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Hyperventilation symptoms and their relationship to panic

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The University of Arizona, 1991
HYPERVENTILATION SYMPTOMS AND THEIR RELATIONSHIP TO PANIC

by

Cheri Joan Shapiro

A Dissertation Submitted to the Faculty of the DEPARTMENT OF PSYCHOLOGY In Partial Fulfillment of the Requirements For the Degree of DOCTOR OF PHILOSOPHY In the Graduate College THE UNIVERSITY OF ARIZONA

1991
As members of the Final Examination Committee, we certify that we have read the dissertation prepared by Cheri Joan Shapiro entitled *Hyperventilation Symptoms and their Relationship to Panic* and recommend that it be accepted as fulfilling the dissertation requirement for the Degree of Doctor of Philosophy.

Final approval and acceptance of this dissertation is contingent upon the candidate's submission of the final copy of the dissertation to the Graduate College.

I hereby certify that I have read this dissertation prepared under my direction and recommend that it be accepted as fulfilling the dissertation requirement.

Dissertation Director
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# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>LIST OF ILLUSTRATIONS</td>
<td>6</td>
</tr>
<tr>
<td>LIST OF TABLES</td>
<td>7</td>
</tr>
<tr>
<td>ABSTRACT</td>
<td>8</td>
</tr>
<tr>
<td>CHAPTER</td>
<td></td>
</tr>
<tr>
<td>1. HYPERVENTILATION SYMPTOMS AND THEIR RELATIONSHIP TO PANIC</td>
<td>10</td>
</tr>
<tr>
<td>chlorHPerventilation and the Hyperventilation Syndrome</td>
<td>11</td>
</tr>
<tr>
<td>Symptom Stability</td>
<td>14</td>
</tr>
<tr>
<td>Measurement Issues in Assessing Hyperventilation Symptoms</td>
<td>15</td>
</tr>
<tr>
<td>Hyperventilation and Anxiety</td>
<td>16</td>
</tr>
<tr>
<td>Hyperventilation and Panic</td>
<td>17</td>
</tr>
<tr>
<td>Role of Repression in Symptom Presentation</td>
<td>24</td>
</tr>
<tr>
<td>HVS and the Cognitive Model of Panic</td>
<td>27</td>
</tr>
<tr>
<td>Role of Anxiety Sensitivity</td>
<td>32</td>
</tr>
<tr>
<td>Summary and Focus of Present Research</td>
<td>34</td>
</tr>
<tr>
<td>2. STUDY 1</td>
<td>42</td>
</tr>
<tr>
<td>Method</td>
<td>42</td>
</tr>
<tr>
<td>Subjects</td>
<td>42</td>
</tr>
<tr>
<td>Materials</td>
<td>44</td>
</tr>
<tr>
<td>Design and Procedure</td>
<td>48</td>
</tr>
<tr>
<td>3. RESULTS</td>
<td>51</td>
</tr>
<tr>
<td>Stability of Hyperventilation Symptoms</td>
<td>51</td>
</tr>
<tr>
<td>Relationship of Panic to Hyperventilation Symptoms</td>
<td>51</td>
</tr>
<tr>
<td>Cognitive and Somatic Symptoms</td>
<td>55</td>
</tr>
<tr>
<td>Repression and Type of Symptoms</td>
<td>55</td>
</tr>
<tr>
<td>Endorsed</td>
<td>56</td>
</tr>
<tr>
<td>Anxiety Sensitivity and Panic</td>
<td>57</td>
</tr>
<tr>
<td>Prediction of Panic</td>
<td>57</td>
</tr>
<tr>
<td>4. DISCUSSION</td>
<td>60</td>
</tr>
<tr>
<td>5. STUDY 2</td>
<td>Page</td>
</tr>
<tr>
<td>------------</td>
<td>------</td>
</tr>
<tr>
<td>Subjects</td>
<td>64</td>
</tr>
<tr>
<td>Materials</td>
<td>64</td>
</tr>
<tr>
<td>Procedure</td>
<td>65</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>6. RESULTS</th>
<th>Page</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>7. DISCUSSION</th>
<th>Page</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>8. GENERAL DISCUSSION</th>
<th>Page</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>REFERENCES</th>
<th>Page</th>
</tr>
</thead>
</table>
LIST OF ILLUSTRATIONS

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Path diagram of hypothesized relationships between physiological processes, personality variables, and environmental events in the development of HVS and PD; measures used are in parentheses.</td>
<td>38</td>
</tr>
<tr>
<td>2.</td>
<td>Timeline for screening and data collection of fall and spring subsamples.</td>
<td>50</td>
</tr>
<tr>
<td>3.</td>
<td>Stability of hyperventilation symptoms over 3 months for Spring sample (n = 23).</td>
<td>53</td>
</tr>
<tr>
<td>4.</td>
<td>Distribution of HVQ scores for large sample (n = 446).</td>
<td>54</td>
</tr>
<tr>
<td>5.</td>
<td>Mean CO₂ level as a function of frequency of hyperventilation symptoms.</td>
<td>69</td>
</tr>
</tbody>
</table>
LIST OF TABLES

Table                                                                 Page
1. Comparison of symptoms of hyperventilation syndrome and panic disorder ... 18
2. Demographics for screening samples by sex and frequency of hyperventilation symptoms .... 43
3. Simple correlations among variables in the multiple regression equation .... 58
4. Promax rotated factor pattern and final communality estimates for hyperventilation symptoms on the HVQ .... 71
ABSTRACT

Symptom overlap between the Hyperventilation Syndrome (HVS) and Panic Disorder (PD) has been noted by many investigators; however, the reasons for this association are unclear. By following 50 college subjects (36 female and 14 male) who displayed extremely high rates of hyperventilation symptoms over four-week to nine-month intervals, symptom stability patterns as well as the relationship to panic attacks were examined. Rates of overlap between hyperventilation symptoms and panic attacks was 66% in the current sample. Furthermore, 4 of 50 (8%) of panic-free subjects developed panic attacks during the course of the investigation. Although the frequency of hyperventilation symptoms decreased over time for most subjects, many individuals continued to be symptomatic over time. One potential mechanism of association between hyperventilation and panic (i.e., focus on either somatic or somatic plus cognitive symptoms of hyperventilation) was not supported, and several personality variables (repression and anxiety sensitivity) were not found to influence the relationship between hyperventilation and panic. Finally, measurement issues related to a hyperventilation symptom questionnaire were addressed. The two-week test-retest
reliability was .79. Evaluation of the validity of theHVQ by assessing end-tidal CO₂ levels of 30 subjects resulted in no significant differences between high and low scorers. Implications of the present findings are discussed in light of current theoretical models of the relationship between HVS and PD.
CHAPTER 1

HYPERVENTILATION SYMPTOMS AND THEIR RELATIONSHIP TO PANIC

Sufferers of the Hyperventilation Syndrome (HVS) can experience discomfort in nearly any organ or system and often seek the advice of numerous specialists in search of a cure (Lum, 1981). HVS symptoms can be psychological as well as physical and can create chronic problems for the estimated 10 to 15% of the general population thought to be affected by this disorder (Fried, 1987). Rates in clinical populations are even higher, ranging from 3.5 to 28% (Huey & Sechrest, 1983). Despite the seriousness and potential magnitude of the problem, the syndrome is infrequently recognized and often misdiagnosed as organic disease (Fried, 1990; Huey & Sechrest, 1983; "Hyperventilation syndromes," 1982; Missri & Alexander, 1978). The relative neglect of the syndrome may be due in part to the general lack of discussion of the phenomenon in medical texts or the psychological literature (Grossman & DeSwart, 1984). Misdiagnosis is likely a function of the wide range of symptoms individuals can present, which may mimic those of other disorders (Lum, 1981). Distinguishing HVS from certain anxiety disorders in particular is extremely
difficult, as the symptoms of HVS overlap to a great degree with the current diagnostic criteria for both panic disorder and generalized anxiety disorder (e.g., Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised, DSM-III-R). Yet, HVS is not listed as a differential diagnosis for either illness (APA, 1987). Furthermore, although the role of hyperventilation as an etiological factor in anxiety disorders has received recent attention, HVS is not discussed or thought of as an etiologic factor for any anxiety disorder. This lack of integration will be addressed here.

To provide an introduction to HVS, the mechanics of hyperventilation as well as the diagnosis of HVS will be reviewed. As HVS most closely resembles Panic Disorder (PD), a discussion of problems in discriminating between PD and HVS will follow, as well as examination of variables likely to be salient in determining the relationship between these disorders.

Hyperventilation and the Hyperventilation Syndrome

Hyperventilation involves ventilation in excess of metabolic needs. Excessive amounts of CO₂ are "blown off," producing respiratory alkalosis (Lum, 1981). The better known form of hyperventilation, the acute hyperventilation attack, is relatively easy to diagnose because of the
dramatic symptoms it produces, such as tetany (severe muscle spasms) or unconsciousness (Huey & Sechrest, 1983). The term hyperventilation syndrome (HVS) refers to the complex of symptoms produced by a second, more common, chronic, and subtle form of hyperventilation (Fried, 1987; Lum, 1981; Lewis, 1964). In these less exaggerated cases of hyperventilation, many somatic and psychological symptoms are produced, including dyspnea (difficulty breathing), light-headedness, breathlessness, palpitations, paresthesias (numbness and tingling in the extremities), nausea, tremors, chest pain, and anxiety (Huey & Sechrest, 1983; Missri & Alexander, 1978). Current theorists believe that this range of symptoms arises from rapid changes in arterial CO₂ produced by an unstable breathing pattern as opposed to being simply caused by lowered absolute levels of CO₂ (Lum, 1987). Magarian (1982) notes that the syndrome commonly consists of recurrent symptoms, while in other cases a persisting state of hyperventilation occurs in which symptoms are usually present or easily triggered by already lowered PaCO₂ (arterial carbon dioxide).

Despite being straightforward, some controversy exists over the diagnosis of HVS. Elements of the diagnosis include assessing symptoms of hyperventilation, results of a hyperventilation provocation test (HVPT), and various signs related to the physiological effects of overbreathing. Some
investigators support the notion of diagnosis by symptoms (e.g., Fried, 1987), while others appear to focus solely on signs of hyperventilation, including measurement of end-tidal (end expiration) CO₂ (a valid approximation to arterial CO₂ levels) or a failure of CO₂ to return to baseline levels quickly after a period of voluntary hyperventilation (not seen in normals) (Grossman & DeSwart, 1984; Van Doorn, Folgering & Colla, 1982). The most common method of diagnosis appears to be assessment of symptoms of hyperventilation and a HVPT. The HVPT is most often relied upon to confirm the presence of the syndrome and requires that an individual voluntarily hyperventilate (without being informed as to expected results) in the presence of a therapist or doctor. It is considered positive if sufficient presenting complaints (symptoms) are reproduced. (What constitutes sufficient reproduction of symptoms is usually unspecified.) Despite the widespread use of the HVPT, Fried (1987; 1990) strongly cautions against its use, as he believes the HVPT may dangerously reduce oxygen delivery to organs and tissues, especially the heart. Possible laboratory evaluations used when the diagnosis is made in a medical or research setting include electrocardiogram if chest pain is present as well as blood gas analysis and analysis of urine bicarbonate levels, both of which can
detect lowered CO₂ levels and consequences such as increased blood pH (Smith, 1985).

**Symptom Stability**

Given that symptom reports are an important component in the diagnosis of HVS, surprisingly little is known about how hyperventilation symptoms vary over time. Symptom stability is likely to reflect individual differences, as in some persons stress may trigger a symptomatic episode, while in others physiological adaptation to a chronically low level of arterial CO₂ may allow symptom production with only minimal provocation, such as a deep breath (Hill, 1979; Magarian, 1982). Lum (1987) states that increased variability of CO₂ over time can trigger symptomatic episodes. This increased variability is likely an individual differences phenomenon. How symptoms vary over time is vital information for research on hyperventilatory phenomena as reported symptoms characteristic of HVS have been used to identify clinical cases of HVS or subjects for research (e.g., Hill, 1979). Furthermore, the frequency and severity of hyperventilation symptoms have been used as outcome measures in HVS treatment studies (e.g., Van Doorn et al., 1982). If symptoms vary greatly over time, results involving symptom changes could be misleading. Although overbreathing in HVS is said to be chronic, whether symptoms of hyperventilation will remain at the same level of
intensity over time is also unknown. Natural variation in symptoms may provide information for treatment because reasons for such variations could be explored.

**Measurement Issues in Assessing Hyperventilation Symptoms**

As symptom experience is a critical factor in identifying HVS, attempts have been made to validate self-report measures of hyperventilation symptoms using physiological criteria. Using a questionnaire assessing frequency and severity of hyperventilation symptoms (here called the Hyperventilation Symptom Questionnaire, or HVQ), it has been documented that those with many symptoms (termed likely hyperventilators) react differently in response to a period of voluntary hyperventilation than those with few symptoms (i.e., unlikely hyperventilators) (Huey & West, 1983). Likely hyperventilators demonstrated greater mean intensity of symptoms than did the unlikely hyperventilators after overbreathing room air but not after overbreathing CO₂-enriched air. This finding suggests that hyperventilation symptoms are in fact due to lowered CO₂, as adding CO₂ to inspired air during the HVPT inhibited symptom production. Likely hyperventilators also evidenced higher minute volume and rate than unlikely hyperventilators during normal breathing (Huey & West, 1983). Although these results provide initial information on the validity of the hyper-
ventilation symptom questionnaire, additional evidence is needed. Specifically, examination of end-tidal CO2 levels would provide valuable information, as lowered PCO2 is indicative of hyperventilation and should be found among individuals reporting higher frequencies of hyperventilation symptoms (Ley, 1988).

**Hyperventilation and Anxiety**

Hyperventilation can be normal or deviant, depending upon the circumstances. Several authors have noted that an increase in respiration occurs as part of the normal response to stress (Ames, 1955; Huey & Sechrest, 1983; Smith, 1985). However, hyperventilation has been and continues to be associated with psychopathological states, especially anxiety and panic (Garssen, 1980; Hibbert, 1984; Huey & Sechrest, 1983; Ley, 1985; Missri & Alexander, 1978). In fact, Lum (1987) notes the dramatic increase of interest during this decade in the role of hyperventilation in anxiety-based disorders. Although a relationship does seem to exist between anxiety and hyperventilation, the direction of causality is a matter of debate. A number of investigators contend that anxiety causes hyperventilation (e.g., Missri & Alexander, 1978); others support the notion that anxiety is produced by the symptoms of hyperventilation (e.g., Lum, 1981). Ley (1985) suggests that anxiety and hyperventilation form a closed system in which the
components of the system become indistinguishable once the system has been set into motion. As the relationship of hyperventilation to anxiety in general remains a matter of debate, the relationship between hyperventilation and certain specific anxiety disorders has also become the focus of recent investigations.

**Hyperventilation and Panic**

Striking similarities exist between the symptoms of panic attacks and those of the hyperventilation syndrome. Panic attacks consist of unexpected, discrete periods of intense fear or discomfort accompanied by associated somatic symptoms (APA, 1987). The somatic sensations are varied, but results of epidemiologic studies suggest that the most common symptoms include palpitations, sweating, trembling or shaking, hot or cold flashes, and fear of dying (Von Korff, Eaton, & Keyl, 1985). Other symptoms include dyspnea, dizziness, choking, nausea, depersonalization, paresthesias, and chest pain (APA, 1987). When panic attacks reach a certain frequency and intensity, panic disorder is diagnosed. The parallels between panic and hyperventilation have been noted by many writers (Bass & Gardner, 1985; Clark, Salkovskis, & Chalkley, 1985; Cowley & Roy-Byrne, 1987; Hoes, Colla, Van Doorn, Folgering, & deSwart, 1987; Huey & Sechrest, 1983; Ley, 1985; Thyer, Papsdorf, & Wright, 1984). Table 1 lists symptoms of HVS and those of panic.
Table 1. Comparison of Symptoms of Hyperventilation Syndrome and Panic Disorder.

<table>
<thead>
<tr>
<th>Hyperventilation Syndrome</th>
<th>Panic Disorder</th>
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<tbody>
<tr>
<td>Dyspnea</td>
<td>Dyspnea</td>
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<tr>
<td>Dizziness, faintness</td>
<td>Dizziness, faintness</td>
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<tr>
<td>Numbness, tingling (paresthesias)</td>
<td>Paresthesias</td>
</tr>
<tr>
<td>Tremors, twitching</td>
<td>Trembling, shaking</td>
</tr>
<tr>
<td>Nausea</td>
<td>Nausea, abd. distress</td>
</tr>
<tr>
<td>Chest pain, discomfort</td>
<td>Chest pain, discomfort</td>
</tr>
<tr>
<td>Headache</td>
<td>Sweating</td>
</tr>
<tr>
<td>Blurred vision</td>
<td>Choking</td>
</tr>
<tr>
<td>Palpitations</td>
<td>Flushes, chills</td>
</tr>
<tr>
<td>Depersonalization, derealization</td>
<td>Depersonalization</td>
</tr>
<tr>
<td>Dry mouth, lump in throat</td>
<td>Fear of dying, going</td>
</tr>
<tr>
<td>weakness, exhaustion apprehension, nervousness</td>
<td>crazy, doing something uncontrolled</td>
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(Sources: APA, 1987; Gorman & Papp, 1990; Huey & Sechrest, 1983)
disorder. The symptoms listed are nearly identical and highlight the overlap between these disorders. Furthermore, in addition to the overlap in symptoms, both panic and hyperventilation are characterized by a predominance of female sufferers. The sex ratio for panic disorder, based on a review of large-scale epidemiologic studies (i.e., the Epidemiologic Catchment Area Study) is approximately 2:1 (Shapiro, 1989). Huey and Sechrest (1983) found a predominance of females among HVS patient populations (ranging from 2:1 to 7:1 in clinical samples), while other investigators (e.g., Lum, 1975) report equal distribution of males and females in a large clinical sample. Yet when these estimates are combined, the majority of HVS sufferers appear to be female. However, further investigation of sex differences in HVS is clearly warranted, as the current estimates of sex ratios come from clinical populations as opposed to surveys of the general population.

Further support for the overlap between HVS and PD was found in an investigation by Hoes et al. (1987). A total of 274 patients referred to several clinics were given an examination of pulmonary function. Individuals were diagnosed as having HVS if they met three of the following five criteria: high resting ventilation, low end-tidal pCO₂, reversed ventilatory response to an increase of pCO₂ in inspired air, absence of a step-change in end-tidal pCO₂
or a failure of end-tidal pCO₂ to return to baseline in 3 minutes after a one-minute HVPT, and last, blood gas analysis showing an excess of base. Of the total sample, 170 were diagnosed with HVS. Using a symptom questionnaire from which DSM-III diagnoses of PD could be obtained, 64 of the total sample were diagnosed with PD. Of the 170 patients with HVS, 35% (59) were diagnosed with PD as opposed to only 5% (5) of the 104 patients without hyperventilation. The fact that panic disorder is relatively rare in the absence of hyperventilation suggests that misinterpretation of hyperventilation episodes could be a cause of panic. Yet this is a tentative finding, as the data are correlational and do not rule out panic as a cause of hyperventilation problems.

In a thorough review, Cowley and Roy-Byrne (1987) detailed similarities between HVS and panic disorder, including historical origins of the diagnostic categories, phenomenology, physiology, and overlap in treatment techniques, which all lend support to their hypothesis that the disorders are causally related. Evidence for the various possible causal relationships was provided (i.e., that hyperventilation causes panic, panic causes hyperventilation, and finally, that both panic disorder and HVS have a common etiology). The authors favor the third hypothesis and propose that most patients with either disorder share a
hypersensitivity of a central alarm system mediating the general response to danger. Activation of this system produces both panic and hyperventilation, which can in some cases reinforce one another to establish a chronic condition. They state that the diagnosis is determined by which symptoms are prominent in a given person and by the physician or patient's emphasis on somatic or psychological symptoms.

Cowley and Roy-Byrne's (1987) suggestion that a focus on somatic and/or psychological symptoms affects differential diagnosis is important and has not been empirically addressed. If an individual emphasizes somatic complaints characteristic of the effects of hyperventilation, a diagnosis of HVS may be more likely. Individuals with this symptom pattern may also be more likely to seek medical attention for their symptoms. Indeed, in their review, Huey and Sechrest (1983) found prevalence rates ranging from 3.5% to 28% in patient groups with cardiac, gastrointestinal, neurological, or respiratory symptoms. The fact that the majority of these estimates come from medical patient populations cannot be overlooked. On the other hand, if psychological symptoms (e.g., subjective reports of fear and anxiety) are emphasized, a diagnosis of panic disorder may be more probable. Individuals with a predominantly cognitive symptom constellation may also be
more likely to seek help from mental health professionals. Which diagnosis an individual receives is a crucial issue for treatment, as interventions for HVS are largely behavioral (e.g., breathing retraining), and those for panic disorder are often pharmacological (e.g., tricyclic antidepressants such as imipramine). Such distinctions between cognitive ("psychological") and somatic symptoms of anxiety have been noted previously, often in terms of exploring treatment options for the different types of anxiety (Linden, Paulhus, & Dobson, 1986; Schwartz, Davidson, & Goleman, 1978). Which avenue of expression is dominant differs for each individual and is likely to be a multiply determined event with biological propensities, learning history, and characteristic modes of affective expression and interpretation as salient factors.

In fact, the importance of a distinction between cognitive/affective and somatic symptoms has received empirical support in the differentiation of panic disorder and HVS. Nelles and Barlow (1988) reviewed the literature on childhood HVS and noted that children appear to experience panic attacks without the cognitive/affective component (i.e., the subjective experience of fears of dying, going crazy, or losing control), which becomes labelled in the medical literature as HVS. A similar process is suggested to occur with adults, whereby presentation of somatic
symptoms of hyperventilation becomes labelled as HVS and not panic disorder; the reverse process occurs for those who present with a focus on cognitive symptoms. The first step in elucidation of such a process involves documenting the existence of separate symptom patterns for individuals with hyperventilation-related symptoms and examining variables that affect which symptoms are more likely to be presented. Identification of separate symptom patterns in individuals with panic has already begun.

Support for the importance of the distinction between cognitive/affective and somatic symptoms of anxiety is provided by Beitman et al. (1987), who noted the occurrence of "non-fearful" panic disorder (NFPD) in a sample of cardiology patients. Of the 14 symptoms listed for panic attacks in DSM-III-R, 12 are somatic while two ("fear of dying" and "fear of going crazy or doing something uncontrolled") are cognitive/affective. Those diagnosed as NFPD (12 of 38 patients) reported that during their last major attack they did not experience intense fear nor the two cognitive/affective symptoms of a panic attack, but they did report at least four of the somatic symptoms, which is consistent with the diagnostic requirements for panic disorder. This finding is limited by the methodology employed (i.e., asking for ratings of the last major panic attack as opposed to the most typical panic attack, which could have resulted
in reports of unique attacks), but it does serve to highlight the fact that individuals can and do experience panic attacks with somatic discomfort but without subjective anxiety. This finding also lends credence to the hypothesized distinction between panic and HVS; i.e., between primarily cognitive/affective and primarily somatic symptoms. Attacks such as these (i.e., without the cognitive/affective component) may be more likely to be labelled as HVS in contexts in which panic disorder have not been identified. Similar symptom distinctions (somatic vs. cognitive) have not yet been made in the adult HVS literature.

**Role of Repression in Symptom Presentation**

Individuals differ in how anxiety is experienced and expressed. These differences play a role in the hypothesized relationship between PD and HVS. Perhaps HVS and PD are one disorder, with differences in symptom presentation being mediated by other variables such as repression. This argument builds upon the ideas set forth by Cowley and Roy-Byrne (1987) by positing an individual differences variable that could lead to differences in symptom focus and presentation (which could, in turn, lead to different diagnoses).

Repression-sensitization refers to avoidance or approach tendencies in coping with anxiety (Bell & Byrne, 1978). Repressors tend to avoid or ignore threatening
information; sensitizers characteristically exaggerate and obsess about threats (Linden, Paulhus, & Dobson, 1986). People with repressive coping styles typically deny having elevated levels of anxiety, but they often respond nonverbally as if they were highly anxious (Weinberger, Schwartz, & Davidson, 1979). In fact, repressors' preoccupation with avoiding awareness of anxiety may interfere with effective coping (Weinberger, 1990). These individuals may be likely to report only somatic manifestations of anxiety but not cognitive/affective symptoms of feeling tense or anxious. This conjecture received some support from Weinberger, Schwartz, and Davidson (1979), who found that although repressors and low anxious subjects did not differ in reports of somatic anxiety on the Cognitive Somatic Anxiety Questionnaire (CSAQ) (Schwartz, Davidson, & Goleman, 1978), repressors' reports of cognitive anxiety were significantly lower than expected relative to the low anxious group or relative to their own levels of somatic anxiety. Although this finding may be interpreted as meaning that repressors have difficulty reporting both cognitive/affective and somatic symptoms of anxiety, it is important to note that the CSAQ requires people to respond by endorsing symptoms they generally feel when they are anxious. It is unclear if repressors would have difficulty reporting ongoing somatic experiences related to anxiety, as they may deny feeling
anxious but be able to report shortness of breath, palpitations, or other somatic experiences. Further support for distinctions between psychological and somatic symptoms was found by Linden, Paulhus, and Dobson (1986), who examined the response styles of self-deception (i.e., repression) and impression management (i.e., other-deception) in relation to the reporting of both psychological and somatic symptoms. The short Repression-Sensitization scale, the Self-Deception Questionnaire (SDQ), and the Other-Deception Questionnaire (ODQ) served as measures of response styles. Physical symptoms were measured by Pennebaker's Inventory of Limbic Languidness (checklist of 54 physical symptoms) and the 7 somatic items from the Cognitive and Somatic Anxiety Questionnaire (CSAQ). Psychological distress was assessed using the 7 items of the CSAQ related to cognitive anxiety, the Trait-Anxiety Questionnaire from the State-Trait Anxiety Inventory, and the Beck Depression Inventory. High levels of either response style, socially desirable and/or self-deceptive responding, predicted attenuated symptom reports. The response styles examined also predicted measures of psychological distress much more strongly than they predicted measures of somatic distress. Yet while response style accounted for much more variance in reports of psychological distress than in reports of physical symptoms, the impact on physical symptom reports was still substantial, with
response style accounting for 16-18% of the variance. Furthermore, a repressive/self-deceptive response style appears to reduce symptom reporting above and beyond any effects of conscious impression management. Those with a repressive style may therefore have great difficulty reporting psychological symptoms but less difficulty reporting somatic symptoms, even though reporting of somatic symptoms may be attenuated. In comparison, individuals who are highly anxious but not using a repressive response style or strategy (i.e., sensitizers) may readily admit to feeling tense and anxious in addition to experiencing various somatic symptoms. Therefore, the use of a repressive coping style may distinguish between those with only somatic complaints and those with somatic as well as cognitive/affective symptoms. This distinction is proposed as one of the crucial elements in the HVS/panic disorder diagnostic controversy and will be investigated in this regard.

HVS and the Cognitive Model of Panic

Individual differences in the expression of anxiety may help determine which disorder an individual is diagnosed as having (i.e., panic disorder or HVS). On a more fundamental level, however, individual differences in symptom interpretation may be a key factor in determining the nature of the relationship between hyperventilation and panic. In contrast to earlier suggestions by Cowley and Roy-Byrne
(1987), it has been proposed that the vulnerability of patients to the experience of acute panic may be the result of an enduring tendency to interpret bodily sensations in a catastrophic fashion rather than the result of biological pathology producing excessive autonomic discharge (Clark et al., 1988). Salkovskis and Clark (1990) suggest that the extent to which sensations produced by hyperventilation are interpreted in a negative and catastrophic way is a major determinant of. They term their view the cognitive model of panic. This model has also been forwarded by Ley (1987), who terms it the hyperventilation theory of PD and agoraphobia. Salkovskis and Clark cite supporting evidence for this model, including the findings of Clark, Salkovskis, and Chalkley (1985), who developed a respiratory treatment for panic attacks. Patients were taught to reattribute the sensations experienced during panic to hyperventilation, as opposed to catastrophic events that they feared (i.e., imminent heart attack). Patients were also taught to avoid hyperventilation to control panic attacks. Respiratory control treatment involved three steps: recognition of symptoms after a 2-minute HVPT, explanation of how hyperventilation induces panic, and training in slow breathing. Although post-treatment reductions in panic attack frequency as well as reduction in measures of avoidance, anxiety, depression, and distress initially appear promising,
conclusions of treatment efficacy are hindered by major methodological flaws. First, the majority of patients were taking psychotropic medication during the time of the study. Also, the treatment was given only to patients who recognized similarities between the effects of overbreathing and their panic attacks. Finally, the respiratory control treatment was given only for two weeks, after which other treatments were added (which differed for each patient). Given that no control group was used, and that later followup confounded the effects of various treatments, the conclusions from this study are limited.

The cognitive model of panic was further tested by Salkovskis, Jones, and Clark (1986). Nine consecutive referrals to the psychology department of a health service who suffered from panic attacks, accompanied by at least three body symptoms characteristic of hyperventilation, were chosen as subjects. A respiratory control treatment was provided, similar to that implemented by Clark et al. (1985). In addition, subjects were instructed in the use of paper bag rebreathing (to increase CO₂ and stop hyperventilation). As before, somatic symptoms were to be attributed to the effects of hyperventilation and not to the catastrophic events these subjects feared; e.g., heart disease, epilepsy, and going mad (Salkovskis et al., 1986). The methodology of this latter study was somewhat improved. The
majority of subjects were taking psychotropic medications. However, the treatment lasted four weeks, and for individuals with situational panic, only one type of treatment (exposure) occurred during the six-month follow-up period. Furthermore, end-tidal CO₂ levels were assessed both before and during treatment. Both panic attack frequency and ratings of general anxiety were significantly reduced, while pCO₂ levels increased during the course of treatment (p. 531). Once again, control groups were not used, thus leaving in doubt which parts of the treatment were most effective. Perhaps the effect of self-monitoring (i.e., keeping a daily diary of panic attacks) was enough to reduce frequency of the attacks. Combining the results of the Clark et al. (1985) and Salkovskis et al.'s (1986) studies, however, suggests that cognitive reframing of symptoms as the results of hyperventilation may be effective in reducing panic attack frequency.

More direct support of the cognitive model of panic, in which catastrophic interpretation of bodily symptoms is crucial for triggering acute panic, is provided by Salkovskis and Clark (1990), who had subjects undergo a brief period of overbreathing after affording them either positive or negative interpretations of the effects of this exercise. If affective responses to acute bodily sensations can be manipulated by interpretations of the valence of the symp-
toms, the cognitive model would receive support. Salkovskis and Clark (1990) used a classic attribution paradigm involving arousal (through hyperventilation) and provision of positive or negative interpretations of the arousal symptoms (attributing symptoms to indications of risk of fainting vs. signs of good adjustment and a higher state of consciousness). Hyperventilation symptoms and mood were both assessed after overbreathing for one minute (mood had also been assessed prior to the exercise). In the negative interpretation group, body sensations were significantly correlated with negative but not with positive affect, and in the positive interpretation group, body sensations were significantly correlated with positive but not negative affect. Therefore, it was demonstrated that the affect associated with a period of voluntary hyperventilation was influenced by the provision of positive or negative interpretations of the sensations experienced. Given that an individual can hold preconceived notions that determine the affective reaction to autonomic arousal, it is conceivable that catastrophic interpretation of hyperventilation symptoms could increase the negative valence of these symptoms and cause panic. Although these data are consistent with a cognitive model, the evidence is not inconsistent with the view that a subgroup of panic patients are likely to be suffering from HVS, in which the primary problem is a
centrally mediated defect in respiratory regulation (Salkovskis & Clark, 1990). The cognitive model well explains how a panic attack may begin once symptoms of hyperventilation are present, but the model does not address the origins of the hyperventilation episode. In other words, the cognitive model of panic is more a theory of maintenance of the disorder than a theory of etiology. In a similar vein, the model currently being proposed, by which symptom episodes become labelled as either HVS or PD, does not address the origins of the symptom episode but the process that occurs once symptoms are detected. In particular, the catastrophic interpretation of symptoms of hyperventilation is posited to occur but is hypothesized to be mediated by personality variables such as repression and anxiety sensitivity.

Role of Anxiety Sensitivity

The tendency to interpret bodily sensations in a catastrophic way has been investigated specifically in the area of anxiety disorders and has been termed anxiety sensitivity. Originally conceptualized as a component of an expectancy model of fear, anxiety sensitivity is viewed as distinct from trait anxiety and refers to the degree to which a person is afraid of becoming anxious, as opposed to the general tendency to be anxious in a variety of situations (Reiss & McNally, 1985). Just as people vary in their frequency of anxiety symptoms, they are assumed to vary in
their fear of such symptoms (McNally & Lorenz, 1987). Anxiety sensitivity denotes the tendency to respond anxiously to arousal symptoms (Donnell & McNally, 1989) and may be a predisposing factor in the development of anxiety disorders (Reiss, 1987). Agoraphobics score high on anxiety sensitivity measures, as do other anxiety disorder patients (Barlow, 1988; McNally & Lorenz, 1987). Anxiety sensitivity has been shown to predict enhanced response to a trial of voluntary hyperventilation (Donnell & McNally, 1989). A history of panic attacks alone may produce enhanced responding to an agent intended to produce panic such as hyperventilation, but panic history is associated with enhanced responsivity to hyperventilation only in subjects with high anxiety sensitivity (AS). Low AS subjects with a history of panic were no more responsive to a trial of voluntary hyperventilation than low anxiety sensitivity subjects who never had a panic attack (Donnell & McNally, 1989). These findings have implications for the diagnosis of HVS. Perhaps only individuals with high levels of anxiety sensitivity reach the level of response to the HVPT in which their presenting symptoms are accurately reproduced. Could high levels of anxiety sensitivity therefore be a crucial part of the HVS phenomenon? Others may experience the symptoms during a HVPT but in a much milder form that could preclude the diagnosis. Given the potential
importance of anxiety sensitivity to the HVPT and to HVS diagnosis, the relationship of anxiety sensitivity to hyperventilation symptoms deserves greater attention than it has received in the literature. Furthermore, Lum (1975) states that hyperventilation may become chronic because of the concern about and fear of the symptoms it induces. Assessing anxiety sensitivity in those with many hyperventilation symptoms thus becomes one possible test of this hypothesis.

The constructs of anxiety sensitivity and repression may have opposite effects on the presence of panic. Whereas individuals high in anxiety sensitivity would respond anxiously to arousal symptoms, thus increasing the likelihood of panic, individuals using a repressive coping style would be unlikely to acknowledge the impact of such symptoms, which could decrease the possibility of panic developing. These personality traits would also be unlikely to be found simultaneously.

Summary and Focus of Present Research

The primary aim of the current investigation was to examine issues related to the stability of hyperventilation symptoms over time and the relationship of these symptoms to panic attacks. Questions regarding long-term symptom stability were addressed using a hyperventilation symptom questionnaire, and individuals with many hyperventilation-related symptoms were followed over time. Measurement
issues related to the short term (i.e., test-retest) reliability and validity of the hyperventilation symptom screening questionnaire (HVQ) were also addressed. Validity of the questionnaire was examined by assessing the end-tidal CO₂ level of individuals endorsing many or few symptoms on the hyperventilation symptom questionnaire.

The relationship between HVS and panic disorder was examined in several ways. Although assessment of hyperventilation symptom stability is useful in itself, investigating the relationship of these symptoms to panic attacks over time can provide valuable information on the nature of the relationship. This investigation also examined empirically the suggestion by Cowley and Roy-Byrne (1987) that an individual's emphasis of somatic or psychological symptoms plays a role in which diagnosis an individual receives (i.e., HVS or PD). (The suggestion that caregiver emphasis on either component is also an important factor in diagnosis is noted; however, this issue will not be examined here.) Do individuals with many hyperventilation symptoms differ in the symptoms they endorse—that is, will there be subgroups of individuals who endorse only somatic symptoms as opposed to both somatic plus cognitive/affective symptoms? Examination of symptoms endorsed is the first step in testing the hypothesis that individual's present different types of symptoms.
Secondarily, this study examined variables that may be potentially related to the formation of separate symptom subgroups, specifically repression and anxiety sensitivity. Use of a repressive coping style may be related to a predominantly somatic symptom presentation, which could lead to a diagnosis of HVS. Individuals who do not use such a coping strategy may endorse somatic and psychological symptoms equally and may be more likely to receive a diagnosis of PD. Furthermore, individuals who use a repressive coping style and ignore signs of anxiety are unlikely to be high on anxiety sensitivity. Given the hypothesized etiologic role of anxiety sensitivity in the development of anxiety disorders (McNally & Lorenz, 1987), examining this variable in conjunction with hyperventilation symptoms may help identify individuals who will experience continued high levels of hyperventilation symptoms over time or possibly the onset of panic attacks during the time of the study. Examination of anxiety sensitivity levels in those with many hyperventilation-related symptoms can provide information relevant to assessing the cognitive model of panic (Salkovskis & Clark, 1990). If an individual is high on anxiety sensitivity, he or she may be more likely to make catastrophic interpretations of anxiety symptoms, which is posited as a central component of the cognitive model of panic. Individuals with many symptoms of hyperventilation
and high AS should therefore be more likely to have experienced panic attacks.

Symptom experience and/or personality variables may predispose an individual to develop psychopathology; however, in the absence of additional proximal stressors, such psychopathology may not emerge. This argument can be considered a diathesis-stress model of the development of pathology. Therefore, assessment of concomitant stressors is necessary. Previous research has demonstrated that the number of significant life events is likely related to the expression of psychopathology (Harder, Strauss, Kokes, Ritzler, & Gift, 1980; Holmes & Rahe, 1967); therefore, stressful life events will be monitored in this study.

Figure 1 displays the hypothesized relationships between the variables to be investigated. Measures to be used are included in parentheses and will be detailed in the Method section.

Several investigations were conducted to address the issues raised above. Study 1 assessed the stability of hyperventilation symptoms over time and the relationship of these symptoms to panic attacks. Information regarding related variables (i.e., repression and anxiety sensitivity) was also collected. The following hypotheses were addressed in this study:
Figure 1. Path diagram of hypothesized relationships between physiological processes, personality variables, and environmental events in the development of HVS and PD; measures used are in parentheses.
1. The stability of hyperventilation symptoms over certain intervals (viz., 4-6 weeks, and 3, 6 and 9 months) was hypothesized to remain high for the majority of individuals.

2. Hyperventilation symptoms and panic were hypothesized to co-occur in approximately one-half of subjects. This number was expected to increase over time; that is, some subjects who were panic-free at the beginning of the study were expected to have onset of panic attacks during the time of the investigation.

3. Individuals with many hyperventilation symptoms were hypothesized to emphasize varying combinations of cognitive or somatic symptoms. Individuals displaying similar levels of both cognitive and somatic symptoms were hypothesized to have a history of panic attacks, and individuals who endorse more somatic than cognitive symptoms of anxiety were hypothesized to have a lower prevalence of panic attacks.

4. It is further hypothesized that those high on repression would tend to endorse a more somatically oriented symptom profile.

5. Frequency of hyperventilation symptoms, high anxiety sensitivity, life stress, and low levels of repres-
sion were hypothesized to predict the presence of panic.

6. Anxiety sensitivity was assessed in all subjects with many hyperventilation symptoms. Those with panic were hypothesized to have high anxiety sensitivity, while those without panic were expected to vary on this variable.

Study 2 examined measurement issues relevant for the hyperventilation symptom questionnaire. Both reliability and validity of the questionnaire were assessed. The following are the specific hypotheses addressed in this regard:

1. In order to examine the validity of the hyperventilation symptom questionnaire, the end-tidal CO$_2$ level of subjects scoring at various ranges on the questionnaire was assessed. CO$_2$ level was expected to vary with the frequency of symptoms endorsed, with CO$_2$ decreasing as frequency of symptoms endorsed increased.

2. Two-week test-retest reliability estimates were calculated to examine stability of hyperventilation symptoms over short periods of time. Symptoms were hypothesized to be relatively stable over this brief interval.
3. The factor structure of the HVQ was hypothesized to provide evidence of various symptom clusters, as the HVQ was created by examining the empirical literature for characteristic symptoms of hyperventilation that resulted in the development of symptom groups.
CHAPTER 2

STUDY 1

Method

Subjects

Undergraduate students participating in introductory psychology courses at the University of Arizona during the Fall 1989 and Spring 1990 semesters served as subjects. Students from two consecutive semesters (Fall 1989, \( N = 1441 \); Spring 1990, \( N = 441 \)) were screened using a hyperventilation symptom questionnaire. Table 2 provides information on the screening samples. Students scoring 35 and above and who were free from respiratory disease were randomly selected and recruited by phone for participation in a long-term study of symptoms and stress in college students. Using these criteria, 61 individuals from the fall semester and 37 from the spring semester were potential subjects. A total of 27 subjects from the fall semester (44% of those eligible) and 23 subjects from the spring semester (62% of those eligible) agreed to participate in the two required testing sessions. Mean age (and SD) for the subjects from the fall semester was 19.04 (1.74), and the mean score on the screening questionnaire was 42.19 (7.89). This sample comprised 20 females and 7 males.
Table 2. Demographics for Screening Samples by Sex and Frequency of Hyperventilation Symptoms.

<table>
<thead>
<tr>
<th>Group</th>
<th>Females</th>
<th>Males</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fall 1989</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Likely</td>
<td>37</td>
<td>24</td>
<td>61</td>
</tr>
<tr>
<td>Midrange</td>
<td>603</td>
<td>330</td>
<td>933</td>
</tr>
<tr>
<td>Unlikely</td>
<td>236</td>
<td>211</td>
<td>447</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1441</td>
</tr>
<tr>
<td>Spring 1990</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Likely</td>
<td>24</td>
<td>13</td>
<td>37</td>
</tr>
<tr>
<td>Midrange</td>
<td>160</td>
<td>141</td>
<td>301</td>
</tr>
<tr>
<td>Unlikely</td>
<td>48</td>
<td>55</td>
<td>103</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>441</td>
</tr>
</tbody>
</table>
Subjects from the spring semester had an average age of 19.52 (2.33) and a mean score on the HVQ of 40.43 (7.89). Once again, females predominated (n = 16) over males (n = 7). Therefore, of the total sample (N = 50), 36 were female and 14 were male. Subjects recruited from the fall sample were paid four dollars/hour for their participation, and subjects recruited from the spring sample received class credit for participating.

Materials

Various questionnaires were used to gather information on hyperventilation symptoms, panic history, anxiety sensitivity, anxiety symptoms, repression, and current stressors. These are described below:

1. Hyperventilation Symptom Questionnaire (HVQ): On this self-report inventory, used in previous research as a hyperventilation screening questionnaire (Huey & West, 1983), subjects report the frequency of 29 hyperventilation-related symptoms during the past four months. Six frequency categories are assigned values of 0 through 6. Symptoms are weighted according to how diagnostic of hyperventilation they are reported to be in the clinical literature. Related symptoms are grouped after weighting; only the highest scoring symptom in any group is added to the total score. The scale has a
high level of internal consistency (Cronbach's alpha = .86). Those scoring 35 and above are designated as likely hyperventilators, and those scoring 5 or below are classified as unlikely hyperventilators. These classifications have been physiologically validated (Huey & West, 1983). Roughly 10% of a nonclinical population evidences symptoms indicative of hyperventilation (Huey & Sechrest, 1981).

2. The Panic Attack Questionnaire (PAQ): This four part questionnaire gathers the following information: demographic data, frequency and symptom profile of panic attacks, panic in first degree relatives, and the effects of panic attacks on a person's lifestyle (Norton, Cairns, Wozney, & Malan, 1988). Test-retest reliability is adequate, with kappa values ranging from .65 to 1.00 for all items except reports of unexpected attacks and those evaluating onset to peak severity of symptoms (Margraf & Ehlers, cited in Norton et al., 1988). Individuals were classified as having panic if, following the description of panic attacks provided on the HVQ, subjects indicated that they had experienced a panic attack during the previous year. If they had, subjects then completed a series of questions concerning the attacks, such as frequency, symptoms
experienced, contextual factors, etc. If subjects had not experienced a panic attack during the previous year, only demographic information and data on panic among immediate family members was obtained. The information obtained from the PAQ for this study involved a dichotomous coding system, with 1 indicating the presence of panic attacks and 0 indicating the absence of panic.

3. Taylor Manifest Anxiety Scale (Bendig Form; TMAS) and the Marlowe-Crowne Social Desirability Scale (M-CSD): Repression was measured by the TMAS and the M-CSD combined. The TMAS has 20 items assessing trait anxiety, and the M-CSD has 33 items that reflect socially desirable behaviors that most individuals do not adhere to rigidly. The M-CSD has been used as a measure of "repressive defensiveness" (Weinberger, Schwartz, & Davidson, 1979). All 53 items on the combined version of the questionnaire are answered in a true-false format. Those who report low anxiety on the TMAS but high defensiveness on the M-CSD are considered to be repressors (Weinberger, Schwartz, & Davidson, 1979). Repressors are defined as scoring 9 or below on the TMAS and 17 or above on the M-CSD (Jamner, Schwartz, & Leigh, 1988).
4. Anxiety sensitivity was measured using the Reiss-Epstein-Gursky Anxiety Sensitivity Index (ASI). The questionnaire consists of 16 items, each expressing a concern about possible negative consequences of anxiety symptoms (McNally & Lorenz, 1987). High anxiety sensitivity subjects score 30 or above on the ASI; low anxiety sensitivity subjects score 10 or below.

5. Cognitive-Somatic Anxiety Questionnaire (CSAQ): This cognitive-somatic trait anxiety inventory was constructed by selecting items from questionnaires that three independent judges agreed reflected cognitive or somatic anxiety (Schwartz, Davidson, & Goleman, 1978). The CSAQ has 14 items, seven for each type of anxiety. Subjects rate the degree to which they typically experience the symptom when they feel anxious (i.e., 1 = "not at all," to 5 = "very much so"). Correlations between the cognitive and somatic scales of the CSAQ and the State-Trait Anxiety Inventory (trait form) are .67 and .40, both of which are significant. The cognitive and somatic scales correlate .42 with each other, indicating only a moderate correlation between these types of anxiety.
6. Student Stress Scale (SSS): This 31-item scale, utilized in student health center populations, assesses stressful life events during the previous six months and those that are anticipated to occur in the coming six months. Each stressor is assigned a number of points (ranging from 20 to 100), depending on the severity of the event. For example, death of a close family member is rated 100, and minor traffic violations are rated 20. This measure provides a rough estimate of the number of stressful life events each subject has and expects to experience, and allows assessment of the change in such stressors during the time of the study.

Design and Procedure

All subjects (N = 50) participated in two data collection sessions, in February 1990 and again in late April/early May of the same year. The average length of time between the two sessions was 9.1 weeks; this time ranged from 8 to 10.5 weeks. The following questionnaires were completed at the first session: HVQ, PAQ, ASI, TMAS/M-CSD, and the SSS. At the second session, subjects completed the HVQ, PAQ, CSAW, and the SSS. Subjects were tested in small groups (e.g., 2-3) during the first session and individually during the second session, which allowed for confidentiality during the debriefing at the end of the
second session. Debriefing involved an explanation of HVS and PD, as well as brief exploration of hyperventilation symptoms and how bothersome they were. Discussion of various questionnaires followed, and clarification of items or other information sought was provided. Subjects were then thanked, paid or given credit for their participation (for Fall or Spring subjects, respectively), and dismissed.

For the 27 subjects recruited from the Fall 1989 sample, the first testing session occurred six months post-initial screening with the HVIJ, and the second session occurred 8-9 months post-initial screening. This group of subjects therefore provided data on the longer term stability of the HVIJ. Subjects recruited from the Spring 1990 sample (n = 23) were evaluated at 4 weeks, then again at 3 months post-initial screening. This latter group of subjects therefore provided information on stability of hyperventilation symptoms over a shorter period of time. Figure 2 depicts graphically the data collection procedure used. Information on the relationship between hyperventilation symptoms and panic attacks, as well as information regarding anxiety sensitivity, repression, types of symptoms, and number of stressful events during the six months preceding, and predicted to be following the data collection session, was gathered from all subjects (N = 50).
Figure 2. Timeline for screening and data collection of fall and spring subsamples.
CHAPTER 3

RESULTS

As t-tests revealed no differences between subjects recruited from the fall semester and those recruited from the spring semester in either age or score on the HVQ at initial screening, these subsamples were combined in subsequent analyses.

Stability of Hyperventilation Symptoms

Pearson product-moment correlations were calculated between HVQ scores over one, two, three, six, and eight-nine month intervals. Stability over a one-month period was calculated from the time of the initial screening to the time of the first testing session for the spring subjects, $r = .48, p = .0205 \ (n = 23)$. Stability over a two-month period was similar, $r = .49, p = .0003, \ (N = 50)$. (Two-month intervals occurred for both spring and fall subjects in the interval between testing sessions.) From the spring sample, stability estimates taken over a three-month period (from screening to second testing session) were slightly lower than for two-month estimates, $r = .39, p > .01 \ (n = 23)$. Estimates of stability of hyperventilation symptoms over intervals of six and eight-nine months were
both taken from the fall sample (n = 27), r = .14, p = .4830, and r = .22, p > .01, respectively. As these correlations are likely attenuated due to the restricted range of HVQ scores in the total sample, these scores are displayed graphically. Figure 3 depicts scores over a three-month interval, and Figure 4 depicts scores over six and nine-month intervals. Figure 3 depicts stability of hyperventilation symptoms over the 3-month interval between screening and the final assessment for the spring sample (n = 23). While for the majority of individuals hyperventilation symptoms decrease over time (i.e., 16 of 23), the symptoms remain present to a significant degree. In fact, 18 of 23 subjects still score at or near 30 on the HVQ after three months. This situation is also apparent in Figure 4, in which stability of hyperventilation symptoms over 6- and 9-month intervals is depicted for the fall sample (n = 27). While hyperventilation symptoms decrease over these intervals, the symptoms are still present in the majority of subjects to a significant degree. At 6 months, 70% of subjects score over 30 on the HVQ, and 52% score over 35 (i.e., remain likely hyperventilators). At 9 months, 74% score over 30 and 44% score over 35 on the HVQ.
Figure 3. Stability of hyperventilation symptoms over 3 months for Spring sample (n = 23).
Figure 4. Distribution of HVQ scores for large sample (n = 446).
Relationship of Panic to Hyperventilation Symptoms

To examine the co-occurrence of panic attacks with hyperventilation symptoms, correlations between scores on the HVQ and panic were calculated. The presence or absence of panic was determined using data from the PAQ given at the second testing session, as it became clear that two subjects who endorsed having panic attacks on the PAQ at the first session did not, in fact, experience true panic attacks. During debriefing, upon hearing a verbal description of panic attacks from the experimenter, these subjects stated that although they had completed the first PAQ as if they had panic attacks, that they did not in fact have attacks. (These subjects described non-clinical, situationally appropriate anxiety or general nervousness.) Therefore, these subjects were not counted as having endorsed panic on the second PAQ. Information on panic attacks from the PAQ given the second time is therefore more accurate and was used in this and subsequent analyses. The Pearson product-moment correlation between HVQ scores at the first testing session and panic scores at the second testing was low: $r = .22$. Correlation between HVQ scores at the second testing and panic at the second session was moderate: $r = .36$. As these correlations are likely attenuated due to restriction of range of HVQ scores, percentages were calculated. The percentage of individuals endorsing panic at the first
session was 70% (35 of 50); this was amended to 66% who endorsed panic at the second session (33 of 50).

A further measure of the relationship between hyperventilation symptoms and panic was determined by the number of individuals who had onset of panic attacks during the course of the study. Of 50 subjects, 4 developed panic attacks during the time of the study, representing 8% of the total sample and 22% of those who were probably truly panic free at the outset of the study.

**Cognitive and Somatic Symptoms**

Individuals with and without panic did not differ in terms of types of symptoms of anxiety they endorsed. One-way ANOVAS for cognitive symptoms by panic were $F(1,48) = .00$; for somatic symptoms by panic, $F(1,48) = 2.19$ were both nonsignificant. Mean scores on cognitive symptoms were 19.00 (6.4) for non-panickers ($n = 17$) and 19.12 (3.2) for panickers ($n = 33$). There was, however, a trend toward those with panic to endorse a higher number of somatic symptoms than non-panickers. Mean scores on somatic symptoms were 19.12 (.58) for panickers and 17.53 (4.22) for non-panickers.

**Repression and Type of Symptoms Endorsed**

A total of 6 subjects met the criteria for being termed a repressor; 44 did not. Repressors endorsed
significantly fewer cognitive symptoms of anxiety than did non-repressors: \( t(48) = 2.15, p < .05 \). Mean numbers of cognitive symptoms endorsed were 14.16 (4.96) and 19.68 (5.99) for repressors and non-repressors, respectively. Repressors and non-repressors did not differ significantly in terms of somatic symptoms: \( t(48) = 1.13 \).

**Anxiety Sensitivity and Panic**

Subjects with and without panic did not differ significantly in anxiety sensitivity, \( t(48) = -1.11 \), although there was a trend for those with panic to have a higher level of anxiety sensitivity. Mean ASI scores were 24.81 (8.58) and 21.94 (8.93) for those with and without panic, respectively.

**Prediction of Panic**

Multiple regression was used to predict the presence of panic from the frequency of hyperventilation symptoms, level of anxiety sensitivity, presence of repression, and the number and severity of life stressors. Table 3 displays the simple correlations between predictors and panic. Intercorrelations between the independent variables are minimal. Simple correlations between each predictor and panic are also low, with the exception of a moderate correlation between HVQ scores and panic. Only frequency of hyperventilation symptoms was a significant predictor of
Table 3. Simple Correlations among Variables in the Multiple Regression Equation.

<table>
<thead>
<tr>
<th></th>
<th>HVQ</th>
<th>ASI</th>
<th>Stress</th>
<th>Repression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Panic</td>
<td>.36</td>
<td>.16</td>
<td>.14</td>
<td>-.12</td>
</tr>
<tr>
<td>HVQ</td>
<td>--</td>
<td>.14</td>
<td>.24</td>
<td>.04</td>
</tr>
<tr>
<td>ASI</td>
<td>--</td>
<td>--</td>
<td>.19</td>
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</tr>
<tr>
<td>Stress</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>.01</td>
</tr>
</tbody>
</table>
panic: $F(1,48) = 7.01$, $p < .05$, explaining 12.7% of the variance in panic. When anxiety sensitivity was forced into the equation, a non-significant increase in variance explained was noted (from 12.7% to 13.9%, $t(47) = .795$, $p = .4305$).
Hyperventilation symptoms are moderately stable over short periods of time (i.e., 1-3 months). Over longer intervals (i.e., 6-9 months) stability declines sharply, yet the majority of individuals with many symptoms do continue to display these symptoms over time. Examination of changes in individual HVQ scores over these intervals reveals that although regression to the mean did occur for nearly half the subjects followed over the academic year, the majority of individuals still scored 30 or greater on the HVQ. Therefore, symptoms decreased over time for many of the subjects studied, but many of these individuals did not become asymptomatic and in fact continued to experience a moderately high level of symptoms. This finding supports suggestions that hyperventilation in some individuals is chronic, while in others it consists of recurrent symptoms (Magarian, 1982). The HVQ has demonstrated sensitivity to fluctuations in hyperventilation symptoms over time, a desirable characteristic in a symptom questionnaire that increases its clinical utility.

The degree of overlap of hyperventilation symptoms and panic is substantial, with 66% of subjects endorsing
many symptoms as well as acknowledging the presence of panic attacks. The majority of individuals who reported panic appeared to experience symptoms of sufficient frequency and severity to meet Norton et al.'s criteria (1988) for his classifications of frequent, recent, and infrequent panickers. These individuals reported that attacks with at least four symptoms of moderate severity had occurred within the previous four weeks (three or more attacks for frequent panickers, one to two for recent panickers, and not during the last four weeks for infrequent panickers). These individuals ($n = 30$) are to be contrasted with what Norton et al. (1988) term limited symptom panickers, who endorse panic but experience fewer than four symptoms that are at mild levels of severity. Only three subjects in the present study met criteria for limited symptom panickers, who were shown by Norton et al. (1988) to differ significantly from the other groups of panickers on various measures of psychopathology (i.e., in the less pathological direction). In addition, the direction of the relationship between symptoms of hyperventilation and panic is clear for a small subgroup of individuals ($n = 4$) in whom a high level of symptoms preceded the onset of panic. For these individuals, a propensity toward responding to stress with the respiratory system may have predisposed them to experience panic attacks. Although these individuals reported a
limited number of attacks and would not fit the diagnostic
criteria for panic disorder as set forth in DSM-III-R, they
are at increased risk for developing the disorder.

No significant differences in types of symptoms
(cognitive vs. somatic) were found for individuals with and
without panic. Individuals with and without panic displayed
similar levels of both somatic and cognitive symptoms,
although there was a tendency for individuals with panic
attacks to endorse more somatic symptoms than non-panickers,
a finding noted by Barlow (1988). The emphasis on cognitive
symptoms, therefore, did not serve to distinguish subgroups
of individuals with many hyperventilation symptoms as hypo-
thesized. Differential emphasis on cognitive vs. somatic
symptoms as a first step in creating separate diagnostic
entities (i.e., HVS vs. PD) does not appear to be supported
with the present data. Repression was the only variable
that was salient in predicting a differential emphasis on
symptoms endorsed. Individuals with repression did endorse
fewer cognitive symptoms of anxiety than non-repressors, but
these individuals did not differ in terms of somatic symp-
toms as hypothesized. No differences in terms of panic were
found among individuals classified as repressors or non-
repressors.

In the current sample, hyperventilation symptoms do
appear to assist in the prediction of panic, accounting for
13% of the variance in panic. As panic disorder is likely a heterogeneous classification, the predictive validity of hyperventilation symptoms is likely to be even more powerful on an individual than on a group level. As noted, for four subjects, hyperventilation symptoms did precede the onset of panic. However, additional variables that were not included in the investigation are salient to the development of panic (e.g., family history); a more inclusive model would likely have resulted in an increased ability to predict panic. Yet other individual differences variables hypothesized to be important in the development of panic were not found to differentiate between subjects with and those without a history of panic. For example, the salience of anxiety sensitivity in the development and perhaps maintenance of panic attacks was not supported. Individuals with and without panic did not differ in terms of this variable.
CHAPTER 5

STUDY 2

Method

Subjects

Undergraduate students enrolled in introductory psychology courses during two consecutive semesters (Spring and Summer 1990) at the University of Arizona served as subjects. Subjects participating in the validity portion of the study (i.e., assessment of end-tidal CO₂) were screened using the HVQ. Seven of the likely hyperventilators from Study 1 participated in Study 2; the other subjects were selected from the summer classes. A total of 112 individuals completed the HVQ from the summer classes. Potential subjects were divided into three categories, based on the HVQ: likely hyperventilators (scoring 35 or greater, n = 14), mid-range (scoring from 5.5 to 34.5, n = 79), and unlikely hyperventilators (scoring 5 or below, n = 19). Individuals scoring in each range who were free from respiratory disease were randomly selected and contacted by phone. Recruitment continued until 10 individuals at each scoring level agreed to participate in the study. (Only three likely hyperventilators were recruited from the summer sample; the other seven came from the Spring 1990 sample.)
Mean scores on the HVQ for the groups tested were 44.00, 19.35, and 1.90 for likely hyperventilators, mid-range scorers, and unlikely hyperventilators, respectively. Seven of the likely hyperventilators were female, as were four of the mid-range scorers and two of the unlikely hyperventilators, for a total of 13 women and 17 men. All subjects received class credit for their participation.

Test-retest reliability estimates were taken from students in the Summer 1990 introductory psychology courses. A total of 112 subjects received the HVQ; 82 completed the HVQ again two weeks later (n = 49 females; n = 33 males). All subjects received class credit for their participation.

Finally, HVQ data from all subjects who participated in the Spring 1990 screening for both Study 1 and Study 2 (N = 441) were used to explore the factor structure of the HVQ.

Materials

A Beckman LB2 Infrared Medical Gas Analyzer was used to assess percent CO₂ in expired air. The instrument was calibrated using a tank of 5.3% CO₂. Straws and noseclips were used to collect air samples; a stopwatch was used to time intervals between breath samples.

The HVQ (described in Study 1) was used for the test-retest reliability study, factor analysis, and screen-
ing of subjects for placement in scoring categories for the assessment of end-tidal CO₂.

**Procedure**

For the end-tidal CO₂ assessment, subjects were contacted by phone and informed that they were eligible to participate in a study assessing moisture content in air samples. If subjects had known that CO₂ content was the subject of study, individuals could have altered their natural breathing patterns as it is common knowledge that humans inspire oxygen and expire CO₂. Ratings of CO₂ would be altered if subjects changed their patterns of expiration.

When subjects arrived at the laboratory, they were seated and given an overview of the air sampling procedure. The experimenter demonstrated use of the noseclip and straw. Subjects placed the noseclip on and were instructed to hold the straw between their teeth (without closing the opening of the straw) and to sit quietly and relax (as if watching television) as the experimenter wrote down data displayed on the analyzer monitor. Direct references to breathing were specifically avoided. The straw was connected to the analyzer by a clear plastic tube, which was long enough to reach the machine while the subjects sat in an upright position. Five five-breath samples, with 20 seconds between samples, were collected from each subject. The first five-breath epoch was to assist adaptation to the equipment and
was not used in subsequent analyses. The CO₂ levels from the following 20 breaths constituted the CO₂ measure. Once sampling was completed, subjects were debriefed as to the true nature of the study and given a full explanation of physiological validation of paper and pencil measures. No subject professed awareness that CO₂ had been the object of investigation. Subjects were then thanked, given their credit for participation, and dismissed.

For the test-retest reliability portion of the study, subjects completed the HVQ twice, with an interval of two weeks between each administration. The HVQ was administered in classrooms to individuals who volunteered to stay after class to participate in the study. Of the four classes used in data collection, approximately 4-5 students per class left before completing the instrument the first time. The experimenter returned to these same classes two weeks later for the retest, which included only subjects who had taken the HVQ the first time. Of the original 112 subjects, 82 completed the HVQ the second time, representing a 73% response rate. After the second administration, subjects were debriefed, thanked, and given credit for participation.
CHAPTER 6

RESULTS

A one-way ANOVA assessing level of CO₂ by group (likely, mid-range, and unlikely) was nonsignificant, F(2, 27) = 2.20. However, mean CO₂ levels were in the expected direction. Likely hyperventilators had a mean CO₂ level of 5.01, mid-range scorers had a mean of 5.18, and unlikely hyperventilators had the highest mean CO₂ level at 5.44. Figure 5 displays CO₂ level as a function of frequency of hyperventilation symptoms. As can be seen, the amount of variance explained by the regression equation describing the fit of the regression line to the data is minimal.

To assess the two-week test-retest reliability of the HVQ, a Pearson product-moment correlation was calculated. The correlation was significant: r = .79, p < .001.

In order to assess the factor structure of the HVQ, responses to 27 of the 29 individual items were factor analyzed through the method of principal factors, with squared multiple correlations in the diagonals of the correlation matrix as the initial communality estimates. (The two items omitted from the factor analysis were the last two items on the HVQ which asked whether or not the
Figure 5. Mean CO$_2$ level as a function of frequency of hyperventilation symptoms.
symptoms appeared during times of stress and a final listing of which symptoms appeared together. Twenty-six items in the factor analysis all pertained to frequency of individual symptoms, and one item was a rating of how bothersome the symptoms are.) An orthogonal varimax prerotation method was followed by an oblique solution using promax rotation. Examination of the scree plot of the eigenvalues showed two possible cutoff points in defining the number of factors, at either one factor or five. Eigenvalues and percent of variance accounted for by each factor are as follows. The eigenvalue for factor 1 is 6.50, accounting for 73.2% of the variance. Eigenvalues for factors 2 and 3 are .94 and .80, which account for 10.65 and 9.04% of the variance, respectively. Eigenvalues for factors 4 and 5 are .58 and .50, which accounts for 6.5 and 5.65% of the variance, respectively. Table 4 lists the promax rotated factor pattern and the final communality estimates for each of the hyperventilation symptoms from the HVQ used in the factor analysis. While a five-factor solution is possible, a one-factor solution, suggesting that the HVQ taps one construct, appears most reasonable, given both the large percentage of variance the first factor accounts for, as well as the correlations between the factors obtained, which range from .52 to .69. The first factor, labelled the cognitive factor, was comprised of symptoms of subjective anxiety, how
Table 4. Promax Rotated Factor Pattern and Final Communality Estimates for Hyperventilation Symptoms on the HVQ.

<table>
<thead>
<tr>
<th></th>
<th>Factor 1</th>
<th>Factor 2</th>
<th>Factor 3</th>
<th>Factor 4</th>
<th>Factor 5</th>
<th>FCE'S</th>
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<td>.46</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.44</td>
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<tr>
<td>How bothersome</td>
<td>.45</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.45</td>
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<tr>
<td>Problems conc.</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>.45</td>
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<tr>
<td>Lightheaded</td>
<td></td>
<td>.56</td>
<td></td>
<td></td>
<td></td>
<td>.56</td>
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<td></td>
<td>.55</td>
<td></td>
<td></td>
<td></td>
<td>.56</td>
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<tr>
<td>Faintness</td>
<td></td>
<td>.55</td>
<td></td>
<td></td>
<td></td>
<td>.42</td>
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<tr>
<td>Faint lights</td>
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<td>.54</td>
<td></td>
<td></td>
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<td>Cold sweat</td>
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<td>.46</td>
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<td>.30</td>
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<tr>
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<td></td>
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<td>Rapid HR</td>
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<td>.62</td>
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<tr>
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<td>.41</td>
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<tr>
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<td>.33</td>
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<td></td>
<td></td>
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<td>.24</td>
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<td>Numbness</td>
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<td></td>
<td></td>
<td>.13</td>
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<tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>.25</td>
</tr>
<tr>
<td>Buzzing in ears</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.19</td>
</tr>
<tr>
<td>Irregular HR</td>
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<td></td>
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<td>.27</td>
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</table>
bothersome the symptoms were, and difficulty concentrating. Later factors appear to highlight the various somatic symptom constellations. The second factor taps the central nervous system symptoms of hyperventilation: i.e., light-headedness, dizziness, and faintness. The third factor consists of heterogenous somatic symptoms, which are indicative of general autonomic arousal, including visual changes (blurred vision, black spots, flashing lights) as well as cold sweat and rapid heart rate. Factor four captures respiratory/thoracic symptoms (e.g., shortness of breath, chest pain); and the fifth factor represents musculoskeletal symptoms (including classic symptoms of tetany): muscle spasms or muscular trembling/twitching.
Although mean CO$_2$ levels did not differ significantly for subjects in the three scoring categories on the HVQ (i.e., likely, mid-range, unlikely), levels exhibited were in the anticipated direction and decreased as the frequency of hyperventilation symptoms increased. These findings indicate that the HVQ is a potentially valid measure of the symptoms of hyperventilation, given that hyperventilation is defined as ventilation in excess of metabolic needs and results in a decrease in the level of arterial carbon dioxide (reflected by end-tidal CO$_2$ measures). One possible reason for the failure to attain significance in the present study is the relatively small sample size used at each scoring level. Replication of the study with a larger sample would address this issue.

Scores on the HVQ were relatively stable, as reflected by the two-week test-retest reliability estimates obtained. Hyperventilation symptoms as measured by the HVQ do not change dramatically over relatively short intervals, which has implications for future research using this instrument. Yet, perusal of the HVQ scores revealed that 17 of 82 subjects, or 20% of the sample, changed scoring
categories over the two-week period. Twelve of these subjects moved from the mid-range to the unlikely category, three from the mid-range to the likely category, and two from the likely to the mid-range category. The movement noted both into and out of the mid-range category reveals adequate flexibility in the HVQ to detect changes in symptoms over time. While good reliability in a clinical questionnaire is highly desirable, the fluctuating nature of clinical phenomena demand the ability to detect change.

Results of the factor analysis suggest that the HVQ measures one construct, although there is some evidence of symptom clusters. Given the high interfactor correlations, it is possible that one higher order factor exists which organizes the smaller factors. This factor may be interpreted as a general awareness of distress or anxiety, accompanied by disturbances in various organ systems which are consistent with the effects of hyperventilation. Which symptom cluster an individual emphasizes will likely be influenced by numerous factors, including genetic predispositions and learning history, although the likelihood of the presence of symptoms from multiple systems is high.
CHAPTER 8

GENERAL DISCUSSION

Hyperventilation symptoms appear to be relatively stable over brief intervals of 1-3 months and are relatively unstable over longer intervals (i.e., 6-9 months). Although hyperventilation symptoms fluctuate over time in the current sample, these symptoms do remain present over time to a degree suggesting chronicity. The majority of subjects did not become symptom-free and continue to score near the extreme end of the distribution of these symptoms compared to a general college population (i.e., a score of 30 or more on the HVQ) after nine months.

When hyperventilation symptoms are assessed by questionnaire in a general population, the percentage of individuals who endorse them who also have a history of panic attacks is quite high. Rate of overlap of hyperventilation symptoms and panic (approximately 60%) is nearly identical to that found in previous research (e.g., Hoes et al., 1987). While the exact nature of the relationship between hyperventilation symptoms and panic remains unclear, the present data suggest that for a small subgroup of individuals (4 of 50), hyperventilation symptoms precede the onset of panic attacks. This finding provides initial
empirical support for the hypothesized relationship between Panic Disorder and Hyperventilation Syndrome. Although there is overlap between the two disorders, they are not likely the same; however, there is hypothesized to be a subgroup of individuals with panic disorder for whom simple hyperventilation produces symptoms (Gorman & Papp, 1990).

An unexpected 8% of the present sample experienced the onset of panic during the time of the study, after a high level of hyperventilation symptoms had been documented in these individuals. For other individuals with panic disorder, hyperventilation may be merely a concomitant of an acute attack, reflecting possible hypersensitivity of CO₂ chemoreceptors that cause both hyperventilation and panic (Gorman & Papp, 1990).

The finding that prediction of panic is enhanced by the presence of hyperventilation symptoms has clinical significance in that individuals whose panic attacks increase in frequency and severity are more likely to meet the diagnostic criteria for panic disorder. Therefore, individuals who present in clinical settings with symptoms consistent with chronic hyperventilation (in whom other organic illness have been ruled out) who are not yet experiencing panic may benefit from education about potential attacks as well as training in abdominal breathing techniques to address the symptoms and possibly ameliorate the attacks. In this way,
isolated panic attacks may be treated before development of the full disorder. It is also possible that the relationship between early hyperventilation symptoms and later panic attacks may be more robust in clinical samples. Future research on the relationship of these variables in clinical populations is clearly warranted.

The relationship between hyperventilation and panic does not appear to be through differential emphasis on separate symptom subgroups (i.e., cognitive and somatic versus predominantly somatic symptoms). The hypothesis of Cowley and Roy-Byrne (1987) was not supported in that emphasis on cognitive plus somatic as opposed to somatic symptoms alone does not appear to differentiate between those with and those without panic.

The issue of the influence of personality variables on the development of panic remains unclear. While repression appears to diminish the likelihood that cognitive symptoms of anxiety will be reported, this does not alter or "protect" one from experiencing panic. Three of six subjects identified as repressors had panic attacks; in fact, two of these were among the four who developed panic during the time of the study. Clearly, the small sample size precludes definitive statements about the role of repression, but the initial results do not appear promising. A tendency to view the self as not experiencing much subjective
distress (i.e., repression) may decrease reporting of subjective feelings of anxiety, but will not in all likelihood protect one from somatic manifestations of anxiety that are central to the experience of panic attacks.

The lack of significant differences between those with and without panic on the measure of anxiety sensitivity fails to support the extant research on this construct. Although implicated in the etiology of anxiety disorders in general and in panic and agoraphobia in particular, those with panic as well as those who developed panic attacks during the time of the study did not evidence elevated levels of anxiety sensitivity. Yet the present data were gathered from a general student population and not a clinical population. The relationship between panic and anxiety sensitivity may be different in a clinical population. The present data also fail to support the cognitive theories of panic, in which catastrophic misinterpretation of the symptoms of hyperventilation leads to panic. If this were the case, then individuals with panic should have evidenced higher scores on the ASI, as it measures the fear of anxiety symptoms. Without underlying fear of such symptoms, it is highly unlikely that catastrophic misinterpretations of the hyperventilation symptoms would occur.
In fact, cognitive theories of panic have recently been challenged. Ley (1989) has revised his theory of hyperventilatory panic attacks, abandoning the idea that fear produced during panic attacks is the result of catastrophic misinterpretation of the symptoms of hyperventilation, and instead positing that the fear experienced during these attacks is due to severe dyspnea and concomitant fear plus the perception of lack of control over the onset of the dyspnea. Therefore, individuals with hyperventilatory panic attacks are posited to have a very real fear of respiratory distress (an expected reaction to severe dyspnea) which is beyond their control as opposed to a general tendency to make catastrophic interpretations about anxiety symptoms they experience. The importance of a sense of control over symptoms experienced is highlighted in a study by Rapee (1986), in which he used 50% CO₂ inhalation to induce panic in the laboratory. Individuals who received no explanation of the symptoms experienced a greater intensity of panic than individuals who did receive an explanation. Future examinations of cognitive variables in panic clearly need to focus on the role of perceived control and factors that can increase or diminish this potentially protective mechanism.

Although the present investigation was not able to identify the personality variables salient in the expression of hyperventilation and panic, various measurement issues
related to assessing hyperventilation symptoms were addressed. For example, a high probability of selecting panic sufferers exists when using the HVQ as a screening device for research purposes. Adequate test-retest reliability is demonstrated, but support for validity of the measure as assessed by end-tidal CO₂ is more qualified. However, future research assessing CO₂ levels for those scoring at all ranges with larger samples is indicated before definitive statements can be made. Previous research demonstrating differential responses to trials of voluntary hyperventilation for likely as opposed to unlikely hyperventilators supports the utility of a self-report measure for assessing hyperventilation. Furthermore, rates of overlap of HVS and PD are nearly identical when identified by paper and pencil measures of symptoms as compared to the more involved and time consuming diagnostic procedures utilized in previous research. Therefore, the HVQ can be used in combination with the PAQ to screen large samples of individuals for further research on the relationship between hyperventilation and panic.

Finally, one of the most intriguing findings in the current research (which was not a focus of study) were the sex differences noted among subjects identified as likely hyperventilators. Among the subjects screened using the HVQ, more females than males were noted in all categories;
however, this discrepancy was greatest among the likely hyperventilators. Furthermore, among the 30 subjects selected for measurement of end-tidal CO₂, females predominated in the likely category (7 of 10) as opposed to the mid-range (4 of 10) or unlikely category (2 of 10). Clearly the issue of sex differences in hyperventilation symptoms deserves further investigation; these preliminary findings are suggestive of similar sex differences seen in hyperventilation symptoms, as have been noted in panic (i.e., approximately 2:1, Shapiro, 1989).

Limitations in the present research prohibit conclusions to be made about panic disorder and hyperventilation syndrome in clinical populations. This research focussed on symptoms of the two disorders, and no attempt was made to formally diagnose the subjects. Furthermore, assessment of both hyperventilation symptoms and panic attacks was accomplished using self-reports. Retrospective examination of panic attacks, in particular using a paper and pencil measure, was shown to be sensitive to the interpretation bias of the individual subject. Combining self-report data from questionnaires with a standard clinical interview would have served to increase validity of the findings. In addition, the length of time over which subjects were followed in the present research was relatively short. Documentation of chronicity of hyperventi-
lation symptoms would require examination over a period of years, not months.

Although the addition of formal diagnosis to the present research would have been desirable, this is not possible at present for HVS. No formally accepted definition of symptomatology or diagnostic criteria exist, although attempts in this direction are being made (Lum, 1987). Given this state of affairs, attempts to delineate the exact relationship between hyperventilation and panic are also hampered. However, the current research is consistent with previous research in demonstrating a substantial overlap of the symptoms of hyperventilation and those of panic. Treatment implications for HVS and PD can be drawn from this confluence of data. Respiratory treatments for panic attacks and PD are clearly warranted, as regardless of the direction of the relationship, the disorders overlap to a significant degree.

Future research on specific diagnostic criteria for HVS is clearly warranted. Once this is accomplished, long-term, prospective studies of children with symptoms of hyperventilation and/or anxiety would highlight variables salient to the development of chronic hyperventilation and panic. The nature of the relationship between these two disorders would also be addressed in this manner. Data collection from multiple sources (i.e., self-report,
physiological, and behavioral channels) would increase the validity of information obtained as well as knowledge of the various manifestations of these disorders. Furthermore, examination of the context within which such disorders develop (i.e., families) would broaden the scope of such research in examining the role of factors such as learning history as well as genetic predisposition, in the development of hyperventilation and panic in particular, as well as psychopathology in general.
REFERENCES


