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Anxiety Sensitivity and Subjective Feelings of Dyspnea in Asthmatic Children

by

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Abstract

The relationships between anxiety sensitivity (i.e, the fear of the consequences of anxiety symptomatology) and self-reported dyspnea, general anxiety, and physiological response in 38 children who presented to an emergency room for treatment of acute asthma were investigated.

At time of treatment, subjects rated their subjective degree of dyspnea; oxygen saturation, an objective measure of airway obstruction, was also recorded. At a follow-up session, subjects underwent pulmonary function testing and completed measures of dyspnea, anxiety sensitivity, and general anxiety.

The results failed to support the hypothesis linking anxiety sensitivity and dyspnea; no relationship was found between anxiety sensitivity and self-reported dyspnea and physiological response. Multiple regression analyses indicated that neither anxiety sensitivity nor general anxiety scores explained a significant amount of variance on measures of dyspnea. Analyses further indicated that subjects were as likely to overrate their degree of dyspnea as they were to underrate it.

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Table of Contents

List of Tables

Anxiety Sensitivity and Subjective Feelings of Dyspnea in Asthmatic Children

Anxiety sensitivity is described by Reiss' expectancy model of fear, anxiety, and panic (1991) as one of two components of the fear of fear. The other component, anxiety expectation, refers to an anticipated outcome that is linked with a feared object or situation; such as, "I expect that I will have an asthma attack if I exercise strenuously". Anxiety sensitivity precedes anxiety expectation in that it provides the impetus for fearing the anticipated outcome; for example, "I fear that an asthma attack will kill me". Reiss' (1991) model provides a basis for understanding a wide variety of fears and phobias by allowing for individual differences in the severity and type of both expectation and sensitivity.

An individual who fears the outcome of an anxiety-provoking event will react with fear to any signs that signal the beginning of that event. Thus, anxiety sensitivity has come to be described as fear of the consequences of anxiety symptomatology, aside from their immediate unpleasantness (Holloway & McNally, 1987; Taylor, Koch, McNally, & Crockett, 1992). As the fear is excessive (e.g., "I fear that I will have a heart attack because my heart is beating quickly"), it has been attributed to a misinterpretation of bodily sensations (Asmundson, Norton, Wilson, & Sandler, 1994) and, when it occurs repeatedly, to interoceptive conditioning (Reiss, Peterson, Gursky, & McNally, 1986).

The origin of anxiety sensitivity has been examined by McNally and his colleagues (Donnell & McNally, 1989; Donnell & McNally, 1990; Holloway & McNally, 1987) in studies of the effect of hyperventilation on college students who had and who had not previously experienced panic attacks. The results of these studies indicated that high scores on the Anxiety Sensitivity Index (ASI) were indeed closely associated with panic attacks but were not found solely in subjects who had directly experienced panic attacks in the past. They further noted that subjects high in anxiety sensitivity were likely to report increases in sensations unrelated to those induced by hyperventilation. They concluded that anxiety sensitivity is the result of neither a biological defect nor a history of panic attacks. Research suggests that anxiety sensitivity is a dispositional construct which leads to increased vigilance and anxious self-preoccupation, particularly in situations where the anxiety-provoking event is more likely to occur (Shostak & Peterson, 1990).

There has been a debate in the literature concerning the existence of anxiety sensitivity as a unique construct. Proponents (e.g., McNally, 1989) maintain that anxiety sensitivity is distinguishable from trait anxiety while their opposition (e.g., Lilienfeld, Jacob, & Turner, 1989) maintains that it is merely a component of trait anxiety. Trait anxiety is generally viewed as an all-encompassing construct, characterized by an anxious response to stressors in general, while anxiety sensitivity represents a more specialized tendency to respond anxiously to the symptoms of arousal. As such, a person high in trait

anxiety may be made anxious in response to stressors without having an additional fear of the anxiety symptoms. Reiss (1991) conducted a meta-analysis of 11 studies with different populations and found that anxiety sensitivity and trait anxiety tend to correlate, but only moderately (*r* squares ranged from 0 to 36%). Other researchers have found anxiety sensitivity measures to explain additional variance on scales of fear that anxiety measures could not (McNally, 1989; Rapee & Medoro, 1994; Silverman et al., 1991). These findings lend support to the most current view of anxiety sensitivity as a distinct construct that overlaps with trait anxiety but is not entirely subsumed by it (Clark, Watson, & Mineka, 1994).

Several studies have demonstrated that anxiety-sensitive individuals subjectively tend to report more anxiety symptomatology and greater increases in cognitive anxiety, independent of the level of physiological arousal (Asmundson et al., 1994; Benton & Allen, 1996; Carr, Lehrer, Rausch, & Hochron, 1994; Holloway & McNally, 1987; Shostak & Peterson, 1990). Holloway and McNally (1987), for example, using hyperventilation to induce autonomic arousal in a sample of college students, found that anxiety-sensitive individuals responded with greater levels of subjective anxiety although all subjects experienced equal levels of arousal. Shostak and Peterson (1990) likewise found the perception of physiological symptoms to be dependent on anxiety sensitivity but not on the actual level of physiological arousal following a performance task.

That anxiety sensitivity has been found to be independent of the actual level of physiological arousal does not imply that it cannot affect physiological response. The increase in subjective reports of anxiety symptomatology in anxiety-sensitive individuals is usually attributed to a cognitive pattern of hypervigilance to internal cues and a tendency to exaggerate the severity and importance of those cues. Such self-focusing behaviour has been found to potentiate the level of anxiety experienced (Shostak & Peterson, 1990) and tends reliably to exacerbate and prolong the state of arousal (Ingram, 1990). Shostak and Peterson (1990), while finding anxiety sensitivity to be independent of physiological arousal, also noted that individuals with anxiety-sensitive dispositions recovered more slowly physiologically following stressful situations. High levels of anxiety sensitivity have also been associated with interpersonal difficulties similar to those experienced by clinical panickers and those with agoraphobia (Benton & Allen, 1996).

Shostak and Peterson (1990) noted in their discussion that a group of interest for further study is composed of individuals with extremely low anxiety-sensitive dispositions. They posit that these individuals may not view physiological arousal as a negative experience or, in fact, may not process arousal at all. While they do not provide an explanation for the existence of such a group, one possibility is that those who score extremely low on a measure of anxiety sensitivity are repressors. As described by Steiner, Higgs, Fritz, Laszlo, and Harvey (1987), repressors are those individuals who report

low anxiety on rating scales but are paradoxically high on autonomic and behavioural measures of anxiety. They are distinguished from those who are truly low-anxious during exposure to stressful situations by the discrepancy between their self-reports and measures of arousal.

Research indicates that females report higher anxiety while males are more likely to be repressors and to respond defensively to anxiety rating scales (Buckelew & Hannay, 1986). There is also evidence that females maintain a higher level of synchrony between indicators of anxiety (i.e., autonomic, verbal, and behavioural), evidencing fewer paradoxical reactions (Leventhal, as cited in Ben-Zur & Zeidner, 1988). Several explanations are offered for the impact of gender, from socialization to differential processing of information to greater awareness on the part of females. Indeed, females have been found to be more likely to self-focus and to initiate negative affect as a response to stressful events (Ingram, 1990).

Anxiety sensitivity is measured by assessing an individual's beliefs regarding the consequences of physical, cognitive, and social symptoms associated with anxious symptomatology (Peterson & Heilbronner, as cited in Silverman, Fleisig, Rabian, & Peterson, 1991). Two measures of anxiety sensitivity in current use are the ASI and its modified counterpart for children, the Childhood Anxiety Sensitivity Index (CASI). Both the ASI and the CASI have been found to possess sound reliability and internal consistency for both clinical and normal samples (McNally & Lorenz, 1987; Rabian, Peterson,

Richters, & Jensen, 1993; Reiss et al., 1986; Silverman et al., 1991). McNally and Lorenz (1987) also found the ASI to be strongly correlated with two questionnaires designed to measure the fear of fear (Arrindell, 1993): the Agoraphobic Cognitions Questionnaire (ACQ; $r(44) = .66$, $p < .05$) and the Body Sensations Questionnaire (BSQ; $r(44) = .64$, $p < .05$), "indicating the coherence of the underlying construct of fear of anxiety" (Chambless & Gracely, 1989, p. 10).

The CASI was developed by Peterson and Reiss (as cited in Silverman et al., 1991) in an effort to create an instrument which would measure the construct of anxiety sensitivity in children. The CASI is very similar to the ASI, except that some of the items on the ASI were modified so that they would be more understandable to children and two items were added to the original scale (Silverman et al., 1991). Use of the CASI has been documented in the literature only twice: one study examined its psychometric properties in a sample of 85 school children (Silverman et al., 1991) and another examined its ability to differentiate clinically anxious children from nonanxious children in a sample of 201 children of military families (Rabian et al., 1993).

Taylor, Koch, and McNally (as cited in Clark et al., 1994) reported that all anxiety disorders, with the exception of simple phobias, are associated with elevated levels of anxiety sensitivity. Other disorders which are primarily biological in origin but which may be strongly influenced by anxiety and anxiety sensitivity - such as asthma - have been given little attention in the

anxiety sensitivity literature.

Asthma is a very serious childhood disorder capable of negatively influencing and limiting many aspects of a child's life, affecting development, and, in approximately 2.4% of childhood asthma cases, causing death (Jurenec, 1988). Asthma is the most common chronic disease of childhood (Davis, as cited in McNabb, Wilson-Pessano, & Jacobs, 1986), its prevalence estimated between 5-10% for children under the age of 15, with males affected 2 to 3 times as frequently as females (Fritz, 1983). Morbidity is also significant, with asthma the leading cause of school days lost each year in the United States due to chronic illness (Weiss, 1994), accounting for approximately 25% of chronic illness absences (Fritz, 1983).

Asthma is of particular interest from a psychological viewpoint since it is a disorder of the respiratory system, the only system in the body that serves an automatic vegetative function yet is also under voluntary control (Naifeh, 1994). While asthma is considered a primarily biological condition, characterized by episodic and reversible airways obstruction, research suggests that psychological factors influence the frequency, severity, and impact of asthma (Fritz, 1983; Weiss, 1994). Measurable physiological components of the asthma reaction include forced expiratory volume, arterial oxygen saturation, peak expiratory flow, forced thoracic breathing (due to the contraction of the abdominal muscles; Naifeh, 1994), and elements of autonomic arousal, such as increased heart rate.

In the past, asthma was understood to be a constriction of bronchial airways which impeded the passage of air, caused by airway hyperreactivity to various internal and external stimuli (Celano & Geller, 1993). Extrinsic asthma was characterised by a hyperreactivity to external stimuli, such as pollens, exercise, and changes in the weather; in particular, shifts to colder temperatures. Alternatively, asthma could be intrinsic: caused by the individual's overresponse to otherwise mild irritants (Celano & Geller, 1993).

Recently, the pathophysiology of asthma has been considered to be more complex. It is recognised as multifactorial in origin and comprising several physiological responses, including: bronchial obstruction (the blockage of the large airways which lead from the lungs to the mouth and nose), bronchial and pulmonary inflammation, and increased bronchial and pulmonary mucoid secretions (the body's natural lubricating fluid becomes thicker and more tenacious; Celano & Geller, 1993).

The initial bronchial response to irritating stimuli, referred to as the 'early asthmatic response' (EAR; Celano & Geller, 1993), occurs within 30 minutes and will last up to two hours. During this time, inflammatory cells are released into the airways, damaging lung tissue, creating debris, and resulting in a reduction in air flow, or bronchoconstriction (Celano & Geller, 1993; Fried & Grimaldi, 1993; Weiss, 1994). Bronchoconstriction involves the contraction of bronchial muscles, swelling of bronchial tissues, and secretion of excess mucous, all of which impede air exchange in the lungs (Janson-Bjerklie,

Ruma, Stulbarg, & Kohlman-Carrieri, 1987). These changes predispose the individual to an increased susceptibility to irritants (bronchial hyperresponsiveness) which ultimately leads to further inflammation and bronchoconstriction and makes the process of breathing increasingly difficult (Celano & Geller, 1993).

In addition to responses to specific intrinsic and extrinsic stimuli, asthma may also be triggered by psychologic stimuli, including a wide range of strong emotions, such as: anxiety, depression, guilt, anger, frustration, anticipatory excitement, and joy (Cohen & Lask, 1983; Yellowlees & Kalucy, 1990). While no amount of emotional distress will cause an asthma attack in an individual who is without an 'immunophysiological vulnerability' to asthma, emotional factors play a key role in determining whether attacks will occur in those who possess such a vulnerability (Celano & Geller, 1993; Cohen & Lask, 1983). Indeed, research has demonstrated that asthma attacks can be reliably induced in asthmatic individuals by discussing information with them which they have previously identified as anxiety-provoking (Dekker & Groen, as cited in Cohen & Lask, 1983).

Cohen and Lask (1983) contend that the type of emotion experienced by an asthmatic individual contributes less to the onset of an asthma attack than does the manner in which that emotion is expressed. They contend that emotion which is suppressed or inadequately expressed is linked to the onset of an asthma attack and that asthmatics, as a group, tend to express their

feelings less easily than do members of the general population. If this is the case, then asthmatics might be expected to deny anxiety even when experiencing an acute asthma attack.

As psychological factors can contribute to the onset of an asthma attack, so too can the experience of living with asthma affect a child's psychological functioning. Children with asthma are required to develop some degree of internal vigilance in order to respond to symptoms of their condition (e.g., chest tightening) with medications or by reducing exercise or exposure to irritants, as necessary. Internal vigilance, however, can be anxiety-provoking and may lead to somatic preoccupation and increased somatic complaints. Furrow, Hambley, and Brazil (as cited in Celano & Geller, 1993) found that somatic complaints, as measured by the Child Behavior Checklist (CBCL; Achenback & Edelbrock, 1986), were indeed the predominant symptom among a sample of children who had been hospitalised for asthma.

Children with asthma may be at risk for a number of functional impairments, such as restricted physical activity and impaired physical conditioning, depending on the severity and nature of their illness (Celano & Geller, 1993). In addition, epidemiological studies have revealed an association between chronic illness in childhood and poor psychological adjustment (e.g., Cadman, Boyle, Szatmari, & Offord, as cited in Celano & Geller, 1993). Decreased psychological adjustment is usually manifested as increased behavioural problems and less resiliance in meeting social challenges

(MacLean, Jr., Perrin, Gortmaker, & Pierre, 1992). Both specific illness variables (e.g., the need to avoid activities which promote breathlessness) and background variables independent of chronic illness (e.g., socioeconomic status, availability of resources) may contribute to poor medical management of the illness and, consequently, to poor psychological adjustment among children with asthma (MacLean et al., 1992).

Children whose psychological adjustment to their asthmatic conditions is maladaptive are also at risk for a range of psychosocial difficulties. Psychological maladjustment has been significantly related to poor treatment adherence, school absence, poor school performance, sleep disturbances, disruption in the parent-child relationship, and death due to asthma (Celano & Geller, 1993; Colland, 1993; Davis & Wasserman, 1992; Richards, 1994). A 'vicious cycle' of asthma may ensue, in which asthma contributes to psychosocial problems or somatic preoccupation which, in turn, may trigger or increase asthmatic symptoms and attacks (Richards, 1994).

Psychological factors may play yet another role in an individual's experience of asthma since asthma's primary symptom - uncomfortable, laboured breathing, referred to as dyspnea - is a subjective experience. The experience of dyspnea is often likened to that of pain (Ley, 1989). It cannot be measured in terms of the metrics of a physical scale (Comroe, cited in Ley, 1989) but is rather experienced subjectively and involves an individual's perception of airways obstruction and his or her reaction to that perception

(Heim, Blaser, & Waidelich, 1972).

Since breathing is a basic physiological function, acute dyspnea is a frightening experience; some fear in response to the sensation of partially occluded airways and laboured breathing is 'normal' (Carr, Lehrer, & Hochron, 1995; Yellowlees & Kalucy, 1990). Asthmatics experiencing severe anxiety, however, may hyperventilate or have feelings of panic which are misconstrued as indicators that the asthma attack is worsening (Yellowlees & Kalucy, 1990). If this occurs, dosages of asthma medications may be inappropriately increased and may make patients feel physiologically more anxious (Carr, Lehrer, & Hochron, 1995; Yellowlees & Kalucy, 1990). The duration and severity of an asthma attack, then, is partially dependent on individuals' perceptions regarding the causes and consequences (anxiety sensitivity) of dyspnea (Carr, Lehrer, & Hochron, 1995; Steiner et al., 1987; Yellowlees & Kalucy, 1990).

Dyspnea is the primary symptom of asthma and is often used as a marker of disease severity by both doctors and patients (Kinsman, Luparello, O'Banion, & Spector, 1973). The intermittent quality of bronchoconstriction makes the subjective report of the patient important during treatment since it is often the first indication of acute difficulty (Kinsman et al., 1973). However, since the experience and reporting of dyspnea is dependent on motivational and affective factors (Kohlman-Carrieri, Kieckhefer, Janson-Bjerklie, & Souza, 1991; Steele & Shaver, 1992), many patients tend to either over- or

underestimate the degree to which their airways are occluded (Heim, Blaser, & Waidelich, 1972; Rubenfeld & Pain, 1976), providing little correspondence between subjective and objective measures of airways obstruction. Dyspnea research has generally failed to find consistent physiological differences between patients reporting high and low levels of dyspnea (Steele & Shaver, 1992).

There are several objective measures of dyspnea, some of which are confounded in that they require cooperation and effort on the part of the patient. If patients believe that they are incapable of forced expiration, of if they are too young or too ill to cooperate (Geelhoed, Landau, & LeSouef, 1990), it will be impossible to obtain accurate measurements. Two objective measures commonly used during the acute asthma episode are peak expiratory flow (PEF) and arterial oxygen saturation (SaO_2). PEF is a measure of forced spirometry, requiring maximal inspiration followed by rapid, forceful, and complete expiration (Wanger, 1992). Arterial oxygen saturation is obtained noninvasively by pulse oximetry using the finger. It is the ratio of the amount of oxygenated hemoglobin to the total available amount of hemoglobin (Ruppel, 1991). When airways are obstructed, both expiratory flow and the level of arterial oxygen saturation are decreased from normal values (Geelhoed, Landau, & LeSouef, 1990).

Geelhoed, Landau, and LeSouef (1990) determined that arterial oxygen saturation was a superior measure of the severity of the acute asthma episode

since it was more discriminating of the need for hospital admission and of relapse than was PEF, and since it had the practical advantage of not requiring conscious effort or cooperation on the part of the patient. In addition, the arterial oxygen saturation value is reflective of the end result of many of the pathologic changes which occur in acute asthma (e.g., airway obstruction at all levels of the bronchial tree), while the PEF value predominantly reflects only major airway obstruction (Geelhoed, Landau, & LeSouef, 1990).

Forced spirometry measures, while less helpful during acute asthma (Geelhoed, Landau, & LeSouef, 1990), are commonly used as the objective measures of dyspnea during pulmonary function testing, outside of the acute asthma episode (Wanger, 1992). Forced spirometry provides information about the flow and volume of air moving in and out of the lungs in a single, rapid inhalation and forced expiration (Wanger, 1992). These measures are indicators of mechanical properties of the lungs, such as compliance and elastic recoil, and of the diameter of the conducting airways. These measures are reduced in asthma when the disease is active and are inversely related to the degree of airways obstruction (Carr, Lehrer, & Hochron, 1995).

Consistent with the concepts of anxiety sensitivity and self-focused attention, individuals with greater awareness of their dyspnea have been shown to report more frequent asthma attacks and to perceive changes in their breathing more easily (Janson-Bjerklie et al., 1987). Awareness of somatic symptoms is beneficial in that it serves as an indicator of the need for action

(e.g., taking medication) on the part of the asthmatic patient (Carr, Lehrer, & Hochron, 1995). Responding with self-focused attention and panic-fear to initial symptoms of dyspnea, however, may lead to maladaptive anxiety and overuse of as-needed medication, factors which may exacerbate the severity and duration of the acute asthma episode (Carr, Lehrer, & Hochron, 1995).

Also consistent with anxiety sensitivity research is the finding that those who tend to panic during acute asthma episodes report greater fear of anxious symptomatology yet do not differ from non-panickers in the actual severity of breathlessness (Carr et al., 1994). Finally, although physiology and subjective reporting of dyspnea have been found to be independent of one another, emotional arousal can serve to increase changes in ventilation, producing a reciprocal relationship where changes in emotions lead to changes in breathing, and vice versa (Ley, 1994). Anxiety-produced changes in ventilation are significant for the purposes of this study in that they could directly affect subjective appraisals of dyspnea intensity.

Steiner et al. (1987) reported that the perception of asthma and of emotional arousal involve similar higher nervous functions. As with the perception of arousal, there are individuals who have difficulty recognizing and acknowledging changes in their asthmatic condition (Steiner et al., 1987). Referred to again as repressors, these individuals tend to deny symptomatology while responding with paradoxical physiological and emotional arousal. Repressors are of interest because of their low anxiety-

sensitive dispositions and because poor perception of the severity of asthma is a predictor of severe asthma; also, denial is one of several features characteristic of individuals who die of asthma (Barnes, 1994).

In the current study, attention will be focused on the relationship between anxiety sensitivity scores, self-reported dyspnea, and the level of physiological arousal, as measured by arterial oxygen saturation and forced spirometry values. It is anticipated that some members of the sample will rate their anxiety sensitivity as very low and that they will likewise rate their subjective experience of dyspnea as less severe. It is expected that such a pattern of response will be more common in males than in females.

In sum, the research to date has suggested that anxiety sensitivity is a distinct construct which taps an individual's fear of the symptoms of anxiety and their consequences. It is not believed that anxiety sensitivity is biologically based or dependent on a history of previous exposure to situations which induced panic. Instead, it is conceptualized as a dispositional variable which leads to internal hypervigilance and the tendency to catastrophize, misattribute, and over-report symptoms of anxiety. Anxiety disorders in general and self-focused behaviour in particular, have been found to be more common in females.

Anxiety sensitivity is assessed by measuring an individual's beliefs associated with the consequences of anxious symptomatology. The literature has given substantial attention to those who score above the average on the

ASI and CASI but has mostly neglected the study of those who score below the average. Anxiety research has indicated that these individuals may be repressing or poorly processing symptoms of arousal and further indicates that they are more likely to be males.

As with anxiety sensitivity, research has indicated that subjective reports of dyspnea are partially independent of actual physiological arousal. One goal of this study is to examine the relationship between subjective reports and physiological arousal; another is to determine if self-reported anxiety sensitivity is positively related to the subjective experience of dyspnea. If it is, it may be possible to lessen the intensity of the subjective experience of dyspnea by addressing the psychological factors which contribute to anxiety sensitivity. This study will also examine whether anxiety sensitivity is a superior predictor of dyspnea intensity than is trait anxiety, in order to determine if anxiety sensitivity makes an independent contribution to the understanding of childhood asthma.

Method

Participants

The sample consisted of 38 children (22 boys and 16 girls) who presented to the Emergency Room of the Alberta Children's Hospital for treatment of asthma attacks. The parents of 48 children were approached for participation in the study; 41 consented. Three of the children and their parents did not attend the second session and so did not complete their

participation in the study. Each of the resulting 38 subjects had been diagnosed with asthma at least 6 months prior to inclusion in the study. Children who had participated in a previous asthma study were excluded from the sample, as were those with coexisting medical conditions or 'pseudo-asthma', a primarily psychogenic condition with symptoms mimicking those of an asthma attack. The children were recruited between March of 1995 and July of 1996. Only 6- to 12-year-old children (M = 8.71; SD = 2.06) were eligible for the study. Mean duration of asthma was 5.45 years (SD = 2.28; range= 1 - 10); the mean age at diagnosis was 3.70 years (SD = 3.59). In the year prior to involvement in the study, asthma resulted in an average of 9.06 (SD = 9.20; range = 0 - 40) missed school days per subject and 2.11 (SD = 2.16; range = 0 - 12) Emergency Room visits.

Psychological Measures

Anxiety Sensitivity was measured with the Childhood Anxiety Sensitivity Index (CASI), an 18-item self-report scale, as presented in Appendix 1. The CASI is a modification of Peterson and Reiss' (as cited in Silverman, Fleisig, Rabian, & Peterson, 1991) Anxiety Sensitivity Index (ASI), and is designed to measure anxiety sensitivity by asking children to rate the aversiveness of symptoms of anxiety. Some of the ASI items were reworded so that they would be more understandable to children. Children are asked to respond to items such as, "It scares me when my heart beats fast", by marking either *none* (1), *some* (2), or *a lot* (3). The child's total anxiety sensitivity score is

calculated as the sum of the points endorsed for each question. The CASI's test-retest reliability has been measured at r = .76 and it's interitem reliability standardised alpha equals .87 (Silverman et al., 1991). The CASI was administered following pulmonary function testing at a follow-up session.

General anxiety was measured in an effort to distinguish the effects of anxiety sensitivity on asthma from those which may be caused by general anxiety. The Revised Children's Manifest Anxiety Scale (RCMAS; Reynolds & Richmond, 1978) is a reliable 37-item scale of anxiety for use with 6- to 19-year-old children. A total score is calculated, as are scores for a 9-item lie scale and three subscales - Physiological Symptoms, Worry/Oversensitivity, and Concentration. The RCMAS contains questions such as "I am afraid of a lot of things" and "I worry about what is going to happen". Subjects receive 1 point for each "yes" response and 0 points for each "no" response. The internal consistency (Cronbach's Alpha) of the RCMAS has been measured at above .80 for both boys and girls, children and adolescents (Hagborg, 1991; King et al., 1991). The RCMAS was also administered following pulmonary function testing at the follow-up session.

Subjective Ratings of Dyspnea:

Participants were asked to rate their subjective experiences of dyspnea both at the emergency room, following presentation for treatment of acute asthma and during a follow-up session when pulmonary function tests were conducted. Questioning was brief and was presented in the form of a Visual

Analogue Scale (VAS) so that verbal responses were not required. Subjects were asked to rate their subjective feeling of breathlessness by pointing to the appropriate point on a dimensional VAS anchored with "not at all breathless" at the low end and "as breathless as can be" at the high end. Self-ratings were calculated in the form of mm, rising from 0mm at the low end of the scale to a maximum possible score of 100mm at the high end. This measure was administered three times to each subject: twice in the emergency room during the acute asthma espisode and once following pulmonary function testing at a follow-up session. The first VAS administered in the emergency room was given just prior to the administration of the first ventolin mask and collection of the first set of physiological readings; the second, just prior to the first reassessment by the attending ER physician (usually following administration of the third ventolin mask). The VAS has the practical advantages of being simple to administer, of not requiring a verbal response from the child, and of avoiding the pitfalls of language involved in graphic rating scales (Gift, 1989).

Physiological Measures:

During the acute asthma episode, the physiological measure of interest was the subject's level of arterial oxygen saturation, since this has been demonstrated to be a superior and less confounded measure during the acute asthma episode than is PEF (Geelhoed et al., 1990). The measure of arterial oxygen saturation was obtained from periodic evaluations made by hospital staff.

A second set of physiological measures were taken by hospital staff during the follow-up appointment. Both percentage expected Forced Expiratory Volume in 1 Second (%FEV_1) and volume of air expired during the first second divided by total Forced Vital Capacity (%FEV_1/FVC) are measures of forced spirometry which are acquired through pulmonary function testing. FEV_1 is the maximum volume that can be expired from a full inspiration in 1 second. It is a measurement of both volume and mean flow in the first second (Miller, 1987a). FEV_1 is also reported as a ratio in relation to the Forced Vital Capacity (FVC), indicating the percentage of the FVC that is expired in 1 second (Miller, 1987a). Both FEV_1 and %FEV_1/FVC are reduced in asthma when the disease is active and are inversely related to the degree of airways obstruction (Carr, Lehrer, & Hochron, 1995).

Procedure

This study was conducted as a component of a larger study. Formal consent was obtained from the guardian(s) for participation in the entire study once their child was admitted into the Emergency Room and while they were waiting for physician examination and treatment. The purpose of the study was explained to the parents by the investigator who then asked them to read and sign the informed consent form, as presented in Appendix 2. Once the guardian(s) agreed to allow their child to participate in the study, the child was informed of the requirements of participation and asked for his or her verbal assent.

Once consent and assent were obtained and treatment started, the first VAS was administered orally to each subject. The oxygen saturation measure was taken at this time by the Emergency Room nurses as part of routine procedure. Values were recorded just before treatment was started and prior to reassessment by the attending physician. As part of the larger study, subjects were fitted with respibands and EMG and ECG electrodes, which remained in place for the duration of treatment. During application of the first ventolin mask, the guardian(s) were asked questions pertaining to the child's asthma and treatment history. The second VAS was administered prior to reassessment (usually following the application of the third ventolin mask).

Subjects were asked to return to hospital approximately two weeks following discharge from the emergency room for a follow-up pulmonary assessment session. Following pulmonary function testing conducted by hospital staff, subjects were asked to complete the CASI, the RCMAS, and the third VAS. All tests were administered orally in order to ensure that participants fully understood each question.

Data Analysis:

Multiple regression analyses were conducted to estimate the independent contribution of general anxiety and anxiety sensitivity to subjectively-rated dyspnea intensity. Analyses were conducted hierarchically with general anxiety entered first, followed by anxiety sensitivity on a subsequent step. The hierarchical approach is a conservative measure which

indicates whether a second predictor or set of predictors entered into a regression equation explains a significant proportion of the variance in the dependent variable, over and above that which is attributable to the original predictor. The predictors are the scores on the RCMAS (general anxiety) and the CASI (anxiety sensitivity). The outcome measure is subjectively-rated dyspnea intensity. Since anxiety sensitivity is the predictor of interest in the present study, it will be entered last to determine if it explains a significant proportion of the variance beyond that predicted by general anxiety.

In addition, Pearson product-moment correlations were conducted to determine relationships among variables. A median split was conducted on the CASI scores to form two groups: 'higher' and 'lower' scorers. A tertile split was also conducted in order to form two more distinct 'high' and 'low' groups of CASI scorers. The two groups obtained from the median split and the three groups from the tertile split were then compared for gender, using a chi-square test and for age, age at diagnosis, SaO_2 values, VAS values, RCMAS scores, FVC values, and FEV_1 values, using independent t-tests.

Finally, individual Subjective Difference Scores (SDS), as described by Heim, Blaser, and Waidelich (1972) were calculated for each subject in an effort to relate self-rated dyspnea (VAS-F) with each of the pulmonary function tests (%FEV_1 and %FEV_1/FVC). Subjects' 'ranks' on the self-rating (VAS-F) were subtracted from their 'ranks' on each of the pulmonary function tests (%FEV_1 and %FEV_1/FVC) to produce the SDS. The first-ranked subject in both the

pulmonary function tests and the self-ratings of dyspnea was the one whose score indicated the least airways resistance. For example, a score of 0mm on the VAS-F earned that subject a rank of 1; a score of 1mm earned a rank of 2, and so forth.

According to Heim, Blaser, and Waidelich (1972), a large positive SDS indicates that a subject's rank on the objective measure is higher (indicating more severe airways resistance) than the subject's rank in the self-rating of dyspnea. Therefore, subjects with large positive scores may be said to underrate their dyspnea and those with large negative scores may be said to overrate their dyspnea. In an effort to distinguish 'repressors' and to determine whether individuals with asthma tend to over- or underrate their experience of dyspnea, a mean SDS was calculated for the entire sample; those who score at least 1 standard deviation above the sample mean were considered to be possible repressors.

Results

In addition to the total sample, several other 'groups' were constructed for the purpose of statistical investigation and comparison. Subjects were separated into two age groups (6 to 9 and 10 to 12), two gender groups, and several response groups ('higher' and 'lower'; 'low', 'medium', and 'high') based on median and tertile splits of their CASI scores. These groups were established in order to examine the data from several different angles and to distinguish the response patterns of younger children from older children,

Table 1

Mean scores on the Childhood Anxiety Sensitivity Index (CASI)

Study/Age Group	N	M	SD
Present Study			
6 to 9[a]	25	30.92	6.22
10 to 12[a]	13	27.31	4.91
6 to 12[a]	38	29.68	6.00
Rabian et al. (1993)			
6 to 11[b]	35	26.38	N/A
12 to 18[b]	27	26.12	N/A
6 to 11[c]	10	30.70	N/A
12 to 18[c]	8	30.33	N/A
Silverman et al. (1991)			
11.4 to 15.8[a]	81	26.53	5.81
11.4 to 15.8[a]	77	25.51	5.77
8 to 15[d]	31	28.07	7.01
8 to 15[d]	18	28.56	7.70

Note. [a]Subjects were not assessed for anxiety disorders. [b]Subjects diagnosed with anxiety disorders were removed from this sample. [c]All subjects in this sample were diagnosed with anxiety disorders (diagnoses included: separation anxiety, overanxious, avoidant, simple phobia, social phobia, and generalized anxiety). [d]Clinical sample with heterogeneous diagnoses (e.g., enuresis, dysthymia, overanxious disorder).

males from females, and those higher in anxiety sensitivity from those with lower scores. The specific groups being examined will be described in each of the sections which follow.

Mean CASI Scores by Age and Gender

The mean CASI scores for this sample and for the two samples previously investigated with the CASI (Rabian et al., 1993; Silverman et al., 1991) are presented, by age group, in Table 1. The difference in anxiety sensitivity scores between the two age groups were examined by means of a t-test, the results of which were not significant, $t(36) = 1.82$, $p>.05$, two-tailed. The same analysis was also conducted on the mean CASI scores as distinguished by gender; again, the results were not significant, $t(36) = -1.75$, $p>.05$, two-tailed.

Intercorrelations Among Measures

Pearson product-moment correlations were computed for the two measures of anxiety (CASI and RCMAS) and each of the physiological (SaO_2-A, SaO_2-T and $\%FEV_1$, $\%FEV_1/FVC$) and subjective dyspnea (VAS-A, VAS-T, VAS-F) measurements. Correlations were also computed for CASI scores and all other variables and for each VAS and the physiological measure which was taken at the time when the VAS was administered. For example, VAS-A was administered when each subject was first admitted to the emergency room, prior to treatment. At that time, each subject's SaO_2-A value was measured by hospital staff. In accordance, VAS-T and SaO_2-T were both measured

Table 2

Correlations Among Measures

	RCMAS	SaO_2-A	SaO_2-T	VAS-A	VAS-T	VAS-F	$\%FEV_1$	$\%FEV_1$/FVC
CASI	.48**	-.18	.34	-.003	.09	.03	-.03	.23
RCMAS		-.08	.27	.20	.16	.09	-.06	.28
VAS-A	.20	-.24						
VAS-T	.16	-.37*	-.30					
VAS-F	.09						-.18	-.21

Note. CASI = Childhood Anxiety Sensitivity Index, RCMAS = Revised Children's Manifest Anxiety Scale, SaO_2 = arterial oxygen saturation, VAS = Visual Analogue Scale (subjective measure of dyspnea), $\%FEV_1$ = Percentage Expected Forced Expiratory Volume in 1 second, $\%FEV_1/FVC$ = volume of air expired during the first second divided by total Forced Vital Capacity (lower scores indicate more impaired pulmonary function). Letters following dashes indicate time at which measure was taken: A = following admission to emergency, T = following treatment in emergency, F = follow-up.
* $p < .02$. ** $p < .002$.

following application of the third ventolin mask in the emergency room and VAS-F, %FEV_1, and %FEV_1/FVC were recorded at follow-up.

Table 2 displays the intercorrelations obtained among measures. The table reveals that RCMAS and CASI scores were moderately correlated, $r = .48$, $p < .002$, indicating a shared variance of 23% for the two measures. This percentage is in keeping with previous research which indicates that measures of anxiety sensitivity and general anxiety tend to correlate moderately (meta-analysis conducted by Reiss, 1991). The CASI scores did not correlate with any other measure, including age, age at diagnosis, gender, and number of visits to the emergency room in the past year.

In addition, there was a negative linear relationship between SaO_2-A and a later subjective measure of dyspnea, VAS-T, $r = -.37$, $p < .02$ but notbetween SaO_2-A and VAS-A or any of the other time-paired dyspnea and physiological measures. This appears to indicate that the subjective measure of dyspnea administered following treatment in the emergency room (VAS-T) decreased in relation to the first measure of oxygen saturation (SaO_2-A), taken upon each subject's arrival at emergency; there was no relationship, however, between SaO_2-A and the subjective measure of dyspnea (VAS-A) administered at the time SaO_2-A was recorded.

Correlations between measures for the younger group of subjects (ages 6-9) and for the older group (ages 10-12) were calculated separately, as were correlations between measures for males and for females. These analyses

Table 3

Correlations Among Measures for Age Groups 6-9 and 10-12

	CASI	RCMAS	VAS-A	VAS-T	VAS-F
RCMAS					
(6-9 yrs)	.49*	N/A	.26	.08	.21
(10-12 yrs)	.33	N/A	.07	.38	-.30
SaO_2-A					
(6-9 yrs)	-.37	-.14	-.19	N/A	N/A
(10-12 yrs)	.44	.15	-.38	N/A	N/A
SaO_2-T					
(6-9 yrs)	.27	.35	N/A	-.37	N/A
(10-12 yrs)	.51	.07	N/A	.01	N/A
VAS-A					
(6-9 yrs)	.03	.26	N/A	N/A	N/A
(10-12 yrs)	.07	-.08	N/A	N/A	N/A
VAS-T					
(6-9 yrs)	.20	.08	N/A	N/A	N/A
(10-12 yrs)	.01	.38	N/A	N/A	N/A

Table 3, continued

Correlations Among Measures for Age Groups 6-9 and 10-12

	CASI	RCMAS	VAS-A	VAS-T	VAS-F
VAS-F					
(6-9 yrs)	-.08	.21	N/A	N/A	N/A
(10-12 yrs)	.20	-.30	N/A	N/A	N/A
$\%FEV_1$					
(6-9 yrs)	-.26	-.25	N/A	N/A	-.17
(10-12 yrs)	.18	.02	N/A	N/A	-.19
$\%FEV_1$/ FVC					
(6-9 yrs)	-.22	-.18	N/A	N/A	-.02
(10-12 yrs)	.50	.28	N/A	N/A	-.07

Note. CASI = Childhood Anxiety Sensitivity Index, RCMAS = Revised Children's Manifest Anxiety Scale, SaO_2 = arterial oxygen saturation, VAS = Visual Analogue Scale (subjective measure of dyspnea), $\%FEV_1$ = Percentage Expected Forced Expiratory Volume in 1 second, $\%FEV_1/FVC$ = volume of air expired during the first second divided by total Forced Vital Capacity (lower scores indicate more impaired pulmonary function). Letters following dashes indicate time at which measure was taken: A = following admission to emergency, T = following treatment in emergency, F = follow-up.
* $p < .02$.

Table 4

Correlations Among Measures for Male and Female Subjects

	CASI	RCMAS	VAS-A	VAS-T	VAS-F
RCMAS					
(males)	.45*	N/A	.24	.05	.03
(females)	.55**	N/A	.17	.26	.20
SaO_2-A					
(males)	-.05	-.07	-.21	N/A	N/A
(females)	-.47	-.08	-.25	N/A	N/A
SaO_2-T					
(males)	.30	.15	N/A	-.20	N/A
(females)	.36	.50	N/A	.12	N/A
VAS-A					
(males)	.02	.24	N/A	N/A	N/A
(females)	.08	.17	N/A	N/A	N/A
VAS-T					
(males)	.03	.05	N/A	N/A	N/A
(females)	.18	.26	N/A	N/A	N/A

Table 4, continued

Correlations Among Measures for Male and Female Subjects

	CASI	RCMAS	VAS-A	VAS-T	VAS-F
VAS-F					
(males)	.06	.03	N/A	N/A	N/A
(females)	.10	.20	N/A	N/A	N/A
$\%FEV_1$					
(males)	-.08	-.06	N/A	N/A	-.26
(females)	-.27	-.01	N/A	N/A	-.30
$\%FEV_1$/ FVC					
(males)	.09	-.14	N/A	N/A	-.39
(females)	-.03	.04	N/A	N/A	-.47

Note. CASI = Childhood Anxiety Sensitivity Index, RCMAS = Revised Children's Manifest Anxiety Scale, SaO_2 = arterial oxygen saturation, VAS = Visual Analogue Scale (subjective measure of dyspnea), $\%FEV_1$ = Percentage Expected Forced Expiratory Volume in 1 second, $\%FEV_1/FVC$ = volume of air expired during the first second divided by total Forced Vital Capacity (lower scores indicate more impaired pulmonary function). Letters following dashes indicate time at which measure was taken: A = following admission to emergency, T = following treatment in emergency, F = follow-up.
* $p < .04$. ** $p < .03$.

indicated that the correlation between the CASI and the RCMAS was present only in the younger age group (6-9). The correlational information for the younger (6-9) and older age groups is presented in Table 3; table 4 details the correlations for males and for females.

Means and Standard Deviations

Table 5 presents the means and standard deviations for each of the measures, across three groups: the total sample, subjects whose score on the CASI was at or below the sample median of 28.50, and subjects whose CASI score was above 28.50. For the purposes of comparison, subjects who scored above the sample median on the CASI were considered 'higher' in anxiety-sensitivity and those who scored at or below the sample median were considered 'lower' in anxiety-sensitivity. Although the higher CASI group scored consistently higher across all measures, with the exception of SaO_2-A, the higher and lower CASI group means differed significantly on only one measure, the RCMAS. The higher CASI group's RCMAS scores ($\underline{M}$ = 50.94, $\underline{SD}$ = 8.99) were significantly higher than were the lower CASI group's scores ($\underline{M}$ = 43.80, $\underline{SD}$ = 7.29), $\underline{t}(36) = -2.32$, $\underline{p} < .03$.

A tertile split was also conducted on the sample in order to compare individuals whose scores comprised the top and bottom third of CASI scores for this group. The results of the tertile split paralleled those of the median split; the upper and lower groups differed significantly only on their RCMAS scores ($\underline{t}(25) = -3.14$, $\underline{p} < .005$), with individuals in the lower tertile scoring

Table 5

Means and Standard Deviations of Variables by Median Split Level of Anxiety Sensitivity (AS)

Variable		t(36)	Higher AS (n = 19)	Lower AS (n = 19)	Total (n = 38)
CASI	M		34.53	24.84	29.68
	SD		4.36	2.32	5.97
RCMAS	M	-2.32	50.32_a	44.05_a	47.03
	SD		9.16	7.40	8.74
SaO_2-A	M	1.40	91.95	93.53	92.46
	SD		3.34	3.61	3.62
SaO_2-T	M	- .29	93.06	92.67	92.65
	SD		3.47	4.07	3.80
VAS-A	M	- .87	7.60	6.21	6.77
	SD		4.02	5.53	4.74
VAS-T	M	- .65	4.11	3.33	3.55
	SD		3.11	4.08	3.54
VAS-F	M	- .67	3.69	2.69	3.10
	SD		4.90	3.91	4.35

Table 5, continued

Means and Standard Deviations of Variables by Median Split Level of Anxiety Sensitivity (AS)

Variable		t(36)	Higher AS (n = 19)	Lower AS (n = 19)	Total (n = 38)
$\%FEV_1$	M	.41	91.12	92.82	91.89
	SD		9.16	14.44	11.77
FEV_1/FVC	M	- .77	84.18	82.11	82.83
	SD		7.38	8.41	7.94

Note. Means with the subscript $_a$ differ significantly at $p < .03$. AS = Anxiety Sensitivity. Level of AS was determined by a median split (median = total score of 28.50 on CASI). CASI = Childhood Anxiety Sensitivity Index, RCMAS = Revised Children's Manifest Anxiety Scale, SaO_2 = arterial oxygen saturation, VAS = Visual Analogue Scale (subjective measure of dyspnea), $\%FEV_1$ = Percentage Expected Forced Expiratory Volume in 1 second, $\%FEV_1/FVC$ = volume of air expired during the first second divided by total Forced Vital Capacity (lower scores indicate more impaired pulmonary function). Letters following dashes indicate time at which measure was taken: (A) = following admission to emergency, (T) = following treatment in emergency, (F) = follow-up.

lower on the RCMAS than individuals in the upper tertile. The results of the tertile split are presented in Table 6.

Mean scores were also calculated for the measures of SaO_2-A and $\%FEV_1/FVC$ for purposes of comparison with asthmatic and normal samples. The sample mean for SaO_2-A was 92.46 which is indeed indicative of acute asthma (Geelhoed, Landau, & LeSouef, 1990). The sample mean for $\%FEV_1/FVC$ of 82.83 was at the low end of the 'normal' range specified for adults under the age of 39, indicating unimpaired breathing (Miller, 1987b), as would be expected given that the $\%FEV_1/FVC$ measure was taken at follow-up, outside of the acute asthma episode. Further, $\%FEV_1/FVC$ values should be higher for young children since flow rates decline with age (Miller, 1987a), possibly indicating mild impaired $\%FEV_1/FVC$ in the sample.

Hierarchical Regression Analyses

Hierarchical multiple regression analyses were conducted to determine the independent contributions of the CASI and the RCMAS to the explained variance on the VAS-F, the $\%FEV_1$, and the $\%FEV_1/FVC$. The other physiological measures and subjective dyspnea ratings were not included in the analyses because the CASI and RCMAS were administered at follow-up and so could not be used to predict measures taken previously in the emergency room.

The results of the regression analyses are presented in Table 7. The data from the VAS-F served as the dependent measure in the first analysis. Neither

Table 6

Means and Standard Deviations of Variables by Tertile Split Level of Anxiety Sensitivity (AS)

Variable		t(25)	High AS (n = 14)	Low AS (n = 13)
CASI	M		35.93	23.62
	SD		4.25	1.66
RCMAS	M	-3.14	52.50_a	42.46_a
	SD		9.58	6.66
SaO_2-A	M	.36	92.36	92.85
	SD		3.25	3.83
SaO_2-T	M	-1.98	93.92	91.10
	SD		2.78	3.90
VAS-A	M	- .50	7.59	6.62
	SD		4.10	5.83
VAS-T	M	- .17	3.73	3.53
	SD		2.34	3.53
VAS-F	M	- .90	4.63	2.90
	SD		5.54	4.01

Table 6, continued

Means and Standard Deviations of Variables by Tertile Split Level of Anxiety Sensitivity (AS)

Variable		t(25)	High AS (n = 14)	Low AS (n = 13)
$\%FEV_1$	M	- .26	91.43	90.15
	SD		9.91	15.58
FEV_1/FVC	M	-1.46	85.54	81.00
	SD		6.42	9.17

Note. Means with the subscript $_a$ differ significantly at $p < .005$. AS = Anxiety Sensitivity. Level of AS was determined by a tertile split (tertile score cutoffs = 25 for 'low' group, 31 for 'medium' group, and 48 for 'high' group. CASI = Childhood Anxiety Sensitivity Index, RCMAS = Revised Children's Manifest Anxiety Scale, SaO_2 = arterial oxygen saturation, VAS = Visual Analogue Scale (subjective measure of dyspnea), $\%FEV_1$ = Percentage Expected Forced Expiratory Volume in 1 second, $\%FEV_1/FVC$ = volume of air expired during the first second divided by total Forced Vital Capacity (lower scores indicate more impaired pulmonary function). Letters following dashes indicate time at which measure was taken: (A) = following admission to emergency, (T) = following treatment in emergency, (F) = follow-up.

the CASI nor the RCMAS scores explained a significant amount of the variance on the VAS-F. Similar calculations were performed in two other regression analyses where the %FEV_1 and %FEV_1/FVC served as the dependent measures. The CASI and RCMAS scores were also unable to explain a significant amount of variance on these measures. These analyses indicated the absence of a linear relationship between CASI and VAS-F scores; the distribution of these scores, as examined in a scatterplot, also did not support the hypothesis of a curvilinear relationship between CASI and VAS-F scores.

Subjective Difference Scores

Three sets of Subjective Difference Scores (SDS) were calculated for each member of the sample. The first set used SaO_2-A as the objective measure of airways resistance and VAS-A as the self-rated measure of dyspnea. The second and third sets of SDS were calculated using both follow-up measures of airway resistance, %FEV_1 and %FEV_1/FVC, and the subjective ratings from the VAS-F.

A score of 0 indicates perfect agreement between subjective and objective ratings; a high negative score indicates a tendency to overrate the subjective experience of dyspnea and a high positive score indicates a tendency to underrate dyspnea. The sample mean for the first set of SDS (calculated with SaO_2-A and VAS-A) was -1.12 with a standard deviation of 7.11. For the second set and third sets, calculated with VAS-F and with %FEV_1 and %FEV_1/FVC as the objective measures, the sample means were 5.88 (SD =

Table 7

Summary of Hierarchical Regression Analysis for Variables (CASI and RCMAS) Predicting Subjective Dyspnea (VAS-F) and Pulmonary Function Results (%FEV_1 and %FEV_1/FVC) at Follow-up

	VAS-F			%FEV_1			%FEV_1/FVC		
Variable	B	SE B	β	B	SE B	β	B	SE B	β
Step 1									
RCMAS	.04	.10	.09	.01	.01	.13	-.07	.28	-.05
Step 2									
CASI	-.01	.15	-.02	-.03	.02	-.32	.01	.40	.01

Note. R^2 = .006 for Step 1 VAS-F; R^2 = .077 for Step 1 %FEV_1; R^2 = .003 for Step 1 %FEV_1/FVC.

7.42) and 4.66 (SD = 8.14), respectively.

The SDS for 12% of the subjects were at least 1 standard deviation above the mean when SaO_2-A was the objective measure. When $\%FEV_1$ was the objective measure, 9% scored at least 1 standard deviation above the mean; 20% when $\%FEV_1/FVC$ was used. Therefore, depending on which objective measure was used, between 9-20% of the subjects in this study tended to underrate their dyspnea in comparison with the rest of the sample.

However, an even greater number of subjects had SDS which were at least 1 standard deviation below the mean in each of the three comparisons (24% with SaO_2-A, 14% with $\%FEV_1$, and 17% with $\%FEV_1/FVC$). This indicates that between 14-24% of the sample tended to overrate their degree of dyspnea. In addition, while no subject's SDS was greater than 1 standard deviation above the mean, regardless of the objective measure used, one subject (representing 3% of the entire sample) scored greater than 2 standard deviations below the mean when each of $\%FEV_1$ and $\%FEV_1/FVC$ were used in the calculations.

Summary of Results

Overall, this study's hypotheses were not borne out by its results. A significant relationship was not found to exist between anxiety sensitivity and subjective ratings of dyspnea. Nor was there any relationship between anxiety sensitivity and objective measures of airway obstruction. A significant positive relationship did exist between CASI and RCMAS scores, in keeping with

previous studies comparing measures of anxiety sensitivity and general anxiety (meta-analysis conducted by Reiss, 1991). In addition, it was discovered that objective and subjective measures of airway obstruction did not correlate, as was predicted by this study and supported by previous studies (e.g., Rubenfeld & Pain, 1976; Steele & Shaver, 1992).

Neither the CASI nor the RCMAS was able to predict a significant amount of variance on either subjective or objective measures of airways obstruction. The calculation of Subjective Difference Scores indicated that asthmatics were as likely to overrate their degree of dyspnea as to underrate it; no gender differences were found between over- and underraters. Dividing the sample by age, gender, and CASI scores into several comparison groups had no significant effect on any of the analyses.

Discussion

Both the ASI and the CASI were designed to measure the belief that the consequences of anxiety symptomatology are negative and both have been used with clinical and non-clinical samples. Much of the literature has focused on the effect of anxiety sensitivity on specific anxiety disorders such as agoraphobia (e.g., McNally & Lorenz, 1987) and panic disorder (e.g., Asmundson & Norton, 1993; Taylor et al., 1992) and on distinguishing between clinical and non-clinical subjects (e.g., Donnell & McNally, 1989; Rabian et al., 1993).

Previous studies have used the CASI to examine anxiety sensitivity in

children between the ages of 6 and 18 (Rabian et al., 1993; Silverman et al., 1991). The current study examined a smaller range of ages and included 38 children between the ages of 6 and 12. The subjects in this study were examined as a group and were also divided by age, gender, and CASI scores into several comparison groups. Males (N = 22) outnumbered females (N = 16) in this sample and more of the subjects were aged 6 to 9 (N = 25) than 10 to 12 (N=13).

Only two studies using the CASI currently exist in the literature (Rabian et al., 1993; Silverman et al., 1991). The first of these studies established the psychometric properties of the instrument (Silverman et al., 1991); the second studied its ability to distinguish anxious from nonanxious children (Rabian et al., 1993). While normative scores have been established for the adult Anxiety Sensitivity Index (ASI), the only comparative scores for the CASI come from the Silverman et al. (1991) and Rabian et al. (1993) studies. These scores and the mean scores from the present study were presented in Table 1.

Direct comparison of scores across studies is difficult due to differences in the composition of each sample. The sample in the present study is purely medical; selection was based only on age, presentation at the emergency room for treatment of an acute asthma episode, and a pre-existing diagnosis of asthma of at least 6 months duration. The children in the non-clinical samples of the other two studies were recruited from non-medical populations; requests for participation in those studies were sent to military families (Rabian et al.,

1993) and to a public school system (Silverman et al., 1991).

None of the children in this study was examined for or diagnosed with anxiety disorders; in the Rabian et al. (1993) study, children were screened for anxiety disorders and their results were examined separately from the children who did not meet criteria for anxiety diagnoses (Rabian et al., 1993). In the Silverman et al. (1991) study's non-clinical sample, the public school children who were recruited were not screened for anxiety disorders; however, comparison between the current sample and this group is constrained by the discrepancy between the participants' ages as the children in the non-clinical sample of the Silverman et al. (range: 11.4 - 15.8, $\underline{M}$ = 13.3; 1991) study are much older than those in the current study (range 6 - 12, $\underline{M}$ = 8.71). Also, neither Silverman et al. (1991) nor Rabian et al. (1993) examined a group of children as young as those in the current study's 6-9-year-old age group.

Given these differences in sample composition and the unknown implications of factors such as age, gender, and medical diagnoses on anxiety sensitivity, it would be imprudent to try to gain meaning from comparing the scores of subjects in this sample with those from subjects in previous CASI samples. That having been noted, the scores in this study may add to the childhood anxiety sensitivity literature the observation that, in a relatively young, medical sample, where children have not been assessed for anxiety disorders, overall CASI scores appear to be higher than those found in other non-clinical samples.

While normative data and 'cut-off' scores above which subjects may be considered to be anxiety-sensitive have been determined for clinical and non-clinical adult populations (Peterson & Reiss, as cited in Donnell & McNally, 1989), similar standards have not been reported for children. In addition, there exists a sample-specific approach for classifying adults as anxiety-sensitive which involves the same calculations used to determine the ASI's normative data. Subjects whose scores are greater than one standard deviation above the sample mean are classified as 'high' anxiety-sensitive and those whose scores are greater than one standard deviation below the sample mean are classified as 'low' (Asmundson & Norton, 1993; Carr et al., 1994; Donnell & McNally, 1990). The use of a similar approach has not been documented in the limited CASI literature (Silverman et al., 1991; Rabian et al., 1993).

Since the sample-specific approach designed for the ASI has not been prescribed for use with the CASI and since its use would eliminate the majority of subjects (28 of 38) from study in an already small sample, a different approach to classifying children's CASI scores as 'higher' or 'lower' was adopted. For the purposes of comparison, and in an effort to include a larger sample for study, median and tertile splits were used to form several groups: 'lower' and 'higher' CASI scorers and 'low', 'medium', and 'high' scorers, respectively. While median and tertile splits have not been employed in the anxiety sensitivity literature to date, the median split has been used with a measure of anxiety (RCMAS) in order to identify 'repressors' in a sample of

83 asthmatic children (Fritz, Spirito, & Yeung, 1994).

The median split was employed in this study in an effort to use CASI scores to create two comparison groups. When the 'higher' and 'lower' CASI scorers were compared across several dimensions, however, they differed only in the manner in which they responded to the RCMAS. As might be expected, subjects in the 'higher' CASI group also tended to have higher scores on the RCMAS. Analyses indicated that the two groups did not differ in gender, age, age at diagnosis, or on physiological measures (SaO_2-A, SaO_2-T, %FEV1, and $\%FEV_1/FVC$) or subjective dyspnea ratings (VAS-A, VAS-T, VAS-F). The results from the tertile split were consistent with these findings. Consistent gender and age differences have yet to be established in the CASI literature (Rabian et al., 1993; Silverman et al., 1991); subjective and objective measures of airway obstruction have yet to be examined.

While the ASI has been used previously with an asthmatic population (Carr et al., 1994; Carr, Lehrer, & Hochron, 1995), the CASI has not. Using the CASI with a medical population was conceived as an exploration into the significance of anxiety sensitivity in a sample of children who were not diagnosed with anxiety disorders and yet whose pre-existing medical condition could be expected to cause periodic episodes of anxiety.

Carr et al. (1994) justified the use of the ASI with an asthmatic population by drawing comparisons between the symptoms of asthma (e.g., dyspnea, dizziness, accelerated heart rate) with those common to panic

disorder. In addition, high rates of anxiety disorders (34%; Yellowlees, Alpers, Bowden, Bryant, & Ruffin, as cited in Carr et al., 1994) and a high incidence of comorbid panic disorder (24%; Kinsman et al., 1973) have been discovered in adult asthmatic populations.

The primary goal of this study was to use the CASI with children experiencing acute asthma episodes in order to determine if their level of anxiety sensitivity was related to their objective (oxygen saturation and pulmonary function measures) and subjective physiological distress (self-rated dyspnea). Pearson product-moment correlations between the CASI and both the objective and subject measures of physiological distress failed to establish a relationship between anxiety sensitivity and the symptoms of asthma. Pearson product-moment correlations were also conducted separately on data from male and from female subjects and on data from younger (6 to 9) and from older (10 to 12) subjects. Dividing the sample by age and by gender, however, provided no additional information; the CASI was uncorrelated with all measures, save the RCMAS, in each analysis.

A relationship between anxiety sensitivity and the symptoms of asthma has yet to be established in either the adult or child anxiety sensitivity literature. It has been attempted once, by Carr et al. (1994), who found that asthma alone was not associated with irrational beliefs about the consequences of anxiety (anxiety sensitivity) among adults. Their results were established, however, after identifying individuals with panic disorder in the sample and

calculating the significance of the presence of asthma on the ASI scores of nonpanickers alone. Since panic attacks are associated with catastrophic misinterpretations of unusual sensations (Koszycki, Cox, & Bradwejn, 1993), individuals who experience panic attacks are also likely to be anxiety sensitive. Indeed, before removing the panickers from the analyses, there was a significant relationship between ASI scores and asthma. Since panickers were neither identified nor excluded from the current study, it was reasonable to expect that a relationship would exist between CASI scores and the symptoms of asthma.

There are at least two possible reasons for the lack of relationship between anxiety sensitivity and asthma symptoms in the children in this study. First, there may have been little comorbidity between a diagnosis of asthma and high levels of anxiety in this sample. Indeed, Janson, Bjornsson, Hetta, and Boman (1994) reported that there is no association between either a diagnosis of asthma or the objective symptoms of asthma and anxiety. In addition, the RCMAS also did not correlate with any of the symptoms of asthma, as would be expected if a significant number of the subjects in the study were clinically anxious. Furthermore, studies which have demonstrated comorbidity between anxiety symptoms and asthma (e.g., Kinsman et al., 1973; Yellowlees et al., as cited in Carr et al., 1994) have been conducted on adult samples and may not be applicable to a group of subjects aged 6 to 12. If few of the subjects in this study had comorbid high levels of anxiety, the results

could be reasonably expected to parallel those found by Carr et al. (1994) once panickers were removed from their sample (i.e., asthma alone was not correlated with ASI scores), as they did.

Second, although there is similarity between symptoms of asthma and those of panic disorder (e.g., dyspnea, accelerated heart rate), the symptoms of asthma are medically explicable. Anxiety sensitivity occurs when individuals fear the consequences of the symptoms of anxiety. Since each of the subjects in this study had been previously diagnosed with asthma and was at least 6 years of age, it is reasonable to expect that they and their parents understood the significance of the symptoms of dyspnea when they occurred. Moreover, children who experienced frequent asthma attacks would have had the opportunity to become familiar with the symptoms of asthma and their consequences. Even a visit to the emergency room may not necessarily have been anxiety-provoking for the children in this sample as they reported having attended the emergency room for treatment of asthma attacks a mean of twice each in the past year.

Several studies have used multiple regression analyses involving the CASI and a measure of anxiety in efforts to establish anxiety sensitivity as a unique construct. These studies have been successful in demonstrating that anxiety sensitivity measures (both the ASI and the CASI) were able to explain a significant amount of variance on subjective measures of fear (e.g., The Fear Survey Schedule for Children - Revised; Ollendick, as cited in Silverman et al.,

1991) that anxiety measures could not (McNally, 1989; Rapee & Medoro, 1994; Silverman et al., 1991). In this study, a similar effort was made to identify the unique contribution of anxiety sensitivity to the experience of dyspnea by examining the amount of variance explained by both the CASI and the RCMAS on a subjective measure of dyspnea (VAS-F).

As in previous studies, the comparisons were made between three subjective measures (CASI scores, RCMAS scores, VAS-F scores); in addition, two other analyses examined the independent contributions of the CASI and RCMAS scores to the variance on objective measures of airway resistance [the percentage expected Forced Expiratory Volume in 1 Second (%FEV_1) and the percentage volume of air expired during the first second divided by total Forced Vital Capacity (%FEV_1/FVC)].

Neither the CASI nor the RCMAS was able to predict a significant amount of variance on either the subjective or objective measures of airway resistance. That these measures were unable to explain significant variance on objective measures of dyspnea is in accordance with previous research which has failed to find correlations between objective and subjective measures of dyspnea. Previous dyspnea research has failed to find consistent physiological differences between subjects reporting high and low levels of dyspnea, probably since the experience and reporting of dyspnea is dependent on motivational and affective factors (Fritz, Klein, & Overholser, 1990; Steele & Shaver, 1992). In accordance with these findings, subjective measures of

dyspnea were also uncorrelated with objective measures in this study.

It was anticipated, however, that the CASI, as a subjective measure, would be able to predict a significant amount of variance on a subjective measure of dyspnea. Although measures of anxiety sensitivity have been used to explain variance on subjective measures in previous studies, they have not been used to predict dyspnea specifically. Since a relationship between anxiety sensitivity and asthma was not established in this study, and since anxiety sensitivity was unable to explain a significant amount of variance on the subjective measure of dyspnea, it appears that anxiety sensitivity did not contribute to subjects' experiences of or responses to dyspnea.

The regression analyses also indicated the lack of a linear relationship between CASI scores and subjective dyspnea scores. In an effort to identify 'repressors' in the sample, it was hypothesized that a curvilinear relationship between those two variables would arise. 'Repressors' have been identified as individuals who report low anxiety on rating scales yet have paradoxically high scores on objective measures of dyspnea (Steiner et al., 1987). The distribution of the data, however, did not support the hypothesis of a curvilinear relationship between CASI scores and the objective measures of airway obstruction.

'Repressors' may also be identified by examining the relationship between subjective and objective measures of dyspnea. Rubenfeld and Pain (1976) reported that many asthmatics tend to either over- or underestimate the

degree to which their airways are occluded. This is in keeping with the established lack of correspondence between subjective and objective measures of dyspnea. In addition to examining relationships between measures of dyspnea using correlational and multiple regression analyses, three sets of Subjective Difference Scores (SDS) were developed for each subject in an effort to identify subjects who underrated their experience of dyspnea (possible repressors). The SDS were calculated by subtracting each subject's rank on a self-rating of dyspnea from their rank on each of three objective measures of dyspnea (SaO_2-A, %FEV1, and $\%FEV_1/FVC$) in the manner described by Heim, Blaser, and Waidelich (1972).

An SDS of zero indicates perfect congruence between subjective and objective ratings. Large positive SDS indicate that dyspnea is being underrated and may be characteristic of individuals who repress or minimize their experience of dyspnea. These individuals are of great interest because they often do not seek the medical help that they require (Heim, Blaser, & Waidelich, 1972) and because denial is a characteristic feature of individuals who die of asthma (Barnes, 1994). In addition, poor perception of the severity of asthma is a predictor of severe asthma (Barnes, 1994).

The Heim, Blaser, and Waidelich (1987) analyses, conducted on 22 asthmatic adults, revealed that asthmatics tended to have high positive SDS ($\underline{M}$ = 21), indicating that they tended to underrate their degree of dyspnea. The mean SDS in the present study ranged from a small negative score

(-1.12) to two moderate positive scores (5.88 and 4.66), depending on which objective measure was used in the analysis (SaO_2-A, %FEV1, and $\%FEV_1/FVC$, respectively), indicating that in 2 out of 3 sets of SDS, the subjects tended to slightly underrate the severity of their airway resistance.

However, it is important to note that - as in the Heim, Blaser, and Waidelich (1972) study - there is considerable variability within the sample; that is, the standard deviation in each of the three sets of SDS is quite high (7.11, 7.42, and 8.15, respectively). Moreover, the SDS indicate that the subjects in this study were as likely to significantly overrate their degree of dyspnea (14-24% had SDS at least 1 SD less than the mean; 3% has SDS at least 2 SD less than the mean) as to underrate it (9-20% had SDS at least 1 SD greater than the mean).

There were no consistent similarities among underraters across the 3 sets of SDS. Underraters were as likely to be male as female and were as likely to be in the 'higher' CASI group as in the 'lower' CASI group. It is of interest to note that, of the subjects who tended to underrate their dyspnea, only 1 did so in all three sets of SDS calculations; that subject also scored 3 standard deviations above the mean on the CASI, by far the highest CASI score in the sample. Since a repressive response style tends to be consistent across measures and time (Steiner et al., 1987), it appears that only one subject in this study could be classified as a repressor.

Limitations of the Study

The nature of the sample of interest to this study made it an interesting exploration of a population not previously examined with the CASI but also served to limit the composition and ultimate size of the sample. Since subjects were recruited as they presented for treatment, the sample could not be chosen at random and was essentially one of convenience. Had the sample been larger, it may have been possible to examine CASI scores in the manner prescribed for ASI scores (i.e., 'high' scorers are classified as those scoring one standard deviation above the mean), without excluding the majority of the sample.

Although samples of comparative and smaller sizes have been used previously in the anxiety sensitivity literature and asthma literature (e.g., 48 in the McNally & Lorenz study, 1987; 12 in the Steiner et al. study, 1987, respectively) both of the studies previously conducted using the CASI made use of larger samples (85 in the Silverman et al., 1991 study; 152 in the Rabian et al study), as did the Carr et al. (135 subjects; 1994) study, which was the only previous investigation to examine the relationship between anxiety sensitivity and asthma.

The nature of the sample would also make establishing a suitable control group difficult for use with this study. The subjects were not simply asthmatics, they were asthmatics who were first examined in the context of an acute asthma episode. The complexity of their clinical presentations was

further compounded by the fact that subjects were presenting at the emergency room for treatment. In addition, the objective measures used in this study would not have been either appropriate or relevant for use with a non-asthmatic population.

Nonetheless, use of a non-asthmatic comparison group may have been able to provide additional information concerning the relationship between anxiety sensitivity and asthma in one important way. Since the CASI was not administered during the acute asthma episode in the emergency room, comparisons between mean CASI scores could have been made between an age-matched non-asthmatic group and those in this study. This would have been particularly informative since no cut-off scores have been established for the CASI.

Finally, since anxiety sensitivity has been studied very little in children, and had yet to be examined in a medical population, it would have been beneficial if this study had taken the opportunity to add to the literature on the CASI's psychometric properties. Had the CASI been administered in the initial session with each subject, test-retest reliabilities could have been calculated for each age and gender group. In addition, the CASI could have been used in multiple regression analyses to predict initial visual analogue scale and oxygen saturation data collected during the initial session.

Future Directions

Since there are only two published studies which have made use of the

CASI with any population and only one study which has examined anxiety sensitivity in an asthmatic population, the most obvious and pressing consideration for the future is that more research be conducted in both areas. Although the CASI is a variation of an instrument which has been established as psychometrically sound, its own psychometric properties have been tested only on one sample of 72 children (Silverman et al., 1991) and its ability to distinguish clinically anxious from non-anxious subjects, on a single sample of 201 children (Rabian et al., 1993).

Since anxious self-preoccupation, which may be induced by an anxiety sensitive disposition (e.g., thoughts of death every time an asthma attack occurs) can exacerbate breathlessness (Ley, 1994), the continued study of anxiety sensitivity in asthmatic populations is essential. Given that the results of both of the known studies of anxiety sensitivity conducted on asthmatic populations (Carr et al., 1994 and this study) indicated that there was no relationship between asthma alone and anxiety sensitivity, future investigations in this area might choose to define their samples selectively. For example, studies investigating the degree of anxiety sensitivity in asthmatics who have comorbid diagnoses of anxiety disorders may provide more useful information on the impact of anxiety sensitivity on asthma symptoms.

The study of anxiety sensitivity may eventually add to the repertoire of techniques asthmatic individuals can use to minimize and manage their symptoms. There exist a number of successful techniques for the prevention

and management of asthma, including medication compliance, symptom discrimination, relaxation, biofeedback (Creer, 1991), and behavioural self-management programs (Colland, 1993). In addition to these, providing patients with education concerning the causes and consequences of dyspnea symptoms should be a matter of course and may help to prevent anxiety sensitivity from arising from a lack of knowledge. Also, Clark (as cited in Carr et al., 1994) suggested the use of cognitive-restructuring as a measure to help prevent asthmatic individuals from developing anxiety disorders. The preventive effects of these techniques could provide an interesting subject for future investigation.

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Appendix 1

Childhood Anxiety Sensitivity Index (CASI)

CASI

Name: _____ Age: ____ Date: _____

Directions: A number of statements which boys and girls use to describe themselves are given below. Read each statement carefully and put an X in the box in front of the words that describe you. There are no right or wrong answers. Remember, find the words that best describe you.

1. I don't want other people to know when I feel afraid. ___None ___Some ___Alot
2. When I cannot keep my mind on my schoolwork I worry that I might be going crazy. ___None ___Some ___Alot
3. It scares me when I feel "shaky". ___None ___Some ___Alot
4. It scares me when I feel like I am going to faint. ___None ___Some ___Alot
5. It is important for me to stay in control of my feelings. ___None ___Some ___Alot
6. I scares me when my heart beats fast. ___None ___Some ___Alot
7. It embarrasses me when my stomach growls (makes noise). ___None ___Some ___Alot

8. It scares me when I feel like
   I am going to throw up. ___None ___Some ___Alot
9. When I notice that my heart is
   beating fast, I worry that there
   might be something wrong with me. ___None ___Some ___Alot
10. It scares me when I have
    trouble getting my breath. ___None ___Some ___Alot
11. When my stomach hurts, I worry
    that I might be really sick. ___None ___Some ___Alot
12. It scares me when I can't keep
    my mind on my schoolwork. ___None ___Some ___Alot
13. Other kids can tell when I
    feel shaky. ___None ___Some ___Alot
14. Unusual feelings in my body scare me. ___None ___Some ___Alot
15. When I am afraid, I worry
    that I might be crazy. ___None ___Some ___Alot
16. It scares me when I feel nervous. ___None ___Some ___Alot
17. I don't like to let my
    feelings show. ___None ___Some ___Alot
18. Funny feelings in my body scare me. ___None ___Some ___Alot

Note. From "Childhood Anxiety Sensitivity Index," by W. K. Silverman, W. Fleisig, B. Rabian, and R. A. Peterson, 1991, Journal of Clinical Child Psychology, 20.

Appendix 2

Consent Form

Research Project: Respiratory parameters, dyspnea, and panic-fear in pediatric asthma.

Investigators: Dr. Ian Mitchell, Dr. Donald Bakal, Linda Rose, M. Sc., Chris Grant, B. A.

Funding Agency: Alberta Mental Health

Contact Numbers: Alberta Children's Hospital, 229-7211; Dr. Bakal, 220-4971; Linda Rose, 220-6352; Chris Grant, 283-0926

This consent form, a copy of which has been given to you, is only part of the process of informed consent. It should give you the basic idea of what the research project is about and what you child's participation will involve. If you would like more detail about something mentioned here, or information not included here, you should feel free to ask. Please take the time to read this carefully and to understand any accompanying information.

The purpose of this research is to develop a better understanding of how children breathe during an asthma attack and the impact of breathing patterns on such factors as the amount of pain and distress experienced. We are also interested in determining whether there is any relationship between distress, knowledge of asthma and asthma self management, as well as between distress, asthma-related hospital visits and school absences and child behaviour.

Your child's participation in this research will occur in two stages. First, while your child is receiving treatment for an asthma attack she or he will be asked about common asthma symptoms, and you will be asked some questions about your child's asthma history. The questions require approximately 15 minutes to complete. Second, you and your child will be asked to return to the hospital the following week. Your child's breathing will be assessed by a respiratory technician and he or she will complete questionnaires on asthma symptoms, asthma knowledge, and asthma self-management. You will also fill in a questionnaire about your child's behaviour. You will spend between 1 and 2 hours at the hospital on this second visit.

We will also require access to you child's Health Record in order to obtain information about his or her asthma history. All information collected during the course of your child's participation in this study will be kept strictly

confidential and no individual other than the investigators will have access to the information.

Your child's participation in this study will help us learn more about how breathing affects the experience of pain and distress during an asthma attack and identify any particular factors which influence distress. As you know, asthma attacks can be a very frightening experience; a better understanding of the factors related to distress may help us improve treatment care for your child during future attacks and for other children with asthma.

A written summary of the research findings will be made available to all participants in this study.

Your signature on this form indicates that you have understood to your satisfaction the information regarding your participation in the research project and agree that your child may participate as a subject. In no way does this waive the legal rights of you or your child, nor release the investigators, sponsors, or involved institutions from their legal or professional responsibilities. Your child is free to withdraw from the study at any time without jeopardizing his or her health care. Your child's continued participation should be as informed as the initial consent, so you should feel free to ask for clarification or new information throughout your child's participation. If you have further questions concerning matters related to this research, please contact the investigators listed above.

If you have any questions concerning your child's rights as a possible participant in this research, please contact the Office of Medical Bioethics, Faculty of Medicine, The University of Calgary at 220-7990.

_____	_____
Parent	Date
_____	_____
Investigator (For Dr. Mitchell)	Date
_____	_____
Witness	Date