human suffering experienced by the more than 23 million Americans with chronic obstructive pulmonary disease (COPD).[3,4]

Research into dyspnea has followed a sensory model not unlike that for the special senses such as vision and hearing. Central to this view is the investigation of receptors, afferent and efferent pathways, central ner-

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vous system processes, threshold perception, and stimulus tolerance.[5] Dyspnea is a complex process that is mediated by mechanoreceptors in the respiratory muscles, chest wall, airways, and lung parenchyma as well as by alterations in respiratory drive through chemoreceptor activity sensitive to hypoxia, hypercapnia, and pH. In addition, direct afferent pathways from the medullary respiratory controller to the cortex are thought to mediate the sensation of ventilatory effort, which has been described as the equivalent of dyspnea. This literature has provided a rich body of evidence as to basic physiologic mechanisms underlying breathing sensation and dyspnea.

However, since dyspnea is not a predictable function of an empirical stimulus, dyspnea might be more comprehensively viewed as a nociceptive phenomenon like pain, with motivational and affective dimensions expressed as distress in response to aversiveness, in addition to the sensory dimension. The purposes of this paper are to (a) review models of dyspnea and discuss knowledge derived from the sensory model, (b) describe dyspnea as a nociceptive phenomenon, and (c) suggest an ecologic model of dyspnea that is relevant to nursing science.

MODELS OF DYSPNEA

Researchers studying dyspnea often induce dyspnea in normal and clinical groups. Transcutaneous nerve blocks and agents such as curare have been used to paralyze the muscles of ventilation.[6,7] More recently, the application of internal and external loads to breathing have proven useful. An example of internal loading is the production of bronchoconstriction with inhaled drugs such as metacholine[8] as well as the enhancement of respiratory drive by breathing hypoxic or hypercapnic gaseous mixtures.[9] Widely used, external resistive or threshold loading is produced by having subjects breathe through an external device that necessitates the performance of greater inspiratory work. Inspiratory resistive and threshold loading have been used in normals and clinical groups and may also be used in conjunction with exercise to induce dyspnea.[10–12]

Incremental exercise testing using treadmill or cycle ergometry, long considered the gold standard for determining maximal exercise capacity, is widely used to study relationships between pulmonary physiologic events and the magnitude of dyspnea sensation under dynamic conditions.[13] In addition, standardized walking tests measuring distance walked over time as well as steady-state exercise and other nonincremental exercise tests have been used to produce and evaluate dyspnea.[14,15]

Nurse researchers studying dyspnea have primarily relied upon historic, retrospective indicators of naturally occurring dyspnea. Using visual analogue scaling and questionnaires, these studies have identified relationships between dyspnea and psychosocial variables.[16,17] Whereas such a cross-sectional approach provides useful information about overall dyspnea-related function, it allows only limited insights into personal, contextual, and physiologic variables comprising the dyspnea experience. More recently, nurse investigators have incorporated a more dynamic model of dyspnea induction, including exercise and inspiratory loading techniques.[18–20]

DYSPNEA AS A SENSORY PHENOMENON

Unlike the relatively discrete and well-defined sensory receptors, afferent pathways, and central nervous system locations known to comprise the special senses, the neuroanatomic substrate of dyspnea remains speculative. Dyspnea sensation is thought to arise during inspiration with inspiratory muscle activation and stimulation of sensory receptors in the airway, lung, chest wall, and diaphragm, including proprioceptive sense organs such as muscle spindles and tension-sensitive Golgi tendon organs. Carotid body and medullary chemoreceptors, sensitive to blood gas alterations, mediate output of the brainstem respiratory controller to inspiratory muscles. The resulting outgoing motor command, or respiratory drive, may also be directly sensed as breathing effort or dyspnea via a feedback loop to the cortex.[21–25] Refer to Fig 1 for a schematic representation of sensory receptors and effectors implicated in dyspnea sensation.

The conscious awareness of outgoing motor command to respiratory muscles under loaded conditions has been proposed and investigated as an all-encompassing explanation for dyspnea.[12,26,27] Consistent with this so-called "effort hypothesis," much of the

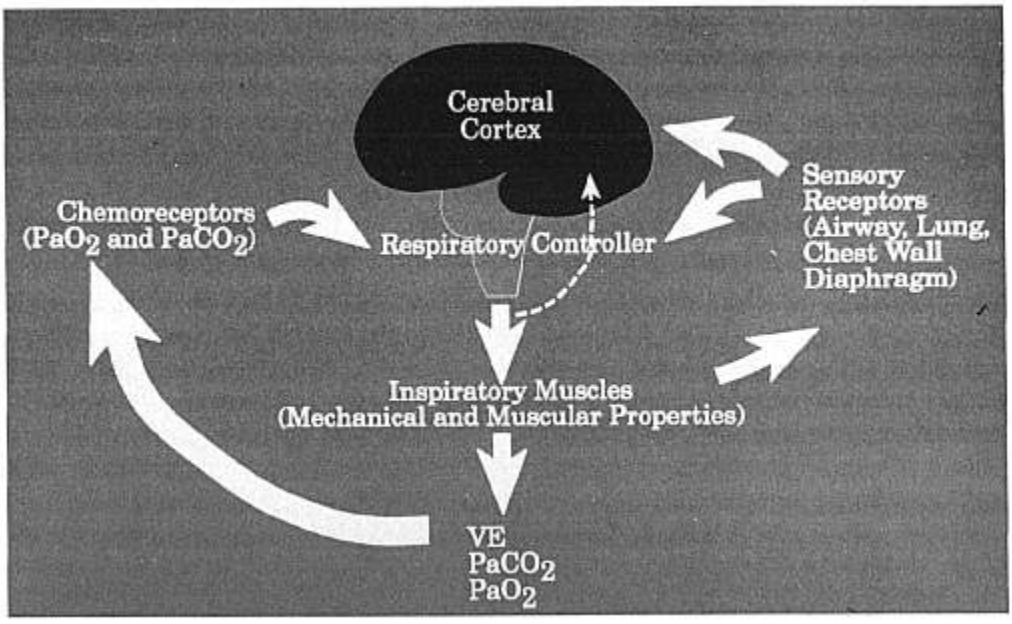

Fig 1. Sensory receptors and effectors implicated in dyspnea. Sensory receptors in the airway, lung, chest wall, and diaphragm are activated with inspiration. Inspiratory muscle function determines minute ventilation (VE) and arterial carbon dioxide and (in part) oxygen levels ($PaCO_2$, PaO_2). Peripheral and central chemoreceptors for PaO_2 and $PaCO_2$ mediate medullary respiratory controller contributions to respiratory drive. Outgoing motor command to inspiratory muscles may be sensed as dyspnea via cortical feedback (broken arrow). Adapted with permission from Tobin, M. Dyspnea: pathophysiologic basis, clinical presentation, and management. *Arch Intern Med.* 1990;150:1604–1613.

pulmonary physiologic literature is centered on dyspnea manifested as the perceived sensation of effort required to produce ventilation. In the absence of respiratory muscle fatigue, breathing effort sensation may be considered a function of the ratio of peak inspiratory pressure (PIP) achieved with tidal breathing to the maximal inspiratory pressure (MIP) possible.[12,21,28] The MIP depends on inspiratory muscle strength and is defined as the maximal negative pressure measured at the mouth during a maximal inspiratory effort from residual volume. Accordingly, breathing effort sensation has been observed to increase with minute ventilation, inspiratory flow rate, breathing frequency, inspiratory time expressed as a fraction of the total respiratory cycle and tidal volume expressed as a function of vital capacity.[12,21] Likewise, clinical observations of dyspneic patients reveal that during a ventilatory challenge, rapid, shallow breathing patterns are often utilized that tend to diminish peak pressures, possibly in an effort to diminish dyspnea.

Lack of consistent findings in studies using solely an effort model of dyspnea have cast doubt on its applicability to different experimental models of dyspnea and diverse clinical groups. In a recent study by Altose and associates,[27] the magnitude of breathing effort sensation was closely linked to minute ventilation during two dyspnogenic conditions of incremental exercise and voluntary hyperventilation but the sense of "shortness of breath" was not. Moreover, underlying pathophysiology may alter the degree to which ventilatory effort relates to dyspnea. McParland and his colleagues[29] found outgoing motor command (inspiratory muscle tension) explained 78% of breathing effort variance in his five COPD patients and only 29% of breathing effort variance in his five patients with congestive heart failure. More recently, COPD patients undergoing a threshold loading condition were able to separately quantify their sensation of breathing effort and discomfort, thus suggesting that dyspnea is comprised of separate effort and discomfort dimensions with implication for conceptualization and measurement.[30]

DYSPNEA AS A NOCICEPTIVE PHENOMENON

Like pain, dyspnea would therefore be better characterized as an unpleasant symptom that consists of subjective sensations signaling potential or actual impairment in biologic integrity as well as the human responses (cognitive, affective, motivational) to those sensations. Accordingly, dyspnea has a sensory component, but the experience of dyspnea incorporates cognitive interpretation of the event as threatening. Therefore, motivation to alleviate threat through behaviors such as slowing or cessation of activities that evoke dyspnea would be expected.

The study of pain as a blend of sensory and affective dimensions has provided evidence that cognitive processing affects sensory responses.[31–34] In this regard, expectation has been seen to influence affective responses to noxious stimulation.[32] Using

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visual analogue scales (VAS) for separate scalings of distress and sensation magnitude during an ischemic pain stimulus, subjects who received a description of expected sensations reported lower distress than subjects who received only a description of the experimental procedure. Sensation magnitude remained unchanged across stimulus conditions. A major contribution of this earlier study was the substantiation of sensation and distress components of the pain experience and the promise of this multidimensional view of pain over traditional unidimensional and pain threshold measurements. Price, Barrell, and Gracely[35] used cross-modality matching in which line lengths were matched to the perceived magnitude of an experimental heat stimulus. Subjects rated the magnitudes of both sensory and distress components of their pain, and although noxious temperatures were rated as less distressing (affective dimension) when the heat stimulus was preceded by a warning signal, sensory magnitude remained unchanged.

Using perceptual sensitivity measures and categorical descriptors, sensory and affective dimensions of pain have been separately measured. Perceptual sensitivity is determined by calculating the rate of rise in the sensory or affective magnitude of the response relative to increases in the physical magnitude of the pain stimulus. Magnitude estimation, as an example of measuring perceptual sensitivity, provides ratio level data to quantify verbal descriptors, commonly used in subjective pain reporting.[35,36] A categorical descriptive scale developed by Tursky and others[37] included three dimensions of the pain experience: intensity (strength, or sensory magnitude), reactivity (unpleasantness, or affective magnitude) and sensation (descriptive qualities). Elmore and Tursky[38] compared the effectiveness of hand warming and temporal pulse artery biofeedback for the treatment of migraine headache. Using this scale, they noted significant differences between the choice of reactivity descriptors in pretreatment and posttreatment diaries, whereas intensity descriptors were not altered by either of these therapeutics.

Another categorical descriptive scale, the McGill Pain Questionnaire, is perhaps the best known tool for measuring multiple dimensions of the pain experience in patients. In similar fashion to the Tursky scale, the McGill Pain Questionnaire quantifies pain along three dimensions: evaluative (overall intensity), affective (tension, fear, autonomic properties), and sensory (temporal, spatial, pressure, thermal, and the like).[34]

Within the dyspnea literature, few researchers have begun measurement of multiple dimensions of dyspnea.[9,28] Simon and associates have identified nine qualitative groupings of descriptive terms related to dyspnea experienced in normals and clinical groups.[22,39] These authors elicited dyspnea by a number of conditions, including breath-holding, carbon dioxide inhalation, inspiratory loading, and exercise. Their findings suggest that different clusters of descriptive terms may be induced by different respiratory stimuli and that patients in different diagnostic categories identify different qualitative properties of their dyspnea. More recently, Elliott and colleagues[40] identified 12 clusters of verbal descriptors for dyspnea that uniquely characterized three diagnostic categories and supported the findings of Simon et al.[39] Elliott noted further that this approach of clarifying the "language" of dyspnea, like

pain, may provide useful information regarding etiology and symptom management.

Supinski and colleagues[41] measured dyspnea dimensions using separate Borg category scales for breathing discomfort and breathing effort following administration of codeine in normal persons breathing against an inspiratory loading stimulus. In spite of a codeine-related analgesic effect documented by an increased tolerance for ice water immersion of the hand, neither breathing effort nor discomfort magnitudes during loaded breathing were altered by codeine. For the high-loading stimuli, however, codeine administration was associated with a lower initial sense of breathing discomfort than breathing effort compared to placebo administration. The latter effect was not statistically significant in view of the small sample size (N=8).

In recent work, Steele[30] reported that magnitude of perceived breathing effort measured by VAS was greater than perceived breathing discomfort across all loads (p=.05) using a threshold loading stimulus applied to 27 stable COPD patients. In addition, subjects evidenced greater perceptual sensitivity to breathing discomfort than breathing effort (p=.053) with greater magnitudes of discomfort relative to effort noted at higher threshold loads. Fig 2 shows the relative trajectories of breathing discomfort and effort with respect to inspiratory loads.

These studies suggest that persons with COPD can discern separate sensory and af-

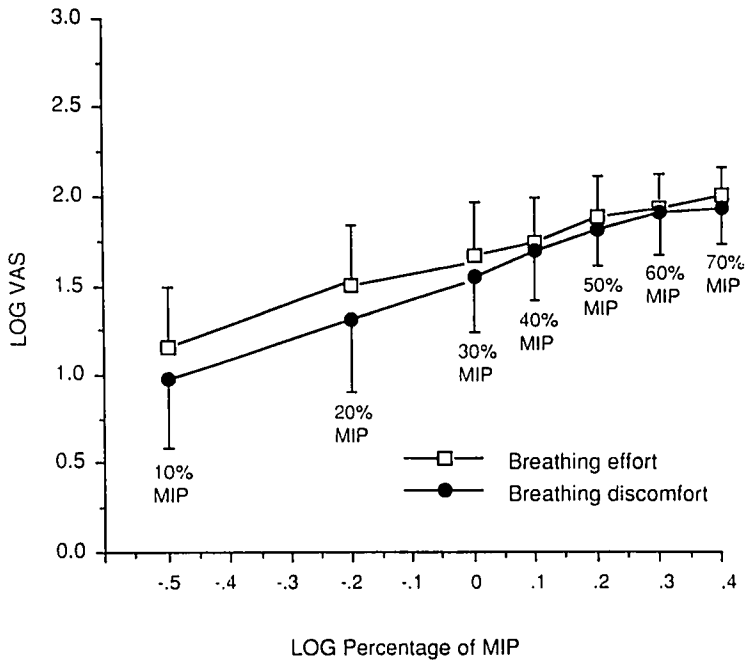

Fig 2. Trajectories and magnitude differences for breathing effort and discomfort (VAS data, log-transformed) in response to threshold loads expressed as a percentage of maximal inspiratory pressure (% MIP, log-transformed).

fective dimensions of their dyspnea. Measurement of both dimensions may provide a better understanding of dyspnea as a nociceptive phenomenon as well as providing new insights into mechanisms of dyspnea therapeutics.

Unlike a purely sensory approach, a nociceptive orientation acknowledges both sensory and affective dimensions of the experience of dyspnea. Two notions associated with noxious phenomena, namely perceptual sensitivity and tolerance, deserve evaluation for their potential in furthering our understanding of the dyspnea experience in clinical populations.

Perceptual sensitivity to dyspnea

Perceptual sensitivity to dyspnea has been defined earlier in this article as the proportional increase in reported dyspnea magnitude accompanying increased magnitude of a physical stimulus, such as an inspiratory load. Perceptual sensitivity measurement is a laboratory measure, necessitating random application of stimuli levels under controlled conditions. Perceptual sensitivity represents a quantifiable estimate of sensitivity to dyspnogenic stimuli.[42] The measurement of perceptual sensitivity to separate dimensions of dyspnea may provide a means to quantify these dimensions and determine relationships with other variables of interest, such as physical functioning and mood. For example, discomfort perceptual sensitivity measurement might be used, separately from breathing effort, to quantify the magnitude of discomfort as aversiveness imposed by inspiratory loading. The perceptual sensitivity to breathing discomfort may be a more potent motivator of biobehavioral adaptations than perceptual sensitivity to breathing effort. Such adaptations usually result in activity curtailment resulting in diminished physical and psychosocial functioning seen in dyspneic clinical groups.[3,43]

The separate measurement of perceptual sensitivity to both discomfort and effort in acute and chronic dyspnea could be useful, especially in the study of adaptation to dyspnea. When an individual first experiences acute dyspnea, the sensory dimension conveys a message of impending danger to the biologic integrity of the individual. This sensation is interpreted cognitively as a real or potential threat, and creates an affective response that motivates the individual to engage in behaviors that will reduce the threat, such as stopping the activity. At the same time, this psychologically interpreted information creates a physiologic response to threat mediated through the autonomic nervous system. Chronic, repetitive, or continuous experience with the noxious stimuli can lead to blunting of both the cognitive evaluation and the physiologic responses. Evidence exists that the perception of inspiratory loads are significantly blunted in COPD patients who experience chronic dyspnea compared to normals matched for age and to asthmatics who experience episodic dyspnea.[11,44] Separate measurement of both dyspnea dimensions might provide insight into selective modification of perceptual sensitivity to breathing effort and discomfort over time.

In one study, analysis of both perceptual sensitivity to breathing effort and discomfort (VAS)[45] revealed significant correlations between level of obstructive disease and within-subject relative effort/discomfort levels during inspiratory threshold loading ($r=-.50$, $p=.01$) and incremental exercise ($r_{Spearman}=-.51$, $p=.04$). In addition,

during exercise, subjects who perceived greater breathing effort than discomfort reported a higher level of overall physical functioning as measured by the Chronic Respiratory Disease Questionnaire (CRDQ) dyspnea subscale (r=.57, p=.02). These findings suggest that with more obstructive disease, perceived breathing effort increases more rapidly than perceived discomfort under increasing physical challenge. These findings might reflect a long-term adaptation wherein perception of the discomfort dimension of dyspnea is selectively reduced so as to preserve function.

The concept of differential effort and discomfort perceptual sensitivity might help to explain the wide variances in dyspnea reports and functional incapacitation among individuals with comparable levels of disease. One could hypothesize that patients with greater relative perceptive sensitivity to dyspnea discomfort than to dyspnea effort sensation would be more likely to suffer greater exercise intolerance than persons who experienced a less rapid rise in discomfort to effort with work.

Dyspnea tolerance

Dyspnea tolerance may be defined as the capacity of an individual to endure the sensation of dyspnea. Operationally, dyspnea tolerance could be measured as dyspnea duration; or as perceived dyspnea magnitude, indicated on a visual analogue or Borg scale, and associated work performance measured in kilopond meters (KPM) or oxygen consumption (VO_2). Self-report by recall of usual dyspnea tolerance in everyday activities (VAS) may also be a valid indicator of this concept.

Understanding what affects dyspnea tolerance has been an objective in those few studies in which it has been investigated. Alpher and his colleagues[46] used a distraction pain relief model of McCall and Malott[47] in the modulation of dyspnea tolerance. Using a breath-holding model of dyspnea, these authors were able to show that distraction lengthens breath-holding span and might be the fundamental mechanism underlying the therapeutic effects of relaxation and biofeedback on dyspnea.[48] Steele et al[49] demonstrated that dyspnea tolerance (VAS) in COPD patients was a better predictor of exercise capacity measured by an incremental exercise test than extent of obstructive pulmonary disease (forced expiratory volume in one second [FEV_1]). Current research by Carrieri and colleagues[20] using systematic desensitization and nurse-coached guided mastery is aimed at improving dyspnea tolerance in clinical groups via modification of symptom appraisal during exercise.

Dyspnea tolerance may be linked to dyspnea perceptual sensitivity and particularly to relative perceptual sensitivity to dyspnea dimensions. One might predict that different levels of perceptual sensitivity for breathing effort and discomfort are associated with different levels of dyspnea and exercise tolerance. In this regard, greater tolerance of dyspnea might be seen in patients reporting higher breathing effort perceptual sensitivity than breathing discomfort perceptual sensitivity during a physical challenge.

Dyspnea tolerance might be interpreted as a global index of individual adaptations with respect to dyspnea. A nociceptive approach to the dyspnea experience that includes the concepts of dyspnea perceptual sensitivity and tolerance could fit with an ecologic model of dyspnea.

AN ECOLOGIC MODEL OF DYSPNEA

An ecologic model of dyspnea provides a biopsychosocial framework for guiding nursing science. Unlike the linear, reductionistic biomedical model with notions of cause, disease, and cure, biopsychosocial approaches acknowledge the interactive effects of multivariate individual and environmental influences upon individual adaptations and health outcomes.

An ecologic model of dyspnea provides a biopsychosocial framework for guiding nursing science.

Factors that may impinge upon individuals and interact to influence biologic, experiential, and behavioral manifestations of dyspnea have been described in the literature.[2] These factors may be divided into environmental factors (risks and supports), and individual factors (vulnerability and resilience). They impact on biologic and behavioral outcome responses as outlined in the box entitled "Environmental and Individual Factors and Biobehavioral Outcome Responses Relating to the Dyspnea Experience."

Environmental factors are external to the individual and include risks and supports such as work and life style, family demands, living conditions, economic status, perceived social support, and proximity of dyspnea-generating environmental stimuli.

Individual factors are present within individuals and contribute to vulnerability-resilience and include variables such as disease severity and duration, trajectory of disease process, age and maturity levels, mood, perceived coping self-efficacy, and the nature of the dyspnea experience, including sensory and affective dimensions, perceptual sensitivity, and qualitative properties (symptom description), as well as temporal characteristics such as chronicity and acuity.

Biologic and behavioral outcome responses may be categorized as physiologic, experiential, or behavioral individual adaptations and health management strategies that mitigate dyspnea and contribute to level of everyday function. In chronic respiratory disease, level of function is the prime indicator of the success of individual adaptations and may be reflected by indicators of exercise capacity (physiologic and behavioral) and reported dyspnea tolerance (experiential).

This model is termed an ecologic model of dyspnea because it incorporates multiple feedback loops among internal and external variables and biobehavioral outcomes reflecting multiple dependencies. Refer to Fig 3 for a representation of this model.

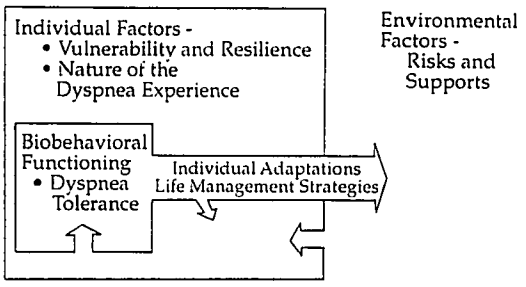

Fig 3. An ecologic model of the dyspnea experience. Environmental factors (risks and supports) interact with individual factors of vulnerability and resilience and the dyspnea experience to define biobehavioral functioning and, in particular, dyspnea tolerance. Individual adaptations and life management strategies influence individual and environmental factors.

Environmental and Individual Factors and Biobehavioral Outcome Responses Relating to the Dyspnea Experience

Environmental factors—risk and supports

- Work and family demands
- Living conditions
- Economic status
- Air quality/allergens
- Perceived social support

Individual factors—vulnerability and resilience

- Disease severity—symptom "load"
- Duration of disease
- Trajectory of disease process
- Age/maturity
- Mood-feeling states
- Perceived coping self-efficacy
- The nature of the dyspnea experience
  - Sensory and affective magnitudes—nociceptive properties
  - Perceptual sensitivity
  - Qualitative properties
  - Temporal properties (continuous/episodic)

Biological and behavioral outcome responses

- Individual adaptations
- Life management strategies
- Dyspnea tolerance

Adapted with permission from Carrieri VE, Janson-Bjerklie S, Jacobs S. The sensation of dyspnea: a review. *Heart Lung*. 1984;13(4):436–443. © 1984.

It is reasonable to suggest that both perceptual sensitivity and tolerance to dyspnea, which are linked, might be influenced by various individual person factors. Abundant evidence exists that dyspnea varies with mood and response style.[4,43] Mood is best depicted as those affective responses that wax and wane with the internal and external demands impinging on the person. More enduring affective states such as depression and hopelessness might be even more predictive of decreased physical functioning and other negative behavioral responses possibly mediated through impaired dyspnea tolerance.

Dudley and Pitts-Poarch[43] have observed that persons with dyspnea may be separated into two groups: dyspnea with depression (and hypoventilation) and dyspnea with anxiety and anger (and hyperventilation). When followed over time, people with COPD did not consistently fall into one

group or the other, but instead the dyspnea experience varied with specific emotionality from day to day. Therefore, mood state as well as more enduring behavioral characteristics may constitute potent mediating factors in the experience of dyspnea. Chronic dyspnea related to emotional activation such as fear or anger results in a kind of emotional blunting and systematic avoidance of experiences likely to precipitate such activation and dyspnea. Withdrawal from activities likely to cause dyspnea may contribute to deconditioning, emotional isolation, and further impairment of dyspnea tolerance and exercise capacity. Whereas these ideas are intuitively appealing, they have yet to undergo rigorous empirical validation.

Another concept linked to the affective dimension of dyspnea within a nociceptive model is perceived coping self-efficacy.[50] Perceived coping self-efficacy is the degree to which individuals believe they can cope successfully with challenging events. Applied to dyspnea, a high degree might be associated with higher levels of effort expenditure, activity, and greater dyspnea tolerance.

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In sum, dominant research approaches, mainly by scientists in other disciplines, have selectively placed emphasis on a unidimensional view of dyspnea. While contributing significantly to knowledge of the neural, chemical, and muscular bases of breathing sensation, such research has not adequately explained features of dyspnea evidenced in various clinical groups. The argument has been made in this article that the phenomenon of dyspnea be depicted within a nociceptive framework based on considering two dimensions of sensory magnitude, breathing effort and breathing discomfort. A nociceptive approach to characterizing dyspnea, coupled with a person-environment ecologic framework accounting for biopsychosocial contributing factors, is suggested for further development and study of clinical nursing therapeutics for this common and challenging symptom of importance to nursing practice.

REFERENCES

1. Comroe J. Dyspnea. *Mod Concepts Cardiovasc Dis.* 1956;25(9):347–349.
2. Carrieri V, Jansen-Bjerklie S, Jacobs S. The sensation of dyspnea: a review. *Heart Lung.* 1984;13(4):436–447.
3. McSweeney A, Grant I, Heaton R, Adams K, Timms R. Life quality of patients with chronic obstructive pulmonary disease. *Arch Int Med.* 1982;142:473–478.
4. Kinsman R, Yaroush R, Fernandez E, Dirks J, Schocket M, Fukuhara J. Symptoms and experiences in chronic bronchitis and emphysema. *Chest.* 1983;83(5):755–761.
5. Stevens S. *Psychophysics: An Introduction to its Perceptual, Neural, and Social Prospects.* New York, NY: Wiley; 1975.
6. Campbell E, Gandevia S, Killian K, Mahutte C, Rigg J. Changes in the perception of inspiratory resistive loads during partial curarization. *J Physiol.* 1980;309:93–100.
7. Guz A, Noble M, Widdicombe J, Trenchard D, Mushin W, Makey A. The role of vagal and glossopharyngeal afferent nerves in respiratory sensation control of breathing and arterial pressure regulation in conscious man. *Clin Sci.* 1966;30:161–170.
8. Peat J, Salome C, Bauman A, Toelle B, Sachinger S, Woolcock A. Repeatability of histamine bronchial challenge and comparability with methacholine bronchial challenge in a population of Australian schoolchildren. *Am Rev Respir Dis.* 1991;144(2):338–343.
9. Chonan R, Mulholland M, Chemiack N, Altose M. Ef-

fects of voluntary constraining of thoracic displacement during hypercapnia. *J Appl Physiol.* 1987;63(5):1822–1828.

10. Chen H, Tang Y. Effects of the menstrual cycle on respiratory muscle function. *Am Rev Respir Dis.* 1989;140(5):1359–1362.
11. Gottfried S, Redline S, Altose M. Respiratory sensation in chronic obstructive pulmonary disease. *Am Rev Respir Dis.* 1985;132:954–959.
12. El-Manshawi A, Killian K, Summers E, Jones N. Breathlessness during exercise with and without resistive loading. *J Appl Physiol.* 1986;61(3):896–905.
13. Killian K, Jones N. The use of exercise testing and other methods in the investigation of dyspnea. *Clin Chest Med.* 1984;5(1):99–108.
14. Guyatt G, Thompson P, Berman L, et al. How should we measure function in patients with chronic heart and lung disease? *J Chron Dis.* 1985;38(6):517–524.
15. O'Neill P, Stark R, Allen R, Stretton T. The relationship between breathlessness and ventilation during steady-state exercise. *Bull Physiopathol Respir.* 1986;22:247–250.
16. Gift A. *Physiological and Psychological Factors Related to Dyspnea in Subjects with Chronic Obstructive Pulmonary Disease.* Baltimore, Md: University of Maryland; 1984. Dissertation.
17. Janson-Bjerklie S, Carrieri V, Hudes M. The sensations of pulmonary dyspnea. *Nurs Res.* 1986;35(3):154–159.
18. Larson J, Kim M, Sharp J, Larson D. Inspiratory muscle training with a pressure threshold breathing device in patients with chronic obstructive pulmonary disease. *Am Rev Respir Dis.* 1988;138:689–696.
19. Nield M. *The Relationship Between Dyspnea and Respiratory Muscle Function in Subjects with Chronic Respiratory Disease.* Chicago, Ill: University of Illinois at Chicago; 1989. Dissertation.
20. Carrieri V, Stulgarg S, Murray J. *Use of desensitization in the relief of dyspnea.* Presented at American Thoracic Society Annual Meeting; May 13, 1991; Anaheim, Calif.
21. LeBlanc P, Bowie D, Summers E, Jones N, Killian K. Breathlessness and exercise in patients with cardiorespiratory disease. *Am Rev Respir Dis.* 1986;133:21–25.
22. Simon P, Basner R, Schwartzstein R, Weinberger S, Fenci V, Weiss J. Oral mucosal stimulation modulates intensity of breathlessness induced in normal subjects. *Am Rev Respir Dis.* 1988;137(4):387.
23. Paintal A. The mechanism of excitation of type J receptors and the J reflex. In: Porter R, ed. *Breathing: Hering Breuer Centenary Symposium.* London, England: Churchill; 1970.
24. Stark R, Gambles J, Lewis J. Methods to assess breathlessness in healthy subjects: a critical evaluation and application to analyse the acute effects of diazepam and promethane on breathlessness induced by exercise or exposure to raised levels of carbon dioxide. *Clin Sci.* 1981;61:429–439.
25. Puddy A, Jung D, Giesbrecht G, Sanii R, Younes M. Mechanism of detection of added respiratory loads. Presented at Aspen Lung Conference; June 1989; Aspen, Colo.
26. Killian K, Gandevia S, Summers E, Campbell E. Effect of increased lung volume on perception of breathlessness, effort, and tension. *J Appl Physiol.* 1984;57:686–691.
27. Altose M, Chonan T, Mulholland M. Comparison of the sensation of effort of breathing and shortness of breath during exercise and voluntary hyperventilation. *Am Rev Respir Dis.* 1988;137(4):387.
28. Cherniack N, Altose M. Mechanisms of dyspnea. *Clin Chest Med.* 1987;8(2):207–214.
29. McParland C, Every B, To T, Orchard R, Gallagher C. Relation between dyspnea and inspiratory motor output in chronic obstructive pulmonary disease (COPD) and chronic heart failure (CHF). *Am Rev Respir Dis.* 1988;137(4):387.
30. Steele B, Shaver J, Hildebrandt J, Schoene R, Tyler M, Betrus P. Dimensions of dyspnea during inspiratory threshold loading (ITL) in chronic obstructive pulmonary disease (COPD). *Am Rev Respir Dis.* 1991;143(4):A595.
31. Melzack R, Chapman C. Psychologic aspects of pain. *Postgrad Med.* 1973;53(6):69–75.
32. Johnson J. Effects of accurate expectations about sensations on the sensory and distress components of pain. *J Pers Soc Psychol.* 1973;27(2):261–275.
33. Gracely R, McGrath P, Dubner R. Ratio scales of sensory and affective verbal pain descriptors. *Pain.* 1978;5:5–18.
34. Melzack R. Measurement of the dimensions of the pain experience. In: Bromm R, ed. *Pain Measurement in Man: Neurophysiological Correlates of Pain.* New York, NY: Elsevier; 1984.
35. Price D, Barrell J, Gracely R. The psychological analysis of experiential factors that selectively influence the affective dimension of pain. *Pain.* 1980;8:137–149.
36. Price D, McGrath P, Rafii A, Buckingham B. The validation of visual analogue scales as ratio scale measures for chronic and experimental pain. *Pain.* 1983;17:45–56.
37. Tursky B, Jamner L, Friedman R. The pain perception profile: a psychophysical approach to the assessment of pain report. *Behav Ther.* 1982;13:376–394.
38. Elmore A, Tursky B. A comparison of two psychophysiological approaches to the treatment of migraine. *Headache.* 1981;21:93–101.
39. Simon P, Schwartzstein R, Weiss J, Fenci V. Teghtsoonian M, Weinberger S. Distinguishable types

of dyspnea in patients with shortness of breath. *Am Rev Respir Dis*. 1990;142(5):1009–1014.
40. Elliott M, Adams L, Cockcroft A, Macrae K, Murphy K, Guz A. The language of breathlessness: use of verbal descriptors by patients with cardiopulmonary disease. *Am Rev Respir Dis*. 1991;144(4):826–832.
41. Supinski G, DiMarco A, Bark H, Chapman K, Clary S, Altose M. Effect of codeine on the sensations elicited by loaded breathing. *Am Rev Respir Dis*. 1990;141(6):1516–1521.
42. Mahler D, Rosiello R, Harver A, Lentine T, McGovern J, Daubenspeck J. Comparison of clinical dyspnea ratings and psychophysical measurements of respiratory sensation in obstructive airway disease. *Am Rev Respir Dis*. 1987;135(6):1229–1233.
43. Dudley D, Pitts-Poarch A. Psychophysiologic aspects of respiratory control. *Clin Chest Med*. 1980;1(1):131–143.
44. Gottfried S, Altose M, Kelsen S, Cherniack N. Perceptions in changes in airflow resistance in obstructive pulmonary disorders. *Am Rev Respir Dis*. 1981;124:566–570.
45. Steele B. *Dimensions of Dyspnea in Chronic Obstructive Pulmonary Disease: A Nociceptive Model*. 1991. Seattle, Wash: University of Washington; 1991. Dissertation.
46. Alpher V, Nelson R, Blanton R. Effects of cognitive and psychomotor tasks on breath-holding span. *J Appl Physiol*. 1986;42:1149–1152.
47. McCall K, Malott J. Distraction and coping with pain. *Psychol Bull*. 1984;95:516–533.
48. Alpher V, Tobin M. Psychologic and pathophysiological factors in respiratory disorders. In: Russell M, ed. *Stress Management for Chronic Disease*. New York, NY: Pergamon. In press.
49. Steele B, Shaver J, Hildebrandt J, Schoene R, Tyler M. Patterns of dyspnea development during progressive exercise in chronic obstructive pulmonary disease (COPD). *Am Rev Respir Dis*. 1991;143(4):A595.
50. Bandura A. Self-efficacy: toward a unifying theory of behavioral change. *Psychol Rev*. 1977;84(2):191–214.