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CHILDRENS PHYSICAL SYMPTOM REPORTING AND
THE TYPE A BEHAVIOR PATTERN

by

LEWIS J. LEIKIN

Thesis submitted to the School of Graduate Studies of
the University of Ottawa as partial fulfillment of the
requirements for the degree of Doctor of Philosophy.

Ottawa, Canada,

1986

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CHAPTER 1

LITERATURE REVIEW AND RATIONALE FOR THE STUDY

Coronary heart disease (CHD) continues to be the leading cause of death in Canada and the United States, accounting for 40% of deaths among middle aged men and costing an estimated 3 billion dollars in medical expenses and lost wages in Canada alone (Canadian Heart Foundation, 1982). By definition, CHD refers to those cardiovascular disorders characterized by inadequate oxygen supply to the heart, and its symptomatic forms are angina pectoris (severe chest pain) and myocardial infarction (heart attack).

Traditionally the effort to predict CHD morbidity and mortality has been based on three major risk factors; elevated blood pressure, serum cholesterol, and smoking (Braunwald, 1980). However, combining these risk factors predicts less than half the incidence of new CHD occurrences (Jenkins, 1983). In the search for additional risk factors an important outcome has been the recognition that behavioral factors may play a role. In broad terms such behavioral risks include smoking, diet, and physical inactivity. Over the past two decades another coronary prone behavior, namely the Type A behavior pattern, has received considerable attention.

The Type A behavior pattern is characterized by a chronic struggle to achieve an ever escalating but ambiguously defined set of goals and consists of a constellation of core behaviors including high levels of competitive achievement striving,

easily aroused hostility, aggressiveness, impatience and time urgency, and a hard driving job involvement (Friedman and Rosenman, 1974).

The coronary risk conferred by the Type A behavior pattern has been established by both retrospective and prospective epidemiological work such as the 11,000 subject Belgian Heart Study (de Backer et al, 1983), the Western Collaborative Group Study (Rosenman et al, 1976) and the Framingham Heart Study (Haynes et al, 1980). For example, 8 year incidence data found initially healthy Type A adults had over twice the rate of infarction, 3 times the rate of angina, were twice as likely to die from their heart disease and 5 times as likely to have a recurrent infarction compared to Type B men (Haynes et al, 1980; Rosenman et al, 1976). The Type A risk is maintained after statistically controlling for other known risk factors (Rosenman et al, 1976; Haynes et al, 1980). The Type A risk status is corroborated by findings from a randomized, controlled clinical trial that lowering Type A behavior significantly reduces the recurrence rate of infarction (Friedman et al, 1984) suggesting a causal relationship between Type A behavior and coronary heart disease (Siegel, 1984). Recently the National Review Panel on Coronary Prone Behavior sponsored by the National Heart, Lung and Blood Institute, declared the Type A behavior pattern as an independent risk factor for CHD, with an order of magnitude equal to the other major risks of smoking, elevated blood pressure and serum cholesterol (N.H.L.B.I., 1981).

It should be noted that 2 recent prospective studies, the

Multiple Risk Factor Intervention Trial (MRFIT, 1982) and the Aspirin Myocardial Infarction Study (Case et al, 1985), found no relationship between Type A behavior and the incidence or recurrence of infarction among high risk males. While there were some methodological problems in these studies, including the use of high risk subjects for whom the predictive validity of traditional risk factors is not yet known (Siegel, 1984) and the assessment of Type A behavior in hospital while on beta-blocker therapy, which reduces Type A expression (Krantz, 1985), the data underscores two important issues in Type A research. Namely, the risk conferred by the Type A pattern varies with the type of assessment used, and secondly, not all aspects of the behavior pattern are pathogenic or coronary prone. These issues are currently dominating the field and are herein reviewed.

ASSESSMENT ISSUES OF THE TYPE A BEHAVIOR PATTERN

Three measures of Type A behavior have been prospectively related to the occurrence of CHD. These are the Structured Interview (Rosenman et al, 1976), the Jenkins Activity Survey (Jenkins, 1971), the Framingham Type A scale (Haynes et al, 1978). A fourth measure, the Bortner Rating Scale (Bortner, 1969) has been retrospectively related to the prevalence of CHD.

The Structured Interview, developed by Friedman and Rosenman and used in the Western Collaborative Group Study, consists of 26 questions that inquire about the individual's drive level, frequency of anger, and typical reactions to delay, competition, and deadlines. Some questions are administered provocatively to assess actual speech behavior as the major focus for assessment

is interviewee behavior rather than content. Individuals are classified as either Type A1 (fully developed), Type A2 (weak or incompletely developed), X (mixed amount of A and B) and Type B (absence of Type A features). Twenty years of research on the measure has yielded strong psychometric support for its use. Inter-rater reliability has ranged from 75% to 90% (Rosenman, 1978) and test-retest reliability over a period of 12 to 20 months is quite high at .82 (Chesney, Eagleston, and Rosenman, 1981). Construct validation studies finds interview assessed adults to be more competitive and impatient, have higher standards of self-evaluation, and show greater hostility when challenged (Matthews, 1982). Predictive validation has been provided by the Western Collaborative Group Study, yielding an independent risk for CHD of 2.2 Type A over Type B subjects. Of the available Type A measures, the interview is the strongest predictor of CHD outcome and for adult populations is the measure of choice (Matthews, 1982). The prevalence of interview identified Type A's varies with gender (Canadian estimates range from 50-60% among males (Howard et al, 1977), occupation and culture, with lower rates in lower socio-economic, European and Japanese groups (Waldron, 1978).

The Jenkins Activity Survey or J.A.S. was developed in order to provide an easy to administer, inexpensive self report measure of Type A. Its 52 items were selected from Structured Interview questions that were statistically best able to discriminate A's from B's. The original scale has been revised several times and now exists in adult and college forms. The

J.A.S. yields an overall Type A score and 3 factor derived scores; speed and impatience (Factor S), job involvement (Factor J) and hard-driving, competitive (Factor H). The J.A.S. has high test-retest reliability (Johnston and Shaper, 1983) and excellent construct validation support (Matthews, 1982). The J.A.S. does not predict CHD as well as the Structured Interview, conferring only a 1.8 times greater risk for Type A's over Type B's (Matthews, 1982). Of the factor scores, only the hard driving component (Factor H) distinguishes coronary patients from controls (Jenkins, Zyanski and Rosenman, 1971). Moreover, the J.A.S. has only a modest degree of overlap with the Structured Interview in classifying subjects ranging from 68% to 72% concordance (Krantz, 1985). These data have led reviewers to conclude that the Structured Interview and the J.A.S. do not measure the same Type A behaviors even though they are both measures of coronary proneness (Matthews, 1982, Krantz, 1985, Siegel, 1984).

The Framingham Type A scale is a 10 item self-report measure. Items were chosen by a panel of experts from a pool of 300 questions, for use in the Framingham Heart Study. The Framingham scale was able to predict the incidence of CHD in males and females at an 8.5 year follow-up, finding a magnitude of risk of 2.9 Type A/Type B for white collar men, 1.4 Type A/Type B in blue collar men, 1.6 Type A/Type B in working women, and 2.9 Type A/Type B in housewives (Matthews, 1982). Available data on the scale indicates it has high internal consistency at .71 and good test-retest reliability at .7 (Haynes et al, 1980).

Its overlap with other Type A measures is also modest ranging from 36% to 68% (Haynes et al, 1980).

The Bortner questionnaire is a 14 item self-report measure that utilizes a semantic differential format. Respondents draw a line between two opposing behaviors thought to reflect Type A-B differences (eg. not competitive--very competitive), to indicate where he belongs between the extremes. No internal consistency data is available for this scale, but test-retest reliability is high at .8 (Johnson and Shaper, 1983). Retrospective data based on over 11,000 subjects found the prevalence of CHD to be 2 times greater among Bortner Type A individuals, compared to Type B (Koskenvuo et al, 1981).

The issue of Type A measurement continues to concern researchers. Not only does the prevalence of Type A behavior vary with education, gender and occupation, it also varies according to which measure is actually used. So too does the risk conferred by the Type A pattern vary with the assessment method utilized. Moreover, there appears to be a modest overlap at best among the various measures (Matthews, 1982). As such, recent reviews by Krantz (1985) and Matthews (1982) suggest that Type A is a multidimensional construct and not a unitary, global pattern as originally proposed. Individuals may present some component behaviors but not all and still be considered Type A. Further, each Type A measure may tap distinct aspects of the behavior pattern. The Structured Interview appears to measure a general reactivity to provocative events, the J.A.S. taps a preference for competitive achievement striving and hurried

living, and the Framingham scale measures negative self-awareness and emotional distress associated with competitive and hurried Type A behavior (Matthews, 1982). Reviewers conclude that while each method is a valid Type A measure, they should not be substituted for one another (Krantz, 1985).

In recognizing the multidimensional nature of the Type A construct there is growing consensus that not all components of the behavior pattern are coronary prone or pathogenic. In a re-analysis of the Western Collaborative Group data using a Type A component scoring system, Matthews et al (1977) found that only vigorous speech stylistics (eg. potential for hostility) and impatience and hostility items discriminated coronary from non-coronary patients. Similar data was recently reported by Dembroski et al. (1985), finding the Type A components of potential for hostility and anger-in (withholding anger), but not global Type A, to predict heart disease severity. Two large scale prospective studies have also recently found high levels of hostility to independently engender a 5-fold risk for CHD (Barefoot et al, 1983; Shekelle et al, 1983). These findings have prompted researchers to suggest that the hostility component may be a critical aspect of what makes the Type A pattern a coronary prone behavior, and that the relative weighting given to this component in Type A assessment may account for the variation across studies in the strength of the relationship between Type A and CHD.

Another issue revealed by the assessment literature concerns

the definition of the Type A behavior pattern as a typology or a continuum. In their original work, Friedman and Rosenman construed the construct as a continuum of behaviors ranging from extreme Type A to extreme non-Type A or Type B. This is reflected in the Structured Interview rating system. However, the questionnaire methods have been using cutting scores to dichotomize the distributions, thus rendering the construct a typology.

The evidence in support of either approach appears to be equivocal. To date, no dose-response relationship has been found between the severity of Type A behavior and the incidence or severity of CHD (Manuck and Krantz, 1984), nor with the progression of atherosclerosis the underlying disease process (Krantz et al, 1979). Moreover, Type A's have been described to respond to behavioral and psychophysiological stimuli in a qualitatively, not simply quantitatively different manner (Glass, 1977; Dembroski et al, 1983). These findings prompted Matthews (1982) to call for the treatment of Type A as a typology.

Contrary views have been offered by Mayes, Sime and Ganster (1984) and the National Review Panel on Coronary Prone Behavior (N.H.L.B.I.) who suggest that continuous measures achieve higher convergent and construct validity, while the typology may produce Type 2 classification errors, lead to failure in finding a dose-response relationship, and reduces comparability of findings across subjects.

In light of apparently equivocal data researchers may best

take advice from compromises offered by both sides. For practical diagnostic purposes, cutting scores may be used to dichotomize the sample (N.H.L.B.I. 1981) but should be consistent with other research, and studies may be designed to permit quantitative as well as qualitative observations (Matthews, 1982).

MECHANISMS LINKING TYPE A AND CORONARY HEART DISEASE

In broad terms there are two distinct but complimentary linkages between Type A behavior and heart disease; physiological and psychological. These are not competing explanations in the Cartesian sense, but are simply a way to conceptualize an enormous body of research. Space limitations restrict this review to a summary of current issues. The reader is directed to Matthews (1982) for an excellent review of the psychological perspectives on Type A, and to Dembroski et al (1983) or Jenkins (1983) for a thorough treatment of the pathophysiological aspects.

In terms of its pathophysiological influence, the Type A pattern appears to contribute to CHD through the sympathetic-adrenal-medullary and pituitary adrenal-cortical pathways (Manuck and Krantz, 1984). Type A individuals have consistently been found to have greater beta lipoprotein concentrations, faster blood clotting times, greater blood pressure and heart rate variability, higher serum cholesterol and serum lipid levels, and greater waking levels of catecholamines than Type B subjects (Dembroski et al, 1983). All of these processes may increase the severity of atherosclerosis, the underlying disease

process of CHD, by damaging blood vessel walls (Hoff, 1974), or promoting the production of low density lipoproteins (Friedman, 1978), the precursor of cholesterol that is deposited on arterial walls.

The dominant biobehavioral model in CHD mechanism research is psychophysiological reactivity. This refers to the repeated and excessive cardiovascular responsiveness to stress that may promote arterial injury and in turn, the progression or exacerbation of atherosclerosis. There is now a large data base demonstrating a Type A-reactivity association, and current thinking emphasizes this hyperresponsiveness as the likely mechanism underlying the behavior-disease linkage (Manuck and Krantz, 1984).

The psychological perspectives on the Type A-heart disease linkage have generically been subsumed under the broad concept of stress. Type A individuals are considered to live under and perpetuate high levels of stress which leads to damaging physio-endocrine changes and eventually to CHD (Eliot, 1979; Suinn, 1982). As Suinn (1982) notes the Type A individual is under prolonged stress from his self-imposition of deadlines, and his rapid, competitive and aggressiveness work style. Moreover, the Type A person deals with this increased stress by increasing their Type A behaviors, creating a self-perpetuating stress cycle.

To date there have been at least 4 complimentary psychological approaches to conceptualize the Type A risk. These are uncontrollability (Glass, 1977), self-involvement (Scherwitz et

al, 1978), ambiguous standards of evaluation (Matthews and Siegel, 1983) and symptom suppression (Carver et al, 1976).

The uncontrollability hypothesis states that Type A behaviors represent an attempt to assert and maintain control over stressful aspects of the environment, i.e. uncontrollable events that are perceived as harmful (Glass, 1977). Type A's therefore engage in a chronic struggle to exert environmental control and over-react, behaviorally and physiologically, when faced with threats. Available data from reactance and loss of control studies (Glass, 1977; Matthews, 1982) suggest that the hypothesis holds when loss of control threats are highly salient or brief, and that it is the perception of threat, not the event itself, that engenders reactivity (Matthews, 1982).

The self-involvement perspective comes from the work of Scherwitz, Berton and Leventhal (1978, 1983), who argue that Type A individuals have a greater focus on themselves and are more personally involved in their work, which intensifies their emotional and physiological reactivity. They found Type A persons to make more frequent self-references (I, me, mine) than Type B's, and that during those statements, Type A's showed higher blood pressure reactivity. The authors feel Type A research is hindered by its atheoretical approach and have attempted to enhance its status as a personality construct with a theoretical grounding. However, the approach carries two major problems with it. Firstly, it is not at all clear whether Type A persons are indeed more self-involved than Type B persons. Explicit examination of this, in operationalized terms, has yet

to occur. Secondly, Type A is usually conceptualized as a behavior pattern rather than a personality construct since the Type A personality correlates have not been consistently established (Glass, 1977; Dembroski et al, 1983). Thus, most theorists view Type A behavior as an outgrowth of the person-situation interaction (Glass, 1983), reflecting what Friedman and Rosenman (1974) originally referred to as an "action-emotion complex".

Matthews and her colleagues (1977, 1982, 1983) have suggested that Type A behavior reflects a strong value in productivity combined with ambiguous standards for evaluating this productivity. These standards engender a sense of time passing too quickly to accomplish ones goals. Matthews' work developed out of her interest to understand the origins of Type A behavior, how it is maintained and self-perpetuated. A growing body of literature with both children and adults confirms that Type A's make greater efforts to excel and achieve, and have higher but more ambiguous standards than Type B's for evaluating their performance (Matthews and Volkin, 1981; Matthews and Siegel, 1983; Houseworth, 1985).

The fourth approach to conceptualize the Type A risk has been called symptom suppression (Carver, Glass and Coleman, 1976). According to this model Type A's under-report their physical symptoms because they suppress their attention to subjective physical states in order to fully direct attention to performance and achievement goals. As a result, Type A's under-use medical services and expose themselves to treatment

delay. Interest in this hypothesis has grown with the recognition that not all Type A component behaviors are coronary prone (N.H.L.B.I. 1982) and that examination of actual health and illness behaviors may provide a more direct behavioral link between the psychological construct and illness end-points, as well as the opportunity to design effective behavioral interventions for specific pathogenic components.

SYMPTOM REPORTING BEHAVIOR AND THE TYPE A PATTERN

1. SYMPTOM REPORTING AND CORONARY RISK

Symptom reporting behavior is of generic importance in health psychology, but particularly so in heart disease research for two major reasons. Firstly, symptoms are the starting point in any illness and in the decision to seek medical treatment (Green, Moss and Goldstein, 1974). Symptoms act to define that something is wrong with one's body, and to represent the illness state (Leventhal, 1983). Once perceived, symptoms form the initial basis upon which individuals decide to seek or not seek medical attention (Green et al, 1974; Safer, 1979). In addition, symptom reporting is critical in doctor-patient communication and health care decision making (Leventhal, 1983).

In all disease, failure to attend to or report physical symptoms can seriously compromise health care (Safer, 1979). In heart disease such behavior may be fatal. Seventy percent of coronary deaths occur within 4 hours of symptom onset (Gillum et al, 1976). Thus, any delay in seeking medical attention for cardiac related symptoms is dangerous, and is a risk factor in

and of itself (Insull, 1973). Moreover, delay may exacerbate the severity of symptoms and the extent of any underlying disease process. The major contributor to delay is the decision to seek treatment (Green et al, 1974; Blumenthal, 1982), and within that decision making process, the major determinant is the symptom reporting experience (Matthews et al, 1982; Safer, 1979). As Gentry and Haney (1975) noted, the "most common reaction to cardiac symptoms is no reaction (p.730)". It is conceivable then, that correcting symptom reporting behavior would reduce patient delay and lower CHD mortality.

The second reason symptom reporting is of importance in CHD is that bodily symptoms provide cues for self-regulatory behavior (Pennebaker, 1982). Symptoms are used to represent underlying physical activity and to guide and alter behavior. Failure to attend to or appreciate the importance of physical symptoms impairs the individual from using somato-visceral feedback as cues to modulate behavior (Weidner and Matthews, 1978). For example, individuals' failure to attend to or report symptoms, does not allow them to use bodily symptoms elicited by stress, as cues to alter their behavior to less stressful forms. This may result in a chronic overexposure to stress, which is a major pathway to CHD.

2. TYPE A RESEARCH ON SYMPTOM REPORTING

Several theorists have attempted to extend the importance of symptom reporting behavior to account for what makes the Type A behavior pattern "coronary-prone". They suggest that Type A's risk for CHD lies in their under-reporting or suppression of cardiac related symptomatology, which results in a chronic overexposure to stress, and to an inordinate delay in seeking medical care after the onset of symptoms (Carver, Glass and Coleman, 1976; Weidner and Matthews, 1978;). This hypothesis which has become known as the symptom suppression hypothesis, places symptom reporting behavior as a causal mechanism in the Type A behavior-CHD linkage.

The hypothesis was originally proposed by Carver, Glass, and Coleman (1976). They found Type A college students exerted themselves more than Type B's on a Balke treadmill test but rated their subjective fatigue significantly lower. Differences could not be accounted for by pre-test levels of fatigue, nor aerobic capacity. Carver et al concluded that Type A's suppressed feelings of fatigue in order to over-achieve and assert mastery and control over the task. They also noted that the Type A subjects unlike the Type B's, responded affirmatively to the Jenkins Activity Survey item "Do you keep pushing yourself at the same pace in spite of tiredness?" and suggested that fatigue suppression is only one example of a generalized tendency among Type A's to under-report a variety of physical symptoms.

Since Carver et al's seminal work a large body of

experimental and clinical research has ensued on the symptom reporting behavior of Type A's. Experimental studies are reviewed first followed by clinical research work.

Weidner and Matthews (1978) assessed the reporting of a variety of symptoms by Type A and B women college women attempting to solve arithmetic problems while being exposed to aversive white noise. Half the subjects were instructed to work on the problem for 4 minutes and half for 8 minutes. However, after 4 minutes all subjects were stopped and asked to complete a 14 item symptom checklist, that include items such as racing heart, fatigue, headache and so on. Type A women failed to report a variety of symptoms despite physiologically measured increases in blood pressure and decreases in hand temperature. Interestingly, symptom under-reporting occurred only in the 8 minute condition, that is when subjects thought the task was to continue. During the 4 minute task, when no further problem solving was anticipated, A-B differences in symptom reporting disappeared. This replicated Snyder and Glass' (1974) finding that A-B symptom reporting differences occur while actively working on an experimental task, but not following its completion. Similar findings were reported by Pennebaker (1982). Hence, as with other Type A behaviors, symptom under-reporting is elicited by environmental challenge, such as work, and influenced by task duration (Walster and Aronson, 1967). These data also indicated that A-B differences are not due to differences in sensory input or failure to experience symptoms, nor to denial as Type A's report just as many symptoms as do

Type B's upon completion of the task.

In addition to environmental and task variables that elicit Type A symptom under-reporting, research has examined person variables. Three studies by Matthews and Brunson (1979) assessed the attentional style of 104 Type A and B subjects, using a dual or simultaneous task paradigm in one study and a single task performed in the presence of a distractor in two others. The results indicated that in the dual task paradigm, Type A subjects relative to Type B's, were better on the central task, the Stroop Color Naming Task, but poorer on the secondary reaction time task to lights. It appeared that Type A's focused most of their attention on central events and allocated less attention to peripheral events. In the other experiments, Type A's outperformed Type B's in the presence of a distracting stimulus, indicating that Type A's actively inhibit or suppress attention to task-irrelevant events that might distract them from task performance. A similar rationale may have accounted for data from Strube et al (1983) that positive, soothing peripheral stimuli increased central task performance for Type B individuals but not for Type A's. Matthews and Brunson argue that Type A and B individuals differ in their allocation of attention to the environment, and use this to explain the failure among Type A's to report symptoms. They reasoned that bodily symptoms are analogous to peripheral events that might distract them from task performance, and so attention to symptoms is inhibited or suppressed to maximize task performance.

Stern, Harris and Elverum (1981) attempted to test the Matthews and Brunson findings using a paradigm in which subjects worked on two tasks sequentially, one defined as important, the other as trivial. Half of 194 subjects worked on math problems as the important task and then rated symptoms as the trivial task, while the other half were asked to rate and memorize their symptoms as the important task, and secondarily to solve problems. They found that Type A's performed better on math problems and recalled more fatigue symptoms when those tasks were deemed important, than when they were trivial. Type B's showed no such differences across manipulations, performing the same in both conditions. Stern et al argue that Type A's inhibit attention to trivial tasks, in order to perform better on important tasks. Similarly, Type A's will under-report symptoms when attention to them is considered unimportant to the task at hand. Clinically, the findings suggest that reporting behavior may be altered by changing the attention allocated to symptoms, as in changing their importance level or simply focussing subjects' attention onto their body (Pennebaker, 1978).

Pardine, Napoli, and Calicchia (1984) took the test of the attention suppression hypothesis one step further. Using the dual task paradigm, in which the peripheral or secondary task involved maintaining the learned control of somatic (E.M.G.) tension levels, Type A and B subjects simultaneously performed an anagrams task. Subjects were instructed to maintain target control "almost as an afterthought (p.5)". Type A and B subjects showed no differences in the primary task of solving anagrams,

but Type A subjects performed more poorly on the peripheral task, with significantly less time on target E.M.G. levels. Differences could not be attributed to discrepancies in the initially learned control of tension levels, as there were none, nor to the amount of distraction experienced between groups. The findings provide direct support for the notion that the Type A individual suppresses attention to bodily cues when engaged in task performance. Unfortunately Pardine et al did not include an experimental group in which somatic responses were the primary task, in order to examine whether somatic attention and control could be enhanced if deemed primary or important. This would have been a stronger test of the attention hypothesis and generated clinical implications. It is noteworthy that the Pardine et al data coincide with the findings from two recent physiological studies. Essau (1985) and Egeran et al (1985) found college Type A's to have poorer self-regulation of cardiovascular functions under stress conditions, due to poorer heart rate estimation and inattention to physiological sensations, despite greater physiological reactivity than Type B's. Together, these findings suggest that while highly physiologically reactive to stress, Type A's external focus of attention and symptom under-reporting may contribute to poorer self-regulation of cardiac functions.

Another important person variable investigated in the Type A symptom reporting research has been the role of attributional processes. Since symptoms may often be ambiguous and unfamiliar, their perception and evaluation may be biased by attribution

errors in the labels or explanations given to them (Pennebaker 1982), thus influencing their reporting. Moreover, misinterpretation of symptoms, for example as signs of anxiety rather than illness may directly influence health behavior as in the delay in seeking medical attention (Rodin, 1978).

Green et al (1974) hypothesized that Type A persons may be particularly subject to such attributional errors. Given their strong need for control Type A's may resist defining physical symptoms as signs of illness in order to avoid the helplessness and loss of control in being a patient. To date there has been relatively little research on interpretive sets and symptom reporting specifically with Type A subjects, and much of this has been inferential and indirect.

Hart (1983) for example, found that in a sample of healthy college males, Type B subjects showed a significantly negative relationship between the number of symptoms reported and their perceived health. Conversely, Type A subjects did not show this inverse relationship. Hart suggests that Type B individuals may translate increased symptomatology into poorer health status, while Type A's may label body symptoms not as indicants of illness but of anxiety or hostility. Unfortunately no measure of either was used to corroborate the suggestion.

Similarly, Somes, Garrity and Marx (1981) found that although young Type A adults reported significantly more life change in the previous year compared to Type B controls, A's reported no more health problems, illness episodes or physical symptoms than did B's. The findings replicated those of Burke.

and Weir (1980) who found Type A and B managers to report about the same number of physical symptoms, illness episodes and perceived health status, despite Type A's greater levels of measured occupational demands, stressful life events and marital dissatisfaction. The authors of both studies suggest that Type A's may interpret stress and physiological reactivity as helpful and positive, rather than disabling or symptomatic, which would account for the relative under-reporting of symptoms. However, the hypothesis requires more rigorous testing using direct attributional measures.

While not concerned directly with symptom reporting, laboratory data has found Type A and B's to markedly differ in their attributions to stress and uncontrollable failure. Type A's attributed failure to stupidity or lack of effort, while Type B's attributed failure to external factors such as task difficulty and luck (Brunson and Matthews, 1981). Similarly, Lueger and Brady (1984) found that Type A college subjects made more internal, unstable and global attributions than Type B's regarding stressful life events. Hence, Type A's appear to assume greater responsibility and control over their higher life stress and occupational demands, which may generate less perceived distress and subsequently less symptomatology for Type A's. For example, the finding noted earlier in physiological studies of a discrepancy between actual and perceived heart rate among Type A's may have been due to differences in attributional style.

In the only study to date to directly examine Type A

attribution and symptom reporting, Gastorf and Suls (1982) asked 32 recovering infarction patients, to indicate using a cardiac and general health symptom checklist, to what extent, when a symptom was present in a friend and one's self, would the friend/self be sick. They found that when asked close to discharge time, when the infarct incident was salient, Type A's perceived and reported illness symptoms in themselves similarly to Type B's. However, when the salience was less, at 180-210 days post discharge, Type A's tended to attribute symptoms as signs of illness in others but not in themselves, while Type B's attributed the same amount for self and for others. Apparently when symptoms are salient, Type A's report them, but may not do so when they become less salient, attributing them instead to non-cardiac or non-physical origins. Available data also suggests that Type A's may not resist the sick role per se, but adopt attributions consistent with their achievement orientation.

Uncontrollability has also been examined in the Type A-symptom under-reporting relationship. Experimental data has consistently found that perceived lack of control leads to heightened symptom reporting, even when the degree of success remains constant (Pennebaker et al, 1977, 1982; Matthews et al, 1980). Among Type A's, who seek and probably have more control than their Type B peers (Glass, 1977) they would be expected to report fewer physical symptoms. Pennebaker (1982) in summarizing a large amount of correlational data found that indeed Type A's had higher measured self-esteem and self-control and lower

symptom reports. However, self-control measures accounted for less than 10% of the reporting variance. One interpretation is that it is not controllability per se but attributions of control and distress that mediate symptom reporting.

To date, the experimental data has supported the original symptom suppression hypothesis. In particular, under conditions of low symptom salience, environmental challenge, low symptom importance and attentional demand focussed externally, Type A's consistently under-report physical symptoms. A major limitation to this data however is that it fails to address the critical question of whether Type A under-reporting extends beyond the laboratory to clinically meaningful, "real life" situations. It is important for heart disease research to know if symptom under-reporting is just an epiphenomena, elicited by experimental tasks, or a genuine aspect of the Type A coronary risk. By and large, the most common methodology used to address this question has been the field study examining whether healthy Type A populations under-report real, as opposed to laboratory induced symptoms. Nine studies of this type have been published, 7 using college students and 2 using managerial subjects. These are reviewed first. Seven known studies have examined symptom reporting in a clinical population of Type A coronary patients.

Matthews and Cara (1982) examined the reporting of menstrual symptoms in college women, predicting that Type A women, but not Type B's, would be motivated to ignore symptoms during menstruation, but not retrospectively or post-menstrually, in

order to avoid stereotypes about impaired functioning. They found that both positive and negative symptoms were under-reported by Type A's during menstruation relative to the other comparison intervals, while Type B's showed no such pattern. The more extreme the Type A behavior, the less intense the symptoms reported during menstruation, adjusting for retrospective and post-menstrual symptoms. These relationships were not due to Type A's reporting less symptoms than Type B's at all times nor due to A-B differences in social anxiety or self-esteem. While the sample size was small ($N = 29$) the authors suggest that Type A symptom under-reporting extends from a suppression of acute symptoms elicited by experimental tasks to chronic symptoms which are familiar.

Several less rigorous studies, primarily with college subjects have yielded inconsistent results, some finding Type A's to under-report. (Hart, 1983; Somes et al, 1981; Anchor et al, 1979; Burke and Weir, 1980) others finding no A-B differences in symptom reporting (Lundberg and Paludi, 1981; Matteson and Ivancevich, 1982;) and some finding Type A's to report more symptomatology than Type B's (Smith and Sheridan, 1983; Howard et al, 1977).

One reason for the inconsistent findings may be the large methodological differences across studies (Hart, 1983). For example, studies differed in subject and demographic characteristics such as age, gender, socio-economic class, marital and work status (college students vs. managers) all of which moderate symptom reporting behavior (Pennebaker, 1982;

Mechanic, 1983). Moreover, while most studies used the Jenkins Activity Survey to assess Type A behavior, not all did, calling into question the degree of overlap in subjects classification. More importantly, the techniques used to measure symptom reporting varied widely. Only two studies used the same symptom reporting measure. In addition some studies used a retrospective assessment, others concurrent and others still did not specify. Finally, an especially important methodological difference across studies was the degree of environmental challenge present during testing. Some studies collected data under challenging (stressful) conditions such as exam week or menstruation, others used hypothetical or ambiguous stressors. To the extent that Type A under-reporting is mediated by environmental challenges, then low challenge conditions in the field may elicit few A-B differences. Overall, the widely differing methodologies make comparisons between studies difficult.

A more important problem however in this field research has been the poor methodology within studies themselves. Sample sizes were often small and none of the studies attempted to match Type B controls with Type A's on any variable, although Matthews and Cara (1982) and Burke and Weir (1980) did use a post hoc covariance procedure on subject variables. Subject randomization or matching is particularly important as certain demographic variables, such as sex, marital status and age independently predict differences in symptom reporting (Mechanic, 1983). Many studies paid little attention to the role of environmental challenge or symptom salience. Most troublesome

however has been the measurement of the key dependent variable, symptom reporting. Several of the questionnaires used, such as the Cornell Medical Index, include psychiatric as well as physical symptomatology, which compromise the assessment of the symptom suppression hypothesis. One study (Smith and Sheridan, 1983) even used an unknown symptom inventory whose validity, reliability and standardization were unclear. While not the measure of choice, one advantage of the Cornell Medical Index or Menstrual Distress Questionnaire used by Matthews and Cara, is their scalar properties, unlike the symptom checklists that use a binary, yes-no response. The latter loses an important source of variance and may unduly skew reporting (Pennebaker, 1982). Surprisingly, none of the field studies have used Pennebaker's (1982) Inventory of Limbic Languidness, a trait measure of symptom reporting with well established psychometric properties and standardized norms. In addition many studies did not specify a time frame for symptom reporting or used retrospective measurement which is unreliable and exaggerates reports (Stunkard et al, 1985). None of the studies measured illness behaviors, such as medical utilization, absenteeism or medication use, or observer ratings to corroborate symptom self-report measures.

The few well controlled field studies with healthy adults confirm that Type A's do under-report a wide variety of chronic and acute physical symptoms in real life when motivated to do so, as when confronted by environmental challenge. Future research of this kind will need to tighten its methodology, by

using larger sample sizes, matching subjects on important demographic variables and using valid measurement methods.

Another approach to studying the symptom suppression hypothesis has been research on the symptom reporting behavior of coronary patients. This method assumes that differences between Type A and B patients' symptom reporting existed prior to illness onset and therefore may have been causal. While not as strong an inference strategy as a prospective design or intervention study, the retrospective design with a clinical sample permits one to look, in a cross-sectional manner, at continuities in behavior from health to illness, and provides ecological validity. To date, 7 studies have used this methodology.

Schleigel et al (1980) examined the symptom reporting of 40 post-myocardial infarction adult males on a laboratory exercise test and during the course of daily living. In the laboratory, subjects rated their fatigue during ergometer testing used in cardiac rehabilitation, and in the field subjects maintained a structured diary over a two week period on their symptoms, perceived stress and perceived challenge. In the lab, Type A's were found to work harder than Type B's but reported the same amount of fatigue, i.e. under-reported relative to their effort. In the field, Type A men who perceived themselves as highly challenged or stressed reported fewer cardiac symptoms than when they felt less challenged (significant inverse relationship). In contrast Type B men reported more symptoms under high, than low challenge conditions. As with healthy subjects, Type A

under-reporting in coronary patients is elicited by appropriately challenging events. Schleigel et al suggest that this may seriously compromise the vigilance to early warning signs of a heart attack, especially as Type A's engage in a chronic pattern of challenge in their daily life. Moreover, the recovering coronary patient who returns to his challenging routine may miss important symptoms or cues to alter this behavior and prevent recurrence.

In this regard the research with cardiac rehabilitation patients is particularly important. Four studies have consistently shown that Type A patients work harder than Type B's during exercise rehabilitation, but report less fatigue (Schleigel et al, 1980; Castell and Blumenthal, 1984; Oldridge, 1978; Rejeski et al, 1984). As a result, Type A's receive a more demanding exercise program from their physician (Oldridge, 1978; Rejeski et al, 1984). However, Type A's are also more likely to drop out of cardiac rehabilitation programs than Type B's (Oldridge, 1978; Rejeski et al, 1984). In addition Type A coronary patients were less likely to avoid activities that caused angina (chest pain) than Type B's (Smith et al, 1984). It is possible that while Type A's work harder when in rehabilitation programs, by suppressing exercise related fatigue, they become non-compliant after the initial impact of their symptoms has subsided (Rejeski et al, 1984). For example, Gastorf and Suls (1982) found that when symptoms were highly salient (close to time of infarction) Type A patients attributed them to cardiac origins and reported them, but at 180-210 days

post-infarct when symptom salience was low, Type A's did not report symptoms while Type B's continued to attribute and report cardiac symptoms. The under-reporting of cardiac symptoms and associated non-compliance may present a serious barrier to effective rehabilitation and a serious risk for recurrent infarction, as evidenced by the 5-fold greater risk among Type A's for re-infarction (Rosenman et al, 1976).

Type A under-reporting may also contribute to coronary risk by delaying medical treatment. Matthews et al (1983) examined the symptom reporting experience of 43 post-infarct patients and their decision to seek medical treatment finding Type A's took significantly longer than Type B's to recognize the seriousness of their symptoms and to interpret them as sign of illness, contributing to a greater time taken to seek medical attention. Type A patients also received more serious medical diagnoses, perhaps because of their inordinate delay in seeking treatment for their cardiac symptoms. The Type A delay was due to their longer illness decision making time, which was associated with their interpretation of symptoms as signs of depression or fatigue rather than cardiac problems. Once the illness decision was made however, Type A's took significantly less time to actually decide to seek medical treatment than Type B's. Hence Type A's delay in the early stages of decision making but once symptoms are attributed to illness, they become quicker to act. This mitigates against a denial or resistance to adopt sick role hypothesis of delay. The findings support laboratory work demonstrating that factors that reduce patients attention to

symptoms, i.e. low symptom salience, ambiguous symptoms misattributed to depression or anxiety, demanding work environments, also reduce symptom reporting and may increase patients delay in seeking medical treatment.

The clinical findings help put in perspective the importance of Type A's symptom under-reporting for actual health and illness behaviors and the development of coronary heart disease. Evidently, Type A's who adopt a chronic pattern of intense and challenging activity may be seriously compromising their vigilance to early and recurring warning signs of a heart attack. Anything that reduces the likelihood of Type A's attending to or interpreting their symptoms as signs of illness, delays them from seeking medical care. Both clinical and experimental data indicate that under-reporting is most likely when symptoms are low in salience or ambiguous, do not interfere with ongoing activities like work, and occur when attention is directed to challenging or more important tasks. Interestingly, once Type A's recognize their symptomatic state they are more responsive than Type B's to treatment, although they may develop poorer adherence to rehabilitation when symptoms no longer remain salient. Moreover, as Type A's under-attend to physical symptoms they are less able to use somato-visceral cues to alter or regulate behavior, such as reducing their exposure to stress.

TYPE A BEHAVIOR PATTERN IN CHILDREN

Why Study Type A behavior in Children?

While 20 years of research on the Type A behavior pattern has provided an abundance of data regarding the assessment,

psychology, and pathophysiology of the Type A pattern, little is actually known about how individuals become Type A. This represents a significant gap in our knowledge about the Type A behavior as a psychological construct, about how and when the coronary risk emerges, and about how to best modify it through risk remediation and prevention programs. Thus, a developmental approach to the study of Type A behavior may shed light on the Type A construct and form part of a coronary risk factor reduction program in the primary prevention of adult onset heart disease (Coates et al, 1981).

A primary prevention approach to CHD with children is further strengthened by the abundant evidence that the atherosclerotic process (the underlying disease process of CHD) begins in childhood (Strong, 1984) and that substantial numbers of healthy young children have a significant prevalence of identifiable CHD risk factors (Wilmore et al, 1982). Once established in adulthood risk factors require intensive educational and psychological effort at great economic cost to reverse them (Herd, 1983). Coronary risk factors related to health behaviors, which are shaped during the childhood years, become exceptionally resistant to change, and are less expensive and more likely to be prevented in childhood than reversed in adulthood (Evans, 1983). Developmental data about Type A behavior may help construct effective and economical prevention and early intervention strategies for CHD (Coates et al, 1981).

ASSESSMENT OF TYPE A BEHAVIOR IN CHILDREN

Research on Type A behavior in the young is predicated on

the accurate measurement of the behavior pattern. To date, several assessment methods have been developed.

Butensky et al (1976) provided the first published attempt to measure children's Type A behavior. They adapted a 10 question interview from the adult Structured Interview, and followed a similar administration format. Scoring was based primarily on content rather than behavior. However, no reliability or validity data were published and the scale has not appeared in other published work.

The Adolescent Structured Interview, (Siegel and Leitch, 1981) is also modelled after the adult Structured Interview, but bases behavior typing on the presence or absence of certain speech stylistics (eg. explosive speech, interruptions) rather than content, as is done with adults. The adolescent interview yields 4 classification categories; A1 (fully developed), A2 (incomplete), X (mixed) and B (absence of Type A). Normative, reliability and validity data have been reported in 2 studies, finding no gender or age differences among adolescents, high inter-rater reliability (75%), factor analytic support for Type A components, and construct validation support using independent questionnaires tapping Type A dimensions such as competitiveness, impatience and hostility (Siegel and Leitch, 1981; Siegel et al, 1981). The measure has also been related to cardiovascular reactivity, considered among adults as a major risk pathway to CHD. Available evidence indicates that with adolescents, the Adolescent Structured Interview taps the the major behavioral components of the Type A behavior pattern.

Recently another interview measure, the Miami Structured Interview (Gerace and Smith, 1985; Smith et al, 1985) has been developed. Preliminary reliability data is favourable with inter-rater agreement at 80% and test-retest agreement at 91%. However the authors did report that interviewers may be subject to assessment bias. Construct validation was assessed by comparing interview derived Type A's with another child Type A measure, the Matthews Youth Test for Health, finding significant agreement using American and Greek adolescent samples. Further validation study is required before use of the scale can be recommended.

An interview has some positive features in assessing childrens' Type A behavior such as providing appropriately challenging environments to elicit the behavior, an opportunity to observe behavior as well as obtain relevant information. However, the potential of the interview to provide a valid measure of children's Type A is seriously compromised by the immaturity of speech and comprehension and their shyness with adult strangers, especially when provoked or challenged in a formal interview (Matthews, 1982). Thus an interview may be inappropriate for a child younger than 9 or 10 years (Matthews, 1978).

In younger children 3 non-interview assessment methods have been developed. One is the Bortner performance battery (Bortner, 1967) consisting of 4 tasks that tap behaviors theoretically related to the Type A construct; writing speed (speed), time per item on an arrow dot task and embedded figures task (reaction to

frustration), arrow dot scores (achievement), and flicker-fusion scores (impulsivity). Initially, validated with adults, the Bortner battery has been used with children as young as 9, but with no reported reliability or validity data. While it has the advantage of minimizing childrens' verbal requirements and social desirability responses, it is cumbersome, time consuming, expensive, and at least one task, embedded figures, is substantially affected by intelligence.

Another child measure is the Hunter-Wolf A/B Rating scale (Wolf, Hunter and Webber, 1979), a self-report measure containing 24 items that children rate on a 7 point scale. In a sample of 5th and 6th grade children, the scale received good psychometric support. It has acceptable test-retest reliability and a factor structure consistent with the major Type A dimensions. In a construct validation study using 6 laboratory tasks to elicit component behaviors such as speed, impatience, and competitive achievement striving, Hunter-Wolf assessed Type A children outperformed Type B's in the predicted direction on 5 of 6 tasks (Wolf et al, 1982). The scale highly correlates with questionnaire measures of theoretically related constructs such as locus of control (Wolf et al, 1979) and is also related to cardiovascular risk factor variables in 10 to 17 year olds (Hunter et al, 1982). The Hunter-Wolf scale is subject to sex, age and racial differences (Wolf et al, 1982), with younger, female and black subjects scoring lower, but this may reflect differential socialization practices rather than scale deficiency (cf. Friedman and Rosenman, 1974). Available evidence

provides partial support for the validity and reliability of the Hunter-Wolf scale with children over 9 years old.

The Matthews Youth Test for Health or MYTH (Matthews and Angulo, 1980) is the most widely used and well researched Type A measure for children. The scale uses teacher ratings, rather than child self-reports, along 17 items with a 5 point scale. Content areas include competitive achievement striving, aggression-hostility and time urgency. The scale yields an overall Type A score and 2 factor derived subscale scores. The MYTH has been used with samples of children aged 4 to 14 and has received excellent psychometric support. Test-retest reliability of .83 across 3 months and .55 after one year and inter-rater reliability of .87 have been reported (Matthews and Avis, 1983; Murray et al, 1983). Gender differences in MYTH rated Type A subjects occur with elementary aged children (Matthews and Angulo, 1980) but not pre-schoolers (Murray and Bruhn, 1983), perhaps because Type A component behaviors such as aggression and competitiveness, are not sex differentiated in the very young (Macoby and Jacklin, 1974). MYTH scores do not appear to vary with age (Sweda et al, 1986), in fact correlations between repeated MYTH scores increase with age, suggesting that MYTH scores become more stable over time (Matthews and Avis, 1983). Construct validity data has been obtained for both pre-school and elementary aged children. In laboratory tasks MYTH Type A children make greater efforts to excel and to control their environment, were more aggressive and impatient, and worked harder and faster when a deadline was imposed than MYTH assessed

Type B children (Matthews and Angulo, 1980; Matthews and Volkin, 1981, Corrigan and Moskowitz, 1983). Discriminative validity is supported by data that MYTH scores predicted independent teacher ratings of aggression and impatience (Corrigan and Moskowitz, 1983) although one recent study found MYTH defined Type A's to modestly overlap with a hyperactive sample (Whalen and Henker, 1986). MYTH scores are not related to intelligence (Corrigan and Moskowitz, 1983), and are also independent of teacher rater's sex and Type A/B status (Murray and Bruhn, 1983). At least 4 studies have found MYTH scores to relate to cardiovascular reactivity, a physiological arousal pattern associated with Type A adults and their coronary proneness (Lawler et al, 1980; Schmidt et al, 1985, Lundberg 1983; Matthews and Jennings, 1984). The MYTH has a moderate degree of overlap with Bortner and Adolescent Structured Interview classification (Lawler et al, 1980; Matthews and Jennings, 1984) a finding similar to the association found among adult Type A measures. No published data is available comparing MYTH and Hunter-Wolf A/B ratings.

In evaluating available Type A measures for children one must consider both psychometric data, that is the adequacy of normative, reliability and validity data, and practical concerns. The question of establishing validity for children's Type A measures is somewhat unique. In adults, a valid Type A measure must be able to predict to criterion, that is CHD endpoints. In children however, criterion-related predictive validity is not appropriate unless one is carrying out a

long-term prospective study (Sweda et al, 1986; Matthews, 1978). While concurrent criterion such as cardiovascular reactivity may be more appropriate one is still faced with the possibility that if no association occurs, it may simply reflect that physiological reactivity occurs only after several decades of Type A behavior. Thus, construct validation, including both convergent and discriminative evidence may be most suitable for establishing validity of childrens Type A measures (Matthews, 1978; Corrigan and Moskowitz, 1983). From this perspective available evidence would suggest that the MYTH is the assessment method of choice in pre-school and school-aged children, as in the present study. It has the most normative data available having been used in multiple samples of children ranging from 4 to 14 years. By comparison, the Hunter-Wolf scale has been less widely used, and only with older children, and interview methods have been restricted to adolescents. Both construct and criterion validity and reliability for the MYTH are strong, while only partial support exists to date for the Hunter-Wolf. From a practical standpoint the MYTH is also preferable. It permits scoring of individual Type A behaviors and as a teacher rating is based on observer reports rather than self-report. The latter is especially important because Type A's are considered poor self-raters (Scherwitz et al 1978) and children, especially young children, tend to be unreliable in making self-assessments (Matthews and Angulo, 1980). In addition, observer Type A ratings in adults are better predictors of disease end-points than are self-reports. While the Hunter-Wolf is a promising

alternate to the MYTH and deserves further attention, to date it has received less empirical and practical support. Future work might expand the psychometric data base for this measure and examine its overlap with the MYTH. A recent study by Hunter et al (1985) found correlations between a teacher version of the Hunter-Wolf scale and the MYTH to range from .75 to .87. The study of overlap in classifying Type A children may suggest unique and common sources of variance in the measures.

DEVELOPMENT OF TYPE A BEHAVIOR

In one of the first developmental studies Matthews (1979) compared the behavior of Type A and B children in response to uncontrollable events. She used a laboratory paradigm in which subjects pushed a bright (high salience) or dim (low salience) button, to earn nickels on either a variable (uncontrollable) or fixed (controllable) reinforcement schedule. The author found that compared to Type B subjects, both Type A children and adults made more vigorous efforts to assert control in response to salient uncontrollable events, but made less effort to low salient uncontrollable events. The types did not differ when they had environmental control. The results suggested that Type A children, like their adult counterparts are more motivated than Type B's to assert and maintain control over their environment, and that they allocate their attention to salient events and neglect peripheral ones (Matthews and Brunson, 1977). The developmental findings are noteworthy in that they are consistent with adult data that the Type A pattern evolves as a coping style to avoid loss of environmental control (Glass,

1977) and that an associated attentional style, to attend to salient or central events and neglect peripheral or unimportant ones, abets this coping style (Matthews and Brunson, 1977).

Other features of the Type A pattern also appear to have their developmental origins early in life. For example, Type A children have been found to be more aggressive and impatient when provoked by a frustrating task (Matthews and Angulo, 1980), more competitive in interpersonal situations (Matthews and Angulo, 1980), and more explosive and rapid in their verbal behavior than Type B peers (Siegel and Leitch, 1983; Wolf et al, 1982). To the extent that these results parallel Type A adults aggressiveness and impatience (Carver and Glass, 1978), competitiveness (Scheffwitz et al, 1983) and speech stylistics (Friedman and Rosenman, 1974), it would seem these component behaviors develop early and persist throughout the life-span. It is interesting in the latter regard to note recent findings that self-rated Type A behaviors appear to peak around age 30, plateau during the 40's and decline during the 50's and 60's (Blumenthal and Herman, 1985).

Psychophysiological studies examining cardiovascular hypereactivity, or greater responsiveness to stress with Type A children have yielded complex results. Lawler et al (1981) found when using the Bortner battery to classify A/B groups, that Type A 6th graders showed higher resting levels and reactivity in systolic blood pressure and heart rate than did Type B peers. However when using the MYTH classification, greater cardiovascular reactivity was found only in Type A girls not

boys. Studies by Siegel et al (1984) and Lundberg (1983) found Type A adolescents and MYTH assessed children to have greater systolic but not diastolic blood pressure variability, compared to Type B's. One reason for these inconsistencies may be the modest overlap in Type A/B classification. Another possibility is suggested by recent work that cardiovascular hypereactivity in children is influenced by the nature of the task. Schmidt et al (1985) found that reactivity in blood pressure was greater for Type A's when competing on a video game, but was no different from B's when working on a non-competitive reaction time task. There were no A/B differences in resting blood pressure or heart rate. Similar work by Matthews and Jennings (1984) found that on competitive, frustrating and slow paced tasks, Type A children demonstrated greater blood pressure and heart rate variability than Type B's, while on mental arithmetic tasks Type A's heart rate reactivity actually decreased relative to B's. The authors suggest that Type A children autonomically over-react to environmental or task conditions that are perceived as stressful or challenging. Low salient events that are not so perceived, fail to elicit hyperresponsiveness. In any case, it is most significant that cardiovascular hypereactivity is evident in Type A children, suggesting that certain risk components associated with the mature behavior pattern emerge early in childhood.

Another Type A component that has been developmentally examined is the hard-driving achievement orientation or struggle to achieve an ever escalating but poorly defined set of goals.

In the first of a series of laboratory studies, Matthews and her colleagues (Matthews and Volkin, 1981) had male and female fourth graders perform a set of arithmetic problems with or without an explicit time deadline and found that Type A children worked harder, longer and solved more problems than did Type B's when no deadline was imposed or when performance criteria were ambiguous. When criteria were explicit, as in the presence of a time deadline, there were no A/B performance differences. A similar result was found in their second study using a physical, rather than mental task. Hence Type A children make greater efforts to excel and achieve at their limit independent of the task requirements, while Type B's only make strong achievement efforts when the task calls for same (Matthews and Volkin, 1981). In a later study Matthews and Siegel (1983) looked at the performance standards that Type A and B children used for self-evaluation. Using another laboratory task that permitted manipulation of the availability and level of external standards, they found that Type A children chose the highest standards for evaluation of their performance regardless of whether it was called for or not. In contrast, Type B children chose high standards only when no other standard was externally provided. The authors suggest that Type A children have ambiguously high self standards and that this maintains their struggle to achieve ever-escalating goals. These findings were supported by data from Murray et al (1985) who found Type A children to evaluate themselves negatively when standards were ambiguous. Similarly, Houseworth (1985) found that Type A

adolescents reported less satisfaction and lower performance ratings than Type B peers despite equivalent objective performance levels. It seems that Type A's learn early on that "good" is not "good enough".

The developmental data indicates that the Type A pattern may be accurately identified in children. More importantly, Type A children have been found to be remarkably similar to their adult counterparts along behavioral, physiological and cognitive parameters.

ETIOLOGY OF CHILDREN'S TYPE A BEHAVIOR

Given the consistency of children's Type A behavior with the adult pattern, developmental research has begun to examine the etiology of the childhood pattern. Both genetic and early socialization experiences have been studied.

A. GENETIC FACTORS

The role of genetic factors has been addressed by several twin studies. The twin method for studying inheritance relies on the fact that monozygotic twins are genetically identical whereas dizygotic twins have on average only half their genes in common. If monozygotic twins are more alike than dizygotic twins, provided environmental experiences are similar, it follows that the characteristic has an inherited component.

Using this method Roseman et al (1974) examined 93 monozygotic and 97 dizygotic middle-aged male twin pairs on the Structured Interview and the Thurstone Temperament Schedule. The latter was used to assess dimensions theoretically related to Type A (eg. activity level, impulsivity) that are known to have

a heritable component. They found no significant heritability in global Type A scores. However they did find that several of the temperament scales, activity level, impulsiveness, sociability and dominance, which correlated highly with global Type A, had significant heritability. They suggested that the Type A pattern is not genetically transmitted but, some of its components may be, and propose that Type A development is potentiated in susceptible individuals who begin life with a high energy output.

In another twin study Matthews and Krantz (1976) studied 53 sets of twin pairs and their parents on the Jenkins Activity Survey and the Thurstone scale. Despite a different A/B classification method, their results resembled those of Rosenman et al. Global Type A did not show a genetic component, while 3 of the 4 temperament scales did. Factor H of the JAS, the hard driving component, did show a moderate heritability. The results lend confidence to the conclusion that Type A does not appear to be inherited but some of its components may be.

In a re-analysis of the Rosenman et al (1974) data, Matthews et al (1984) examined the heritability of other components, specifically speech stylistics (speed, loudness and explosiveness) and potential for hostility. They found that loudness of voice and potential for hostility had a significant genetic component, and suggested that underlying both is a general temperament of hyperresponsivity to environmental challenge. They propose that the hyperresponsive temperament is genetically determined and predisposes individuals to develop

the Type A behavior pattern.

This proposal is consistent with the majority of evidence regarding the genetics of Type A behavior. That is, global Type A behavior does not appear to have a genetic component. However, it may develop in susceptible individuals who have a genetic predisposition to behave in a hyperresponsive, energetic, hard driving and aggressive manner. In other words, underlying Type A behavior may be a genetically determined temperamental substrate upon which the behavior pattern is built. This view is consistent with longitudinal data that early childhood temperament accounts for 15% to 25% of the variance in adult Type A scores (Steinberg, 1985).

B. EARLY SOCIALIZATION

As genetic factors appear to play an indirect and modest role in Type A development, attention has been directed to early familial and cultural factors. These factors, suggest Matthews et al (1984) are probably the most important of all influences in shaping Type A behavior, and work in this area has focussed on parent-child relationships, reasoning that parents may act as a model of Type A behavior for their children (Matthews, 1978).

Early research partially supported this view finding that Type A parents tended to have Type A children. Bortner et al (1969) found that Type A fathers were more likely to have Type A than Type B sons, and Matthews and Krantz (1976) found the Type A scores of male twins to be closer to those of their father than their mother, while the female twins Type A scores correlated significantly with those of their mother but not with

their father. Recently a large scale epidemiological study by Sweda et al (1986) found that components of mothers' JAS defined Type A behavior aggregated with components of childrens' MYTH behavior, with different aggregations for sons than for daughters. However fathers' Type A behavior was unrelated to both sons' and daughters' Type A behavior.

Similarity between child and parents' Type A scores does not hold when the Type A measurement technique for adult and child differs, or when the child is less than 11. Matthews et al (1977) and Copeland et al (1984) found no relationship between the J.A.S. defined Type A scores of parents and the MYTH measured scores of younger children. Whether these discrepant findings are due to the use of same/different measurement or to the age of the child (and thus the opportunity for social learning) remains unclear.

As an effort to further explore the social learning of Type A development in the context of parent-child relationships some work has examined child rearing practices and parent-child interactions. In the first such study, Matthews, Glass and Richins (1977) observed mothers assisting their sons on 3 laboratory tasks designed to elicit parental level of aspiration for the child, parental satisfaction with the child's performance, parental support and rejection. They found that compared to Type B's, Type A mothers were both more supportive and rejecting when their child failed a task and tended to hurry task performance more. Mothers of Type A children gave fewer positive than negative evaluations, encouraged achievement

and effort more, and rejected failure more than mothers of Type B children. In short, Type A mothers were more concerned about achievement, support success and reject failure. The Type A child the authors argue, is taught to strive for higher and higher goals. This view is consistent with recent findings that Type A college males perceived their parents as more critical and controlling than did Type B's (Smith and Houston, 1985). Parents of Type A boys also report being more critical and punitive than parents of Type B's (Bracke, 1985).

In order to examine whether these results in parental behavior were in fact elicited by the children's own behavior, Matthews (1977) used the above laboratory paradigm to examine separately the effects of children's Type A/B status on caregivers behavior. Using confederate Type A and B boys and mothers of same-aged boys, Matthews found that Type A children elicited more pushing to achieve and received more positive evaluation of their task performance than did Type B boys, especially by Type B caregivers. The findings suggested that Type A children may elicit the very behavior that maintains their own pattern, leading Matthews to propose that a snowballing, interaction effect between parent and child creates a self-perpetuating dynamic which propels the Type A behavior into adulthood.

Previous interaction data is limited by its restriction to male child samples. In the only study to date using both boys and girls Copeland et al (1984) found mother child interactions to vary with the sex of the child. Type A mothers were more

directive and pushing to Type A than Type B children, but only when their child was a son, not to daughters. Type B mothers were more directive to their daughters than to their sons. Like the previous studies parental behavior interacted with the child's Type A/B status, but in addition was influenced by the child's gender. The meaning of child's gender may be quite different for Type A and B mothers, and may underly the discrepancy seen later in life between male and female adults in their coronary risk.

The early socialization studies indicate that no single coherent theory of Type A development currently exists. Family aggregation studies have found inconsistent results, varying with the ages of children studied and the use of same or different techniques to assess parent and child Type A behavior. When similarities have been demonstrated between parent and child Type A scores, the correlations tend to be rather modest, accounting for a small portion of the variance. This has prompted Sweda et al (1986) to call for the direct examination of environmental variables such as parent-child interactions that may be related to Type A development.

When this environmental data is examined there is somewhat greater consistency in the findings. There is a trend that Type A parents are more directive and achievement oriented than Type B parents when their child is also Type A and male. Type A children in turn elicit from their parents and strangers more pushing and encouragement to achieve than do Type B children, which may reinforce and maintain the behavior pattern.

While these studies have been well controlled, using randomized and experimental paradigms, they are limited in having studied predominately mothers' behavior, while ignoring the important role of fathers behavior. Moreover, the generalizability of laboratory behavior to real life settings is unclear. Future work may be well served by examining father influences on Type A development in boys in light of the skewed risk for heart disease among males, and the growing evidence that one component of the behavior pattern, namely hostility and aggression which may be particularly pathogenic (Dembroski et al, 1985), has been linked to paternal behavior and male sex role development. (Biller, 1982).

STABILITY OF CHILDRENS TYPE A BEHAVIOR

As Steinberg (1986) notes, demonstrating that there are Type A children and that they may be reliably identified is one thing. Demonstrating that they grow up to be Type A adults is quite another. Two recent longitudinal studies of Type A behavior from childhood to adulthood help shed some light on this problem.

Bergman and Magnusson (1986) studied 233 Swedish children from age 13 to 27. While not initially interested in Type A behavior they did measure 4 behavioral aspects related to the construct; aggression, motor hyperactivity, overambition and over-achievement, using teacher ratings and test scores. At age 27 the same subjects were administered a Swedish version of the JAS. They found that for men, aggression, motor hyper-activity and overambition in childhood were highly related to adult Type

A behavior, with aggression and overambition accounting for most of the prediction. In fact 88% of the men who were high in both aggression and overambition at age 13 were Type A at age 27. For women, childhood variables were also highly related to adult Type A, with motor hyperactivity accounting for most of the prediction. The authors concluded that while there may be somewhat different patterns of development for boys and girls due to socialization practices, the Type A pattern shows a strong degree of stability over a 14 year period.

Steinberg (1985,1986) reported on data taken from the New York Longitudinal Study that followed 133 individuals from grade 1 through to college (age 7 to 21). Again, no standardized Type A measures were used due to the early date of the study, but from existing interview data with teachers and subjects behavioral variables were coded in comparable fashion to the MYTH (for children) and the Structured Interview (for adults). Type A factor scores were very similar to standardized measures, yielding a prosocial dimension called achievement striving and an antisocial dimension of impatience-aggression. The correlation between these dimensions was found to shift with age, from significantly positive during childhood, to unrelated during adolescence to significantly negative in young adulthood. Steinberg suggests that the Type A components become increasingly differentiated over the lifespan, supporting other views (Matthews, 1982) that the Type A individual may demonstrate one component behavior but not others. The stability of Type A behavior was strong from adolescence to adulthood for

both males and females. However, it did not show stability from childhood to adulthood. While the structure of the behavior pattern in children resembled that in adults, individual differences in the behavior pattern did not remain stable. Measures of childhood temperament though, did predict adult Type A behavior (Steinberg, 1985).

The longitudinal data suggests that type A behavior shows a high degree of stability over the lifespan, particularly from adolescence to adulthood. While childhood Type A behavior did not predict adult Type A, childhood temperament does. It is not yet possible to determine whether methodology problems, such as non-standardized assessment or different data sources (teacher versus self-report) for child and adult assessment accounted for the lack of continuity in the Steinberg (1986) study. This must await further research. It is clear though that the underpinnings of adult Type A behavior are present early in childhood, and become increasingly predictive as one moves up the lifespan. Type A behavior may stabilize in middle childhood or early adolescence and become increasingly stable over time. Certainly from adolescence, the behavior pattern appears to remain exceptionally stable. The next step for longitudinal research would appear to be a study to determine the relationship between childhood Type A behavior and adult heart disease. The etiologic value of Type A classification during childhood has not yet been examined in this way.

DEVELOPMENT OF SYMPTOM REPORTING BEHAVIOR

In light of the importance of symptom reporting behavior as

a mediator of Type A coronary risk, it would be helpful to know more about the development of this behavior and its origins in Type A children. Regardless of whether the behavior pattern itself persists over time, the associated health behaviors and outcomes of childhood Type A are of particular importance. Yet in contrast to the growing amount of data concerning symptom reporting among Type A adults, little is known about how these individuals actually become symptom under-reporters. This represents a significant gap in our knowledge about the Type A construct, and about the development and prevention of risk behaviors. For example, it is unclear whether the symptom under-reporting risk is continuous across the lifespan and begins in childhood, and when the Type A pattern becomes associated with coronary prone components.

Developmental study of symptom reporting in Type A children has been neglected in part because of the generally limited attention paid to developmental issues in health psychology (Maddux et al, 1986), and specifically to the determinants of children's symptom reporting. Only a handful of studies have examined children's symptom reporting and but one has used a sample of Type A children.

Gochman (1971) examined children's willingness to report symptoms and utilize medical services using an interview methodology. He found that children who perceived themselves as vulnerable and under less internal control were more willing to report symptoms and use health services. Lewis and Lewis (1982) found, using an adult free, child-initiated system of health

delivery in a school, that 8% to 12% of the children made over 50% of the visits, with highest symptom reporting among girls, first borns, and lower socio-economic subjects. Pennebaker (1982) and Campion and Gabriel (1985) have also found only children and first borns to report more symptoms. Indirect measures of childrens' symptom reporting such as school absences and medication use have been related to teacher and parents rating of child's low sociability (Pennebaker et al, 1982), mothers anxiety (Campion and Gabriel, 1985), mothers attitude to physician and medication (Maiman et al, 1986) and parental discipline use (Pratt, 1975).

The most extensive work in the area has been the 16 year longitudinal study of 350 mother-child dyads by Mechanic (1980, 1983). At year one, he found that the best predictors of childrens' symptom reports were mothers' self-reported personal problems and her own symptomatology. At year 16 the best predictors were the number of days missed from school as a child, mothers' personal problems, the number of illness episodes in childhood and child ratings of parental health concern. Thirty-nine percent of year 16 reporting was due to parental health concern which Mechanic argues forces the child's attention on to his own symptoms and reinforces body monitoring. He argues that actual illness episodes are less important in symptom reporting development than socializing, a pattern of bodily attention. Similar findings were reported by Maiman et al (1986) that 36% of medication use for childrens' symptoms was due to mothers' health attitudes, and by Hamell

(1985) that parental introspectiveness predicted both child introspectiveness and child symptom-reporting.

Several complimentary hypotheses regarding symptom reporting development have been generated by these authors. The attentiveness hypothesis suggests that parental attention and over-concern socializes the child to monitor internal states and rewards symptom reporting through negative reinforcement (letting the child stay home from school, Mechanic, 1980, 1983; Hamell, 1985). As evidence the authors use their own data and findings that symptom reporting increases among first borns and only children, who presumably receive more attention. The parental interpretation or attribution hypothesis suggests that children adopt the definitions and interpretations of their parents for ambiguous sensations through imitation and social learning (Mechanic, 1983; Leventhal, 1980; Pennebaker, 1982). For example, high reporting mothers have high reporting children (Mechanic, 1980) and childrens' illness attributions are similar to their mothers' (Cambell, 1978). A third view is that reporting is maintained by reinforcement and secondary gain (Pennebaker, 1982), as evidenced by reporting predicted by school absence. Loosely tied to this is the hypothesis that symptom reporting may reflect psychological distress or a means to an end. To date however, none of these hypotheses have been directly tested using experimental designs. Moreover, alternate explanations, for example that parental concern increases stress and subsequent child symptomatology, have not been examined.

Only one study has examined the relationship between

childrens' symptom reporting and Type A behavior. Matthews and Volkin (1981) used a dual task paradigm similar to adult research with 21 male sixth grade boys. Subjects were instructed to rate fatigue from holding a weight (matched to hand strength) while simultaneously rating the attractiveness of slides. They found that Type A's held the weight 50% longer than Type B's, but reported significantly less subjective fatigue during comparative time intervals. Differences were not due to baseline fatigue levels. The study provides evidence for the first time that Type A children in a laboratory analogue situation, are similar to their adult counterparts and under-report physical symptomatology relative to their Type B peers and relative to the effort they exert. The study indicated that some components of the Type A pattern associated with coronary risk in adults, may emerge early in life.

The developmental data on the determinants of symptom reporting behavior, both generally and for Type A children, are limited primarily by their paucity. In addition, only one study has used a prospective design, and many have relied on indirect measures of reporting behavior. While the lone study with Type A children was methodologically sound, its generalizability is rather limited. The study was conducted only with boys, used a small sample size and a narrow age range. Furthermore, the study did not address the crucial question of whether the under-reporting phenomenon associated with the Type A pattern occurs with clinical symptoms of real life illness in children, in addition to laboratory induced symptoms that may be perceived

as ambiguous or unimportant. Hence, it remains unclear whether the Type A symptom under-reporting phenomenon does indeed emerge in childhood, and if so, when it does and how pervasive it is. Further study of symptom reporting behavior in Type A children is therefore needed to answer these important concerns. —

RATIONALE AND HYPOTHESES FOR THE STUDY

Research in the area of Type A behavior has established that one mechanism by which the behavior pattern exerts its coronary influence is through the suppression of attention to physical symptoms. The symptom suppression, or symptom under-reporting hypothesis as it is called, mediates coronary risk by compromising the Type A's vigilance to early and recurring warning signs of coronary disease, delaying their decision to seek medical treatment, and impairing the individuals ability to use physical symptoms as cues to reduce stress-inducing behaviors or comply with cardiac rehabilitation. Symptom under-reporting may contribute to the exacerbation of symptoms and any underlying disease, and to coronary mortality and recurrence risk.

In contrast to the accumulated data on the adult correlates of the phenomenon, little is known about how the Type A individual actually becomes a symptom under-reporter. There is a growing body of literature demonstrating the Type A pattern emerges early in life and can be readily identified in children, and is relatively stable over the lifespan. Yet, virtually no data exist on the childhood determinants of the Type A symptom under-reporting risk. This represents a significant gap in our knowledge about the Type A construct and about how to effectively and economically remediate and prevent coronary heart disease.

There is a general lack of knowledge about children's

symptom reporting behavior, and only one study has examined it in Type A children. However, it has rather limited generalizability, having studied only boys, from a narrow age range, using a small sample size. In addition the study has not been replicated. More importantly, the study only used a laboratory analogue situation, leaving unanswered the critical question of whether Type A children, like their adult counterparts, under-report real, clinical symptoms as well as ambiguous, laboratory induced ones.

The main purpose of this study is to address these questions by examining the symptom reporting behavior of male and female Type A children across age groups, in both a clinical and laboratory situation. A second purpose is to examine the relationship of symptom reporting in children to other health related variables, such as parent's symptom reporting and Type A behavior, health locus of control, and symptom salience. The study will attempt to redress methodological problems of previous research by utilizing a valid and concurrent symptom reporting measurement strategy, using both self and observer ratings and corroborating illness behavior measures, such as medication use and school attendance, and by matching subjects.

As the study has little precedent it is considered exploratory, with implications in several areas. Theoretically, it will help address whether there are continuities in Type A symptom under-reporting across the lifespan, and whether the under-reporting risk on clinical symptoms emerges during childhood and can be identified. In other words, does the Type

A pattern have coronary prone aspects in children? This may point to particular strategies in the remediation and prevention of coronary heart disease. In addition, the study may extend knowledge about the influences on children's symptom reporting behavior and therefore how to best alter it. Finally, the results may have more general implications for the treatment of children with illnesses whose symptoms are important cues for compliance and self-regulation.

The study is concerned with 4 major research questions.

1. Do Type A children like their adult counterparts, under-report physical symptomatology compared to Type B children?
2. Are there differences in symptom reporting between Type A and B children based on the type of symptomatology?
3. Are there differences in symptom reporting between Type A and B children based on age?
4. Is Type A children's symptom reporting related to other health or illness behaviors?

Hypotheses

1. Type A subjects will report less clinical symptomatology than Type B subjects on the Symptom Inventory Rating Scale (Child and Adult)
2. Type A subjects will report less fatigue than Type B subjects on the laboratory induction task.
3. Older Type A subjects (9-12 years) compared to younger Type A subjects (5-8 years) will report fewer clinical symptoms on the Symptom Inventory Rating Scale (Child and Adult) and

less fatigue on the laboratory task.

4. Type A subjects will demonstrate larger under-reporting differences from Type B subjects when clinical symptoms are of low salience compared to high salience i.e. at 7 days post surgery compared to 1 day post surgery.
5. Subjects with Type A and under-reporting parents will report the fewest clinical symptoms and least fatigue compared to other subjects.
6. Internal health locus of control will be highest among low symptom reporters.

CHAPTER 2
METHODOLOGY

Subjects

The subjects of this study consisted of 122 male and female children, aged 5 to 14 who underwent tonsil and adenoid (T & A) surgery at the Childrens Hospital of Eastern Ontario between January 1985 and January 1986. T & A surgery is performed on a brief stay basis, and on average, children spend only one night in hospital. (C.H.E.O. 1984). It is considered elective surgery, and its sequelae are ideal for the study of symptom reporting behavior in children for several reasons. Firstly, the symptoms are clinically meaningful. They are indicative of real and important physical changes that have occurred in the body. The symptoms signal underlying tissue damage, which require some level of health care and alteration in the child's activity in order to achieve rapid and full recovery. Thus, they are important body signals, in contrast to laboratory induced symptoms that are ambiguous and may lack functional meaning. Secondly, post-surgical symptomatology is well circumscribed. It has an identifiable onset, is normally acute, and clearly unambiguous. Finally, the effects are readily observable by others for confirmation. In addition, T & A surgery is performed when the child is healthy, that is symptom-free, so that pre-surgery baseline levels of symptomatology across subjects may be considered equivalent.

Subject selection was based on the following inclusion and exclusion criteria.

Inclusion Criteria:

1. The child had been seen by a member of the Department of Otolaryngology at the Childrens Hospital of Eastern Ontario between September 1984 and January 1986.
2. The child had received tonsil and adenoid surgery between January 1985 and January 1986 at the same hospital.
3. The child was between 5 and 14 years of age as of September 1984.
4. The child regularly attended elementary or pre-school classes, as of September 1984.
5. The child was in good non-tonsil/adenoid health.
6. The child received general anaesthesia for surgery.

Exclusion Criteria:

1. The child had been hospitalized in the past 6 months.
2. The child had a history of psychiatric disturbance.
3. There had been parental separation/divorce in the past 6 months.
4. There were post-surgical complications.
5. The child was receiving concurrent medical treatment for another disorder.

Measures

1. Matthews Youth Test for Health - Form O (MYTH; Appendix A)

The MYTH (Matthews and Angulo, 1980) is the best researched and widely used measure of Type A behavior in children. It taps the three dimensions of the Type A construct; competitive achievement striving, impatience and hostility. The child's teacher rates how characteristic each item is of a child's

behavior, on a 1 (extremely uncharacteristic) to 5 (extremely characteristic) scale. The MYTH has several advantages over other available child measures. Firstly it has strong psychometric support, with well established normative, validity and reliability data. The MYTH was standardized on a sample of 500 elementary school aged children, achieving a test-retest reliability of .83 (across 3 months) and an internal consistency coefficient of .90 (Matthews and Angulo, 1980). MYTH ratings are moderately stable over time ($r=.55$ over a 1 year period) becoming more so at older ages (Matthews and Avis, 1983). This stability is comparable to that found for adult Type A behavior and for other coronary risk factors. The MYTH has also been used with both pre-school and elementary aged children, yielding good test-retest reliability ($r=.87$) across 3 months (Murray and Bruhn, 1983). Validation studies with the MYTH have consistently yielded 2 orthogonal factors labelled competitive achievement striving, and impatience-hostility and aggression (Matthews and Avis, 1983; Murray and Bruhn, 1983; Matthews et al, 1983). Construct validity has been well established from several laboratory studies finding MYTH Type A children to be more aggressive and competitive when challenged by a task, to make greater efforts to excel, and to be more reactive in cardiovascular responses to stress (Matthews and Angulo, 1980; Corrigan and Moskowitz, 1983). A second advantage to the MYTH is that it relies on teacher ratings rather than interview assessments or self reports. Child interviews are costly and time consuming, they may tend to be unreliable

because of children's immature speech and verbal behavior, and they may tend to generate withdrawal or shyness with adult strangers, particularly when provoked or challenged (Matthews and Angulo, 1980). Type A assessment is also better made by an external observer rather than self-report, in terms of its predictive power for CHD (Jenkins et al, 1971) and reliability (Matthews and Siegel 1982). Thirdly, the MYTH permits scoring of individual Type A behaviors while other techniques do not. This is important because individual behaviors may emerge at different times or become stable at different ages (Matthews and Siegel, 1982), and because certain Type A behavior components like hostility, are more predictive of CHD than others (Matthews et al, 1977). Classification of Type A/B using the MYTH tends to vary across studies, and is therefore treated in the Results section.

2. Jenkins Activity Survey - Form C (JAS; Appendix B)

The JAS has become the most commonly used method of assessing the Type A pattern in adults and was used to assess the Type A/B of parents in the study. It was originally designed by Jenkins and his colleagues (1971, 1974) to provide an objective and standardized measure in place of the more clinical Structured Interview procedure of Friedman and Rosenman (1974). The JAS is a brief, easy to administer and economical 52 item self report questionnaire that generates an overall Type A score and 3 factor analytically derived subscales; hard-driving, speed and impatience, and job involvement. The validity and reliability of the JAS has been

well established. It has high test-retest reliability averaging around .80 (Johnson and Shaper, 1982), is internally consistent (Jenkins et al, 1974) and is stable over time (Glass, 1977). Furthermore, it has been prospectively related to the incidence and prevalence of CHD (Jenkins et al, 1971), is associated with cardiovascular reactivity to stressors (Dembroski et al, 1978) and has a fair degree of overlap with the Structured Interview (Jenkins et al, 1974). Classification of Type A/B in adults using the JAS may use the available standardized norms. This is preferable to a median or percentile split procedure as it increases comparability across studies.

3. Symptom Inventory Rating Scale - Child. (SIRS-C, Appendix C)

The SIRS was developed specifically for the purposes of this study to assess children's symptom reporting behavior, that is the tendency to report physical symptoms.

Rationale for the Development of the Scale.

The decision to develop a scale specific to T & A surgery, rather than use available symptom report scales was based on 2 factors. First, the published symptom rating scales that are available such as the Cornell Medical Index, the Hopkins Symptom Checklist, the Pennebaker Inventory, or the Symptom Checklist-90, are all too general for the purposes of this study, having been designed to assess general health status across all organ systems. They would simply not be a sensitive enough measure to assess the specific symptomatology that occurs following T & A surgery. Secondly, available symptom report scales have all been developed for use with adults.

There are no known symptom report scales developed for a child population. Thus the lack of validated measures of childrens' symptomatology that was appropriate for measuring post T & A surgical symptoms led to the development of the SIRS.

Design and Development of the Scale.

The methodology used to develop the scale closely paralleled that used to develop measures of childrens pain, due to the overlap in the nature of the symptomatology and in the conceptual similarity of measuring a psychophysiological phenomenon through self-report. The format chosen for the SIRS was an inventory of symptoms which children must rate according to the presence and severity of occurrence using a 5 point rating scale. Each numeric rating is also given a verbal descriptor that acts as an anchor or referent, such that 1 = no, 2 = a little bit, 3 = some, 4 = a lot, 5 = really bad. The choice of a combined numerical/verbal category rating scale was based on several issues. Firstly, it permits usage by a wide age range of children, including young children as in the present study. Data from research in the area of childrens' pain measurement indicates that subjects as young as 4 years of age are able to reliably use a numerical scale and the verbal categories are useable by children over 7 (McGrath et al, 1985; Beales, 1982). Pilot testing indicated that the 5 point scale was easily understood and appropriately used by children as young as 5 with a minimal amount of instruction. Secondly, the numeric/verbal scale is preferable to available alternatives such as faces scales or visual analogue scales. The former

usually consist of 5 to 7 facial expressions ranging from total misery to apparent laughter from which the child must choose the one that most closely represents their current state. The problem with this alternative is that there is no agreed upon standard among the various scales (McGrath et al, 1985). The visual analogue scale while used widely in assessing childrens' pain (McGrath et al, 1985) has not been used in assessing other physical states such as post-surgical symptomatology. Moreover, recent research suggests that the visual analogue scale may be best suited for assessing change in symptom severity rather than severity level itself (Beales, 1982; Lundberg, 1980). Hence it may be more effective for comparing scores within the same child over time rather than comparing between child differences, as in this study.

On the other hand, category rating scales for self-reported symptomatology have received strong support among adult populations (Pennebaker, 1982) and are easily adapted for use with children. It is easily and quickly administered with minimal instructions and scored easily (Chapman et al, 1985). The numeric and verbal anchors such as in the SIRS, facilitate use by young and old children. Moreover, the use of a 5 point rather than binary, yes-no, rating introduces greater variability into sample scores.

The SIRS consists of 50 items that include post-surgical symptoms specific to T & A surgery as well as general medical symptomatology. Item selection was initially based on a content analysis of medical records of previous T & A surgery patients.

Thereafter items were reviewed for medical and clinical relevance by 2 pediatric otolaryngologists, a nurse and a child psychologist. A revised inventory of symptoms was generated that was easy to understand by young children, easily operationalized and observable, and occurred with sufficient frequency in the normal recovery process. Pilot testing in a non-patient sample of 5 to 14 year olds confirmed that the scale and the items were easily understood and completed. Similar findings occurred post-operatively with a same-aged patient sample of 20 pilot children (undergoing T & A surgery).
Validation of the Scale.

Validation of the SIRS was determined in two stages, the first using the small pilot sample of 20 surgical patients, and the second using data from the larger study sample. The later results are presented at the end of the Discussion section.

Pilot testing consisted of administering the SIRS to 20 patients, aged 5 to 14 at four times; one day prior to T & A surgery, immediately (5 to 8 hours) after surgery (Post 1), one day (29 to 32 hours) post-surgery (Post 2), and one week post-surgery (Post 3). Children were also given a visual analogue scale requiring them to rate how they felt along a continuum from "the worst I've ever felt" to "no problems, the best I've ever felt", following each completion of the SIRS. Parents also completed the SIRS at Post 2 and Post 3. These procedures provided preliminary construct validation data by comparing the scale with another symptom report measure and by measuring changes in the scale due to surgery and recovery, as

well as test-retest and inter-rater reliabilities.

Changes in SIRS scores over time revealed it was sensitive to the effects of surgery (pre-post 1 $t(18)=4.63$; $p<.001$) and the recovery process (post 1-post 3 $t(18)=4.8$; $p<.001$, and pre-post 3 $t(18)=1.79$; $p>.05$). Thus there is support for the SIRS ability to detect changes in symptomatology as a result of surgery and recovery. Correlations between the SIRS and the visual analogue scale were all significant, ranging from .52 at pre-surgery to .70 at Post 2. providing good construct validity evidence that symptom reports are associated with subjective distress. Correlations between SIRS ratings over time were significant and modestly high ranging from .54 (post 1 to post 3) to .61 (pre-surgery to post 1) and provided good construct validity evidence that the SIRS taps reporting tendencies, not simply symptom levels. The correlations also support the test-retest reliability of the scale, with ratings staying moderately stable over time. Inter-rater reliability between parents and children was also acceptable at .83 .

Hence there was preliminary construct validity and reliability support for the use of the SIRS as a measure of childrens' tendency to report physical symptoms.

Administration and Scoring of the Scale.

The SIRS is self-administered except when childrens' reading skill is limited as in the young subject. In this case parents are permitted to help the child but are instructed to limit their assistance solely to reading the item and defining it when necessary. Children are instructed to record their own

responses. In the case where this is not possible, eg. severe discomfort, limited writing skill, the parent may record the child's response. In this study, the SIRS was scored simply by summing the ratings made on the 50 symptoms.

4. Symptom Inventory Rating Scale - Parent Form (SIRS-P, Appendix D)

In this study the identical scale as outlined above will also be completed by the child's parent in order to provide an independent observer rating of the child's symptom reporting. While nurse ratings may provide greater objectivity they are impractical on busy surgical wards, and cannot be done outside of the hospital. Moreover as parents spend more time with the child they have access to a larger sample of behavior, thus enhancing the veracity of their ratings. Parents were instructed to complete the SIRS before they assist their child and to not discuss their ratings with the child.

5. Pennebaker Inventory of Limbic Languidness (PILL; Appendix E.)

The PILL (Pennebaker et al, 1977) is a 54 item symptom inventory that was used to assess the symptom reporting behavior of parents in the study. The scale requires subjects to rate the frequency of occurrence of common physical symptoms along a 5 point scale ranging from 1 = have never or almost never experienced the symptom to 5 = more than once a week. The PILL is considered a ~~valid~~ valid measure. Of the available symptom inventories, the PILL has several advantages. The first is that it has normative data with well established psychometric

properties. It is internally consistent (Cronbach alpha = .91), has good test-retest reliability ($r = .83$ over 2 months), and good item reliability ($r = .72$, averaging across all symptoms after testing 2 weeks apart). Cross validation with other symptom inventories is moderately high, and construct validation studies indicate that high PILL scorers engage in more health-related behaviors than people who do not report many symptoms. Other advantages are that the PILL is easy to administer compared to some of the long, time consuming inventories, is restricted to physical symptomatology, has an appropriate time frame, uses a 5 point rather than binary yes-no scale, and emphasizes common symptoms rather than infrequent or primarily severe ones (Pennebaker, 1982). Scoring of the PILL consists simply of summing the ratings made on all 54 items.

6. Children's Health Locus of Control (CHLC; Appendix F.)

The Children's Health Locus of Control Scale (Parcel and Meyer, 1978) was used to measure children's expectancy for control of health related events. It may be used to measure an individual's estimate of the likelihood that a given action will result in a particular goal, and to a lesser extent the belief or value placed by a child on that goal (Maiman and Becker, 1974; Parcel and Meyer, 1978). Although there are scales to measure children's generalized locus of control, it is believed that specific scales provide better predictors of specific categories of behaviors (Rotter, 1975). Among adults, health locus of control measures are more congruent with health

behaviors than general internal-external measures (Rotter, 1975). The CHLC was modelled after the Health Locus of Control measure for adults, and taps 3 dimensions of control; internal, chance and powerful others. Factor analytic studies with the CHLC confirm 3 consistent factors corresponding to the major dimensions. The CHLC is internally consistent ($r = .75$) and has acceptable test-retest reliability ($r = .63$, which is comparable to other child measures of locus of control). Construct validity data confirms that the CHLC is related to locus of control insofar as it significantly related to the Nowicki-Strickland children's locus of control measure. Standardization data found no male female differences in CHLC scores, but that internal locus of health control does appear to increase with age. The CHLC generates a total score and 3 factor derived sub-scores. For the purposes of this study only total scores were used. The higher this score the more internal the health locus of control, and conversely, the lower the score the more external is the health locus of control.

7. Children's Illness Behaviors

Measures of children's illness behaviors include: 1, number of days missed from school following medical procedure; 2, number of days missed from school prior to surgery; 3, number of aspirin (analgesics) taken following the medical procedure.

8. Demographic Variables

Demographic variables of interest are child's birth order, number of siblings, socio-economic status of parents (education and income) and number of previous hospitalizations or

surgeries.

Procedure

All subjects participating in the study were assessed under two symptom induction conditions: a laboratory procedure designed to induce fatigue, and a clinical condition, where subjects were assessed following surgery to remove tonsils and adenoids. In cooperation with the Division of Otolaryngology (Ear, Nose and Throat), Childrens' Hospital of Eastern Ontario, children referred for consultation regarding tonsil and adenoid (T & A) surgery were invited through their parents, to participate in a study to examine "the relationship between behavioral factors and childrens' experience with physical symptoms (see Informed Consent, Appendix G) following a briefing on the purpose of the study. Participation in the study was voluntary and entirely non-contingent for receiving medical services.

Children were seen approximately one week prior to their hospital admission, following their pre-surgery orientation. At that time parents and children were re-acquainted with the study and instructed in the use of the SIRS. A practice administration using 3 to 5 symptom items was done with parent and child to familiarize them further with the procedure. The child and parent was next seen shortly after surgery in order to deliver the package of SIRS questionnaires and to inform them when they were to be completed, and to initiate the first self-report by the child.

The SIRS' were independently completed by the child and

parent at several times. This time-sampling method was used in order to examine the effects of symptom salience and environmental challenge on symptom reporting, and to provide validity and reliability data on the SIRS. Children completed the SIRS at 3 time points: in hospital immediately (5 to 8 hours) after surgery; at home on evening of the first day after surgery (29 to 32 hours); and at home on the evening of the 7th day after surgery. Parents completed their ratings of the child at two time points: 1 day (29 to 32 hours) and 7 days post-surgery, again in the evening. Parents completed their ratings prior to supervising their child, under strict instructions that their ratings should be independent of the child's. Parents were also instructed to record the number of analgesics used by the child at the end of each SIRS rating. One week following the child's discharge from hospital, the experimenter telephoned each parent to remind them of the last rating. Questionnaires were also pre-dated to remind subjects and parents when they were to be completed, and were returned to the examiner in a pre-stamped, self-addressed envelope.

Forms for teacher ratings on the MYTH and school absence data were sent to each child's teacher, who returned these directly to the examiner. In addition to completing a MYTH on the patient, each teacher was requested to complete a second MYTH on a same-age, same-sex classmate whose surname was closest alphabetically to the patient's. This procedure was used in order to compare the Type A/B scores of the surgical patients with the general population.

Approximately one month after surgery subjects returned to the hospital for the second phase of the study, the laboratory symptom induction task. Their appointment coincided with the child's post-surgery follow-up appointment in the Ear, Nose and Throat clinic, in order to increase compliance with the study. All laboratory testing was done in a small office of that clinic. While waiting for their child, parents completed the JAS, PILL, and a demographic questionnaire.

The laboratory procedure uses a dual task paradigm in which symptoms of fatigue are induced by requiring the child to hold a weight while simultaneously completing a visual rating task. This paradigm has been used in symptom reporting research in order to prevent boredom in subjects and to study the role of cognitive and attentional processes. The laboratory procedure is included in the study for two reasons. Firstly, it provides a measure of construct validity of the clinical symptom report scale, and secondly, it permits a comparison of Type A and B children's symptom reporting on a different type of symptom, namely one that is ambiguous and benign in contrast to clinical symptoms experienced following the T and A surgery.

The laboratory procedure is taken from the Matthews and Volkin (1981) study and was used in the present study without alteration.

Upon meeting with the examiner, who was blind to the child's Type A/B assessment, the child was reacquainted with the purpose of the study, and accompanied to the experimental room. There the child was given a brief description of what was in

the room and what was about to follow. Then the subjects were asked to squeeze a hand dynamometer as hard as they could three separate times. After doing so, the subjects rested for two minutes, and completed the CHLC scale. During that time, the experimenter filled a small container with lead shot, the amount of which was .24 times the subjects highest dynamometer reading. This procedure equates the widely varying strength of the subjects (DeVries, 1968). The experimenter next demonstrated the correct way to hold the weight (elbow bent in 90 degree angle at waistline slightly away from body, container in hand, palm upward), and how to complete the fatigue ratings. The experimenter indicated "In order for me to know how tiring this is for you, I'm going to ask you every minute or so how tired you are. The way you will do this is by choosing a number on this chart that shows exactly how you are feeling at this moment" The chart consisted of a 7 point scale ranging from 7 "very energetic, not all tired", to 1, "very tired, not at all energetic". All subjects were also asked to rate the attractiveness of a series of simultaneously projected slides depicting science fiction scenes and landscapes. This procedure is used in order to prevent boredom with holding the weight. The ratings of the slides were made every half minute, and the fatigue rating every minute.

After a baseline rating of the subjects fatigue was obtained, subjects were informed that they would be told when to stop by the experimenter, but if they could not continue, they could put the weight down. In fact, subjects were not told

when to stop. However, if after 15 minutes the child had not put the weight down, the experimenter then terminated the task. Following these instructions the subjects picked up the weight, and the experimenter recorded the fatigue rating at each minute and the time to putting the weight down. At that point subjects completed the following questions: "How hard did you try? How well did you think you did? Do you believe you could have done better at some other time?" Finally subjects were asked their opinions of the procedures, fully debriefed, and thanked for their participation with a small certificate.

The scoring procedure for the laboratory task used the time taken by subjects to report the maximum fatigue level of "1; very tired, not at all energetic". This fatigue measure is called "Time to 1". The procedure differs from that used by Matthews and Volkin (1981), who used the subjects average fatigue rating over the last three time intervals. This measure is also included in the current study in order to permit a comparison of results. The "Time to 1" score is this study's primary measure as it is considered preferable for two reasons. First of all, it is a more sensitive measure, taking into account both intensity and rate of fatigue, the later providing a measure of severity, while the Matthews and Volkin measure taps only intensity. Secondly, in providing a score measured in time, permits an analysis of the stages of symptom reporting, and allows comparisons to similar work with adults.

Data Analysis

The study is based on a 2 (Group) X 2 (Age) X 2 (Sex) factorial design. All data are of an interval nature, allowing rank ordering as well as differentiation in constant units of measurement permitting the use of parametric analyses (Keppel, 1982). The populations from which the samples were drawn were assumed to be normally distributed with respect to the variables under consideration. Homogeneity of variance was also assumed since data indicating that even sizeable differences among the variances does not distort the F distribution seriously (Keppel, 1982). All observations were independent in accord with randomization requirements.

Hypotheses related to clinical symptom reporting were tested using a 2 x 2 X 2 Analysis of Variance procedure with repeated measures. The BMDP-P2V statistical program was used for this purpose. Since repeated measures designs are prone to greater distortions in the F distribution if homogeneity violations occur, the study used the highly conservative Geisser-Greenhouse correction on all clinical ANOVAs. This procedure performs the usual ANOVA but evaluates F ratios against a critical value that assumes maximum heterogeneity. While this procedure tends to over-correct, it also provides protection against Type I errors, reducing the number of null hypotheses falsely rejected (Keppel, 1982). The latter protection is also helpful in studies such as this, where multiple comparisons are made and may lead, if uncorrected to an increase in Type I errors. A 2 X 2 X 2 Analysis of Variance

model was used to examine symptom reporting differences on the laboratory task. In order to examine which variables best predicted symptom reporting behavior both in the laboratory and clinically, a Multiple Regression procedure was carried out. All correlation procedures refer to Pearson product-moment correlations. The Statistical Package for the Social Sciences was used for these analyses.

Significance Levels

The significance level was set at .05 for all analyses in order to both reduce the Type I error risk and to be of practical importance. As the current study was exploratory and was interested in breaking new ground, obtained alphas in the range between .05 and .08 were treated as a statistical trend for discussion purposes. This decision was based on the desire to generate hypotheses for future research and to facilitate new ideas and discussion.

Sample Size

Sample size determination permits the reduction of Type II error risk and insures the study has adequate statistical power. In order to determine the sample size for the study the statistical approach of Cohen (1977) was used as it provides the most comprehensive treatment available on sample size and power analysis.

Using this method the investigator must draw upon previous findings and theory, as well as scientific judgement, to

specify the means and their distribution, the expected population variance and in turn the resulting effect size statistic. When the population variance is not known, as was the case in the present study, the effect size must be based on practical and clinical considerations. The only point of comparison to previous research was the study by Matthews and Volkin (1981) who obtained a $t(18)=2.67$, $p.<.03$, which corresponded to a "small" effect size (Cohen, 1977) and a power value equal to .39. Cohen (1977) recommends that a power value of .80 be used. This convention was followed in the present study, and when combined with an alpha level of .05 balances the risk of Type I and Type II errors to $(1-.80)/.05$ or 4 times as serious to make a Type I than Type II error. This is consistent with the goals of the present research. It was assumed that with an adequate power value of .80 a medium effect size ($f=.30$) would provide a strong test of the hypotheses. The N to detect such an effect with alpha equal to .05 is 45 subjects per group.

CHAPTER 3

RESULTS

Subject Information

Data from 122 children were collected, of which 56 (45.9%) were boys and 66 (54.0%) were girls. Subjects ranged in age from 5 years 2 months to 14 years 6 months ($M = 8$ years 1 month). For purposes of data analysis subjects were divided into younger (5 to 8 years) and older (9 to 14 years) groups, of which 78 (63.9%) were in the younger group and 44 (36.1%) were older. Subjects had a mean of $1.2 \pm .7$ siblings and a mean of $.6 \pm 1.0$ previous hospitalizations.

Operational Definition of Type A Behavior for Children.

Throughout the study Type A and Type B children are defined as subjects whose score on the Matthews Youth Test for Health (MYTH) falls within the upper and lower 30th percentile respectively of the entire sample. Using this criterion for grouping Type A and B subjects creates a sample of 43 Type A subjects, 42 Type B subjects and 37 subjects whose scores are out of range. The use of 30 percentile cutting scores was based on several arguments. On the theoretical front, Type A behavior is considered a typology rather than a continuum. That is Type A individuals are considered to behave in a qualitatively, not simply quantitatively different manner than Type B's (Matthews, 1982). Thus, the use of cutting scores that dichotomizes the sample is consistent with the conceptual basis of the Type A construct.

The decision to use a cut-off at the upper and lower third of the sample distribution, rather than a median split also rests on several empirical issues. Firstly, the mean MYTH score of the present sample of surgical patients is significantly lower than the mean of a sample of non-surgical children drawn from the general population, matched on age and sex. The latter sample of children consisted of classmates of the surgical patients, who were assessed on the MYTH by the same teachers. These comparisons are seen in Table 1, which reveals a significant difference between the two groups $t(224) = 2.81, p < .01$. Thus, the surgical sample is significantly more Type B than the general population. That is it contained a greater distribution of Type B children than normal. Using the upper third of the MYTH distribution of surgical patients ensures that these Type A subjects are more like or comparable with the general population. The second reason for the 30 percentile splits in this study is that it permits comparison to previous research. Table 1 also provides normative data from 3 recent American samples of Type A children. Once again, the mean MYTH scores of the current surgical sample are markedly lower than those of similar American youngsters drawn from a general population. Had a median split been used in this study, children with MYTH scores over 42 would have been classified as Type A, rendering them more comparable to American Type B subjects. As it is, the decision to use the upper 30th percentile, selects Type A subjects with MYTH scores over 48, which while still somewhat lower than the American norms does

improve the overlap and permits comparisons. The 30th percentile cut-off represents a conservative decision to improve classification and comparability, while retaining adequate sample size. It is noted that there is a growing consensus, at least as judged by trends in the recent Type A literature (eg. Matthews and Jennings, 1984; Jennings and Matthews, 1985) that when extreme scores are used, 30 percentile splits are preferable. Hence, the decision here is consistent with the treatment of MYTH scores in other research.

TABLE 1

MYTH Scores of Surgical and Non-surgical Patients
and Normative American Samples

Sample	A	B	C	D	E
<u>N</u>	122	108	485	88	41
<u>M</u> Age	8:1	8:4	gr.2	9:8	gr.6
<u>SD</u>	3:0	2:10	kgt to gr.6 (range)	9:3-10:11 (range)	-
<u>M</u> MYTH	42.95	47.55	51.4	n/a	54
<u>SD</u>	11.4	12.2	11.9	58 (>30%) 48 (<30%)	27 - 76 (range)

Note. A, Surgical Patients; B, Non-surgical; C, Matthews and Angulo (1980); D, Matthews and Jennings (1984); E, Lawler et al (1981).

It is recognized that extreme score groupings have an inherent danger of potentially excluding important information about mid-range subjects, if their performance is discrepant from a linear distribution. In order to examine whether Type A scores have a linear relationship with the variable of most importance, symptom reporting, and not some, curvilinear distribution that would mitigate against the use of extreme cut-off scores, a scattergram analysis was carried out. Using this procedure permits an analysis of the "goodness of fit" of the obtained regression line, and provides a measure "r" of the strength of the linear relationship. Thus, computing a regression line and associated "r" value tells one how closely a straight or linear relationship defines the bivariate distribution, and how strong this linear relationship is. Table 2 summarizes the scattergram analyses of all symptom report-Type A distributions. It indicates that in all cases, the linear regression line is a good fit to the data, accounting for a significant proportion of the concomittant variation. In other words, there is a linear relationship between symptom reports and Type A scores, and while not highly robust in strength, does fit a straight line nonetheless. Thus, the use of 30 percentile cut-off scores does not misrepresent the distribution by eliminating outliers but simply uses the extremes of a linear distribution.

TABLE 2

Scattergram Analysis of Relationship between MYTH Scores and
Symptom Inventory Rating Scale (SIRS) and Fatigue Scores

	<u>r</u>	<u>r</u> ²	<u>B</u> (slope)	<u>p</u>
MYTH score and:				
SIRS-C (Post 1)	-.29	.08	-.419	.0008
SIRS-C (Post 2)	-.32	.11	-.486	.0002
SIRS-C (Post 3)	-.23	.05	-.316	.006
SIRS-P (Post 2)	-.27	.08	-.33	.002
SIRS-P (Post 3)	-.19	.04	-.19	.024
Lab Fatigue	.35	.12	.15	.0002

Age and Sex Differences on Childrens Type A Scores

A 2 (Age) X 2 (Sex) ANOVA on MYTH scores was carried out in order to examine any effects of age and sex on Type A/B scores. As illustrated in Table 3, there was no statistical sex difference between these scores $F(1,83) = 2.74, p > .05$. As boys had somewhat, albeit non-significantly higher Type A scores than girls, a chi square analysis was carried out to ascertain whether they were unevenly represented in the Type A and B groups. The results (Appendix H) showed no significant difference between the observed and expected distributions

$\chi^2(1, N=85) = 2.79; p > .05$, indicating that boys are no more likely in this sample to be Type A than girls. The analysis

permits one to conclude that MYTH scores are statistically independent from the sex of the subject.

The effect of age on MYTH scores is also seen in Table 3. The Type A scores of younger children (ages 5 to 8) are not significantly different from those of the older (ages 9 to 14) children $F(1,83) = .012, p > .05$. There was no difference in the distribution of young and old children in the Type A and B groups (Appendix I) $\chi^2 (1, N=85) = .01, p > .05$ demonstrating that the A/B typology is also independent of age.

TABLE 3

Age and Sex Effects on MYTH Scores

	<u>N</u>	<u>M</u>	<u>SS</u>	<u>F</u>	<u>df</u>	<u>p</u>
<u>AGE</u>						
Young	55	43.5+13.8	2.6	.012	1,83	.91
Old	30	43.2+10.6				
<u>SEX</u>						
Boys	40	46.3+12.1	629	2.74	1,83	.11
Girls	45	40.9+12.9				

Demographic Characteristics of Type A and B Children

Table 4 summarizes the demographic characteristics of Type A and B children. As this information was not available on all subjects the n varies slightly. Type A children do not differ from Type B's in their family income, parental education, birth

order, number of siblings or the number of hospitalizations previous to the current surgery. The absence of significant findings on any demographic measure indicates that in these respects the Type A and B children may be considered equivalent.

TABLE 4

Demographic Characteristics of Type A and B Children

	n	Mean	SD	t	df	p
Family Income (\$)						
Type A	36	26,600	1100	1.27	-1,71	.21
Type B	37	30,200	1200			
Parents Education (yr)						
Type A	37	12.7	1.9	.59	1,71	.55
Type B	36	13.0	1.9			
Birth Order						
Type A	38	1.6	.8	.06	1,73	.95
Type B	37	1.6	.7			
Siblings						
Type A	38	1.1	.6	1.24	1,71	.22
Type B	35	1.2	.7			
Previous Hospitalizations						
Type A	38	.55	.9	.18	1,73	.85
Type B	37	.51	.8			

Parental Demographic Characteristics and Type A Scores

Data was available on 104 of the parents (one parent per child), 17 fathers (16%) and 87 mothers (84%). The mean parental education was 12.8 \pm 2.0 years and the mean family income was \$28,000 \pm \$1,200.

Table 5 provides a breakdown of demographics according to Type A and B grouping of parents. Parents whose score on the Jenkins Activity Survey was in the 50th percentile or above of normative standardized data were classified as Type A, while parents whose score corresponded to the lower 50th percentile of standardized data were classified as Type B. This procedure was followed because it is consistent with the treatment of adult Type A scores in the literature, and because JAS scores have been standardized, unlike MYTH scores with children, thus permitting comparisons with previous research.

TABLE 5

Demographic Data for Type A and B Parents

	<u>Type A</u>	<u>Type B</u>
N Total	48	56
<u>n</u> Female	38 (36.5%)	49 (47.1%)
<u>n</u> Male	10 (9.6%)	7 (6.7%)
Mean Education	13.5 years	12.3 years
SD	2.2 years	1.8 years
Mean Family Income	\$30,900	\$25,900
SD	\$1,000	\$1,300

~~Type A~~ parents have a higher mean number of years of education $t(102) = 2.16$; $p < .05$, and a higher mean family income $t(102) = 2.71$; $p < .01$. There were no sex differences in the JAS scores of parents. Fathers had a mean JAS score of 46.6 ± 6.2 and mothers had a mean JAS score of 44.0 ± 3.1 , $t(102) = .35$, $p > .05$.

Fathers were rather under-represented in both Type A and B groups, as they constituted only 16% of the total of participating parents. There were however no significant differences between the Type A and B groups of parents in the distribution of fathers and mothers $\chi^2 (1, N=104) = .77$; $p > .05$. That is, fathers were equally as likely to be Type A or B, as were mothers.

In order to examine the relationship between the Type A scores of children and their parents a correlation coefficient was calculated between MYTH and JAS scores. No significant association was found using the entire sample $r(104) = .008$; $p > .05$ or the 30% MYTH groups $r(85) = .006$; $p > .05$. Table 6 indicates that the parents of Type A children do not differ from the parents of Type B children in JAS scores, nor do the children of Type A parents differ in MYTH scores from the children of Type B parents. Chi square analysis confirmed that there was no significant difference in the distribution of Type A and B children among Type A and B parents $\chi^2 (1, N=104) = 1.63$; $p > .05$.

TABLE 6

Type A Scores of Parents and Children

	Mean	SD	t	df	p
Child's MYTH score by:					
Parents' Type A Group	44.2	13.5	.63	102	.53
Parents' Type B Group	42.7	12.0			
Parents' JAS scores by:					
Child's Type A Group	43.3	3.9	.40	73	.69
Child's Type B Group	40.8	4.7			

Examination of Hypothesis 1

Hypothesis 1 predicted that Type A children would report fewer clinical symptoms than Type B children following Tonsil and Adenoid surgery, as measured by both child self-report (SIRS-C) and parent (observer) report (SIRS-P). In order to examine this hypothesis a 2(Type A/B) X 2(Sex) X 3(Time) Analysis of Variance with Repeated Measures was run on SIRS-C scores (Appendix J), and a 2(TypeA/B) X 2(Sex) X 2(Time) Analysis of Variance with Repeated Measures was run on SIRS-P scores (Appendix K).

Type A and B children differed significantly in the mean number of clinical symptoms they reported after surgery ($F(1,83) = 10.68; p < .01$ and Table 7 demonstrates that this difference was in the predicted direction, with Type A children reporting significantly fewer symptoms than Type B's.

Table 7

Means and Standard Deviations of SIRS-C and SIRS-P Scores for Type A and B Children and for Boys and Girls

	<u>n</u>	<u>SIRS-C</u>		<u>SIRS-P</u>	
		<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>
Type A	43	71.90	12.4	67.5	11.7
Type B	42	80.90	17.5	75.7	14.0
Boys	41	79.45	14.4	71.1	13.0
Girls	44	74.20	15.2	71.0	12.6

There was a trend for boys to report more symptoms than girls $F(1,83) = 3.55$; $p = .06$, but there was no significant interaction between sex and Type A/B on SIRS-C scores $F(1,83) = 1.12$; $p > .05$. As expected there was a significant decrease in symptoms over time $F(2,83) = 93.72$; $p < .001$. There were no other significant findings from this analysis.

A 2(Type A/B) X 2(Sex) X 2(Time) ANOVA with Repeated Measures was carried out on the parents' rating of child symptoms (SIRS-P) and appears in Appendix K. These findings parallel the self-report measures. As seen in Table 7 parents rated Type A children to report significantly fewer symptoms post tonsillectomy/ adenoidectomy, than did the parents of Type B children $F(1,82) = 7.52$; $p < .01$. In addition the time effect was replicated $F(1,82) = 160.9$; $p < .001$. There was also no Type A/B X Sex interaction $F(1,82) = 0.1$; $p > .05$.

Results of the preceding ANOVAs provide full support for Hypothesis 1. On both Child and Parent reports, Type A children reported fewer clinical symptoms than Type B children. The Type

A effect on symptom reports was consistent across boys and girls and across all post-surgical rating periods. In light of the absence of any significant main or interaction effects due to sex on Type A scores or symptom reports, this variable was dropped from further analyses.

Correlations between Parent and Child Symptom Reports

The finding that Type A children under-report symptoms as measured by both child (self) and parent (observer) reports led to an examination of the concordance of parent-child ratings. The matrix of these correlations appears in Table 8 along with the correlations between parents' symptom ratings over time and between children's ratings over time. Correlations of importance between parent and child are those where symptom reports were made concurrently, that is on the same day. This occurred on Day 2 post-surgery (SIRS-C2 and SIRS-P1) and Day 7 post-surgery (SIRS-C3 and SIRS-P2). The correlation on Day 2 was .64, $p < .01$ and on Day 7 was .79, $p < .01$. The concordance or overlap in ratings is statistically significant, and accounts for 41% (Day 2) to 62% (Day 7) of the variance in these ratings.

Correlations between children's own ratings over the 3 post surgical periods were also significant; $r = .55$, $p < .01$ from Day 1 to Day 2, $r = .58$, $p < .01$ from Day 1 to Day 7, and $r = .55$, $p < .01$ from Day 2 to Day 7. Similarly, parents ratings were also consistent over time, $r = .57$, $p < .01$ from Day 2 to Day 7.

TABLE 8

Correlation Matrix of Parent-Child Symptom Ratings,
and Child and Parent Ratings over Time

	SIRS-C1	SIRS-C2	SIRS-C3	SIRS-P1	SIRS-P2
SIRS-C1 (Day 1)		r=.55 (n=84) p=.000	r=.58 (n=80) p=.000		
SIRS-C2 (Day 2)			r=.55 (n=80) p.000	r=.64 (n=84) p=.000	
SIRS-C3 (Day 7)					r=.79 (n=80) p=.000
SIRS-P1 (Day 2)					r=.57 (n=84) p=.000

Examination of Hypothesis 2

Hypothesis 2 predicted that Type A children, compared to Type B's, would report less fatigue on the laboratory symptom induction task. In order to examine this hypothesis a one-way ANOVA was calculated on the fatigue measure, "Time to 1", the time taken by subjects to report a maximum fatigue rating of "1". One-way ANOVAS were also calculated on the Amount of Weight (in grams) held by Type A and B subjects and on the Total Time (in minutes) that subjects actually held the weight. For purposes of comparison to previous research a one-way ANOVA was also calculated for Type A and B children on the mean of the last 3 fatigue ratings prior to putting the weight down. This measure is abbreviated "AvLas3" in the following tables. The results of these ANOVAS are seen in Table 9. It indicates that Type A and B children did not statistically differ in the

amount of weight held during the laboratory task $F(1,83) = .171$; $p > .05$. Type A and B children also did not differ in the amount of time they held the weight $F(1,83) = .73$; $p > .05$. Type A children did take a significantly longer time than Type B's on the Time to 1 measure, that is to report maximum fatigue $F(1,83) = 5.43$; $p < .05$, taking on average more than 3 minutes longer to report the same level of fatigue (8.8 minutes for Type A compared to 5.5 minutes for Type B's). Similarly, Type A children also reported significantly less mean fatigue over the last 3 ratings than did Type B subjects $F(1,83) = 12.49$; $p < .01$. These findings provide full support for hypothesis 2.

TABLE 9

ONEWAY ANOVA Comparing Type A and B Subjects
on Laboratory Fatigue

	<u>M</u>	SD	SS	df	<u>F</u>	<u>p</u>
Amount of Weight (gms)						
Type A	171	85	2626.5	1,83	.171	.68
Type B	159	90				
Total Time Held (min)						
Type A	9.58	5.0	5528.3	1,83	.73	.39
Type B	10.56	4.1				
Time to 1 (min)						
Type A	8.8	5.1	182.2	1,83	5.43	.023
Type B	5.5	4.1				
AVLas3						
Type A	27.06	7.0	3320.6	1,83	12.49	.001
Type B	12.66	6.4				

Concordance Between Clinical and Laboratory Symptom Reports

In order to ascertain the degree of association between symptom reporting in the laboratory with the reporting of real clinical symptoms, a correlational analysis was carried out (Table 10). With one exception, the laboratory fatigue measure correlated significantly with all clinical symptom ratings, indicating a significant degree of association between symptom reporting behavior in the lab and in real life.

TABLE 10

Correlation Between Clinical and Laboratory Symptom Reports

	Time to 1	AvLas3
SIRS-C1	r = -.294 (p = .01) n = 85	r = -.22 (p = .03) n = 85
SIRS-C2	r = -.273 (p = .017) n = 84	r = -.24 (p = .02) n = 84
SIRS-C3	r = -.33 (p = .004) n = 80	r = -.26 (p = .01) n = 80
SIRS-P1	r = -.14 (p = .13) n = 84	r = -.19 (p = .05) n = 84
SIRS-P2	r = -.34 (p = .004) n = 84	r = -.26 (p = .01) n = 84

Laboratory Fatigue and Reporting Delay

In light of the importance of symptom reporting to the patterns of delay in seeking medical attention, a further analysis on the laboratory fatigue data was carried out. Having obtained a measure of fatigue in terms of time, as well as a measure of total time worked, permitted an analysis of the stages in the reporting process that was analogous to stages found among adults in the delay in seeking medical attention (Safer et al 1979). It was assumed that the time taken to report maximum fatigue (Time to 1) was analogous to measures

used in the adult delay literature of the time taken to recognize symptoms as important or severe. The interval between "Time to 1" and "Total Time Held", when the subject actually discontinued the task by putting down the weight, may be analogous to the adult measure of time taken to decide to seek medical attention once symptoms are recognized, since both are remedial actions taken to alleviate symptoms. Thus a redefinition in terms of delay renders Time to 1 equivalent to "Recognition Time" or the delay due to time taken to recognize symptoms as serious or important, and Total Time Held minus Time to 1 is "Decision Time" or the delay due to time taken to decide symptoms warrant attention or remediation.

An analysis of variance comparing the stages of delay for Type A and B subjects was carried out (Table 11). It reveals that while there are no differences between Type A and B children in the total amount of time taken to discontinue the procedure, there were significant differences within the stages of the delay process. Specifically, it took Type A's a significantly greater length of time to recognize their fatigue as important or serious $F(1,83) = 5.43; p < .05$. Once symptoms were recognized as serious, Type A's took significantly less time to remediate the fatigue by terminating the procedure $F(1,83) = 7.6; p < .01$.

TABLE 11

ANOVA Comparing the Stages of Delay
between Type A and B Children

	<u>M</u>	SD	SS	df	<u>F</u>	<u>p</u>
Total Time (min)						
Type A	9.6	5.0				
			5528.3	1,83	.73	.396
Type B	10.5	4.1				
Recognition Time (min)						
Type A	8.8	5.1				
			182.2	1,83	5.43	.023
Type B	5.5	4.1				
Decision Time (min)						
Type A	1.3	.8				
			155.59	1,83	7.6	.007
Type B	3.9	.9				

Examination of Hypothesis 3

Hypothesis 3 predicted that among Type A subjects, older children would report fewer clinical symptoms than younger subjects as measured by the SIRS-C and SIRS-P, and less laboratory fatigue.

A. CLINICAL SYMPTOMS

Table 12 summarizes the Analysis of Variance with Repeated Measures examining the main effects of age on the SIRS-C and SIRS-P of Type A children. Older Type A subjects self-reported significantly more clinical symptoms than younger Type A's

$F(1, 41) = 5.27$; $p < .05$. There were no differences between younger and older Type A's as rated by parents $F(1, 41) = 2.08$; $p > .05$. While these findings run counter to the predicted direction a 2(Age) X 2(TypeA/B) ANOVA with Repeated Measures on SIRS-C and SIRS-P scores (Appendix L) found that age had a significant main effect across both Type A and B groups. In general, older children reported significantly more clinical symptoms than younger children on both self-report $F(1, 83) = 5.58$; $p < .05$ and observer ratings $F(1, 82) = 5.42$; $p < .05$ with no Age X Type A/B interaction on either measure. Hence, age had a significant main effect on symptom reports following Tonsil and Adenoid surgery, regardless of Type A behavior.

TABLE 12

Clinical Symptom Reports of Young and Old Type A Children

	<u>n</u>	<u>M</u>	<u>SD</u>	<u>SS</u>	<u>df</u>	<u>F</u>	<u>p</u>
SIRS-C							
Young	27	69.4	10.7	1473.1	1, 41	5.27	.027
Old	13	76.9	13.7				
SIRS-P							
Young	28	65.9	9.4	420.0	1, 41	2.08	.156
Old	13	70.8	15.5				

B. LABORATORY FATIGUE

An initial examination of hypothesis 3 on the laboratory task using a one-way ANOVA (Appendix M), found a similar result to the clinical symptoms. Older Type A's reported more fatigue than younger A's, in taking significantly less time to report maximum fatigue levels $F(1,41)=8.27$; $p<.01$. To report the same level of maximum fatigue it took older Type A's on average 3.75 minutes while younger Type A's took 11.0 minutes, about a 3-fold difference. This finding is confounded however by the significantly greater amount of weight held by older Type A's (315 gm) than younger Type A's (116gm), again about a 3-fold difference $F(1,41)=29.8$; $p<.01$. Weight contributed significantly to the fatigue measure $F(1,41)=4.65$; $p<.05$ accounting for approximately 21% of the fatigue variance (Adjusted Beta = .465). Thus, a more accurate examination of the effects of age on Type A fatigue reports requires a statistical partialling out of the effects of weight using an Analysis of Covariance procedure. When an ANCOVA is carried out (Appendix M) on Age effects within Type A subjects with weight partialled out, the previously significant difference between younger and older A's is eliminated, rendering no difference on fatigue ratings between younger and older Type A's $F(1,41)=3.41$; $p >.05$.

On the surface, this analysis suggests that there are no age-related changes in Type A's reporting of fatigue. However, using a statistical procedure to partial out the effects of weight from fatigue ratings fails to capture the full

relationship between age and Type A's symptom reporting. That is, older Type A's may have held more weight simply because they were older and presumably stronger, for which an ANCOVA procedure would be appropriate. However, it is also possible that older Type A's held significantly more weight than younger A's because they actually tried harder, i.e. made greater efforts to excel and to hold more weight by under-reporting their subjective fatigue. In order to fully explore this possibility it was necessary to examine the fatigue ratings in the context of the amount of work done by the subject. Work can be measured in "joules" and is defined by the term:

Mass X (Distance)² / (Time)² (Stevenson and Moore, 1967). As all subjects kept the weight stationary, the distance travelled was constant. Hence the distance term was given the value of 1 for all subjects, reducing the work equation to:

Work = Weight X (1)/(Total Time Held)². A simple transformation that multiplies the fatigue measure by the amount of work expended provided a measure of the fatigue experienced due to the work output of the subject. This ought to provide a better account of age-related fatigue changes. Table 13 summarizes the transformed fatigue ratings and work outputs of Type A subjects.

When examined in this way it is seen that in fact older Type A's did make greater efforts to excel than did younger A's, working 4 times as hard $F(1,41)=16.97; p<.001$. Fatigue ratings when transformed by work expenditure also showed a marked difference between younger and older Type A's. For the amount

of work expended older A's took longer to report maximum fatigue than did younger A's, almost 3 times as long (3.36 minutes compared to 1.78 minutes. This difference just failed to reach statistical significance $F(1,41)=3.62$; $p=.06$.

Hypothesis 3 did not receive statistical support.

TABLE 13

Work Output and Work-Related Fatigue of Type A Subjects

	<u>n</u>	<u>M</u>	<u>SS</u>	<u>df</u>	<u>F</u>	<u>P</u>
Total Work (joules)						
Young A	30	1.29	7.36	1,41	16.96	.000
Old A	13	3.28				
Work-Related Fatigue (min-joule)						
Young A	30	1.78	18.89	1,41	3.62	.066
Old A	13	3.36				

Examination of Hypothesis 4

This hypothesis predicted that Type A children would under-report more symptoms relative to Type B's, at Day 7 post-surgery than at Day 1. In other words it was expected that clinical symptom report differences between A's and B's would be greater at Day 7 than Day 1. In order to test this hypothesis a $2(\text{Type A/B}) \times 2(\text{Time})$ ANOVA with Repeated Measures on children's clinical symptom reports (SIRS-C) was carried out (Table 14). As a parent rating (SIRS-P) was not available at Day 1, no comparison of child and parent symptom reports was done.

TABLE 14

Time Effects on Type A and B Children's Clinical Symptom Reports

	<u>n</u>	<u>M</u>	SD	SS	df	<u>F</u>	<u>p</u>
Time							
Day 1	80	87.6	18.5				
Day 7	80	64.0	17.4	2234.8	1,78	164.7	.000
Type A/B							
Type A	41	70.7	12.1				
Type B	39	81.2	20.5	4333.5	1,78	9.45	.003
Time X Type A/B				3.1	1,78	.02	.88

The ANOVA indicates that overall there was a significant reduction in the number of symptoms reported by subjects over time $F(1,78)=164.7$; $p<.001$. Regardless of Type A/B grouping, children reported fewer symptoms one week following surgery than they did immediately afterwards. Consistent with the results of Hypothesis 1, Type A subjects reported significantly fewer clinical symptoms than Type B subjects when Days 1 and 7 data are collapsed $F(1,78)=9.45$; $p<.01$. However, there was no significant interaction between time of rating and Type A $F(1,78)=.02$; $p>.05$, indicating that reporting differences between Type A and B subjects are not significantly different at Day 1 from Day 7. Hypothesis 4 is not supported.

Examination of Hypothesis 5

This hypothesis predicted that symptom reporting would be lowest among children whose parents were both Type A and low symptom reporters themselves. In parents, Type A was assessed by the Jenkins Activity Survey (JAS) and symptom reporting assessed by the Pennebaker Inventory of Limbic Languidness (PILL). For the purposes of data analysis, PILL scores were dichotomized into high and low groups using a median split procedure ($ms = 82$).

A. CLINICAL SYMPTOMS

In order to test Hypothesis 5 a 2(Type A/B Parent) X 2(PILL) ANOVA with Repeated Measures was performed on children's clinical symptom ratings, both self-report (SIRS-C) and observer report (SIRS-P). For this hypothesis children's symptom reports were compared between 4 groups of parents; Type A under-reporters, Type B under-reporters, Type A over-reporters, and Type B over-reporters. The appropriate statistical analyses for the hypothesis were the Interaction term of the 2(Type A/B) X 2(PILL) ANOVA with Repeated Measures and a Duncans Multiple Range test comparing all 4 groups.

Tables 15 and 16 summarize the SIRS-C and SIRS-P means and standard deviations for the parental Type A and PILL groups, and the ANOVA results are presented in Appendix N.

TABLE 15

Means and Standard Deviations of Children's Clinical Symptom Reports by Parent Type A and Parent PILL Groups

		SIRS-C	
		Type A	Type B
LO PILL	x =	74.0 ±	74.2 ±
	SD	15.9	15.3
	n =	23	25
HI PILL	x =	83.5 ±	78.1 ±
	SD	18.3	16.2
	n =	26	25
		SIRS-P	
		Type A	Type B
LO PILL	x =	69.5 ±	69.9 ±
	SD	13.3	11.7
	n =	22	26
HI PILL	x =	75.0 ±	72.5 ±
	SD	16.0	13.7
	n =	26	26

Table 15 presents the SIRS-C and SIRS-P ratings made by the four parental groups. There were no statistical differences between the means of these 4 groups on the SIRS-C $F(1,97)=1.01$; $p>.05$, or the SIRS-P $F(1,98)=.18$; $p>.05$.

There were differences in children's clinical symptom ratings when parents own symptom reporting behavior alone was taken into account. As seen in Table 16, under-reporting (Lo PILL) parents had children who reported fewer clinical symptoms, as assessed by both self and observer rating, compared to children whose parents were high reporters (Hi PILL).

Analysis of variance on these differences (Appendix N) found a significant main effect due to parent's symptom reporting on children's self-reported clinical symptoms $F(1,97)=5.57$; $p < .05$, and a trend on parent ratings of children's clinical symptoms $F(1,98)=3.31$; $p = .07$.

There were no statistical differences between the children of Type A and B parents in their reporting of clinical symptoms on either the SIRS-C $F(1,97)=.85$; $p > .05$, or SIRS-P $F(1,98)=.33$; $p > .05$.

ANOVAs examining the interaction effects of children's Type A status with parental variables were all non-significant. That is, the effects of parental variables on children's symptom reporting were consistent across Type A and B children.

TABLE 16

Marginal Means and Standard Deviations of Children's Clinical Symptoms by Parent Type A Groups and by Parent PILL Groups.

<u>SIRS-C</u>	<u>n</u>	<u>Mean</u>	<u>SD</u>
Type A Parent	49	76.2	18.9
Type B Parent	50	79.6	20.6
Lo PILL	48	74.6	18.4
Hi PILL	51	80.9	20.6
<u>SIRS-P</u>			
Type A Parent	48	72.6	17.0
Type B Parent	52	73.0	16.9
Lo PILL	48	70.9	15.4
Hi PILL	52	74.5	18.0

B. LABOARTORY FATIGUE SYMPTOMS

Examination of Hypothesis 5 on children's laboratory fatigue was carried out by a 2(Parent Type A/B) X 2(PILL) ANOVA on fatigue measures. The statistic of interest is the interaction term (Type A/B X PILL). In order to more closely examine differences in children's fatigue reports between parental groups, a Duncans Multiple Range test was also carried out. The ANOVA and Duncans results are presented in Appendix O. Table 18 summarizes the means and standard deviations of children's fatigue reports across parent groups.

There was a significant interaction on the ANOVA comparing the children' fatigue scores between parent's Type A and PILL groups $F(1,98) = 6.47; p < .05$. Table 17 indicates that children of Type A under-reporting parents had the lowest fatigue reports, as measured by the time taken to report maximum fatigue. In order to confirm that it was the Type A under-reporting parent group that statistically accounted for the significant interaction term, the Duncans Multiple Range test results were examined. The overall F ratio for the Duncans procedure comparing multiple means $F(3,92)=3.45; p<.05$ indicates that there is a significant difference between at least two of the parent group means. Using the shortest significant range at the .05 level (range = 3.88), two subsets of groups were found to have highest and lowest means to differ by more than the shortest range. These significant differences were between the Type A under-reporters and Type A over-reporters, and between the Type A under-reporters and Type B

under-reporters. While Type A under-reporters also had less children's fatigue than Type B over-reporters, the difference failed to exceed the critical range value. These analyses indicate that the fatigue reporting of children is statistically lowest when parents are Type A under-reporters. Secondary analyses of variance found no significant interactions between children's Type A and parental variables on fatigue scores, although there was a trend for the Type A children of Type A parents to report less fatigue than other children $F(1,82) = 3.10$; $p = .08$. Table 18 presents these means.

In summary, hypothesis 5 received partial support with the prediction substantiated on laboratory induced symptoms but not clinical ones. For the latter, it was found that clinical reports were only significantly affected by parents PILL scores.

TABLE 17

Means and Standard Deviations of
Children's Fatigue by Parent Variables

	Type A	Type B
LO	x = 10.15	x = 5.36
PILL	+ 1.4	+ .86
	(n = 20)	(n = 28)
HI	x = 6.13	x = 7.83
PILL	+ 1.0	+ 1.1
	(n = 23)	(n = 23)

TABLE 18

Mean Fatigue Scores by Type A and B Child and Parent Groups

	CHILD	
	Type A	Type B
Type A	11.67 +1.0	5.07 +.89
PARENT		
Type B	6.41 +1.1	5.83 +1.0

Examination of Hypothesis 6

This hypothesis predicted that symptom reporting would be lowest among children whose health locus of control was highest. Stated differently, it was predicted that high (or internal) health locus of control children would report fewer symptoms than children whose health locus of control was low (or external).

A. CLINICAL SYMPTOMS

The statistical test for this hypothesis is a 2(Locus) X 3(Time) Analysis of Variance with Repeated Measures of Health Locus of Control on self (SIRS-C) and observer (SIRS-P) clinical symptom reports. The results of these ANOVAs appear in Appendix P. Also presented in Appendix P are the results of a 2(Locus) X 2(Type A/B) X 3(Time) ANOVA with Repeated Measures on clinical symptom reports, carried out in order to examine any interaction effects between health locus of control and children's Type A behavior on symptom reporting. The means and standard deviations of health locus of control groups clinical reports appear in Table 19.

The analyses indicate that there were no significant differences in children's clinical symptom reports between those with high and low health locus of control, using both self-report $F(1,120)=.54; p>.05$ or observer report $F(1,119)=.44; p>.05$. There was no significant interaction between health locus of control and Type A/B on clinical symptom ratings, either for self report $F(1,83) = .69, p > .05$, or observer reports $F(1,82) = .11, p > .05$. The one significant

finding was that with observer (parent) reports, there was a significant interaction between health locus of control and time of rating $F(1,120)=4.6$; $p<.05$. This finding is presented in Table 20.

The main hypothesis concerning health locus of control and symptom reporting was not supported using both self and observer ratings.

TABLE 19

Means and Standard Deviations of Children's Clinical Symptom Reports by Health Locus of Control Groups

<u>SIRS-C</u>		<u>SIRS-P</u>	
LO LOCUS	HI LOCUS	LO LOCUS	HI LOCUS
$\bar{M} = 75.8$	$\bar{M} = 78.1$	$\bar{M} = 70.6$	$\bar{M} = 72.4$
$\bar{SD} = 14.8$	$\bar{SD} = 16.6$	$\bar{SD} = 11.9$	$\bar{SD} = 14.4$

Table 20

Observer (SIRS-P) Symptom Reports for Health Locus of Control Groups Over Time

	POST 1	POST 2
LO LOCUS	$\bar{M} = 80.8$ $\bar{SD} = 15.8$	$\bar{M} = 60.4$ $\bar{SD} = 8.1$
HI LOCUS	$\bar{M} = 79.6$ $\bar{SD} = 15.8$	$\bar{M} = 65.2$ $\bar{SD} = 13.7$

B. LABORATORY FATIGUE SYMPTOMS

A Oneway Analysis of Variance on the laboratory fatigue scores for low and high health locus of control children (Appendix Q) was not significant $F(1,120)=.17; p>.05$. Low health locus of control children reported maximum fatigue after 7.3 minutes compared to 6.97 minutes for high locus of health control subjects. A 2(Locus of Control) X 2(Type A/B) ANOVA on the laboratory fatigue measure indicated no significant interaction between health locus of control and Type A behavior $F(1,83)=1.33; p>.05$.

The hypothesis was not supported for laboratory fatigue symptom reporting.

Relationship of Health Locus of Control and Type A Behavior

While no significant main or interaction effects due to health locus of control were found, it was of interest theoretically to know how health locus of control related to Type A behavior in this sample of children. A univariate ANOVA was run on the Type A/B scores of high and low health locus of control children. MYTH scores of high health locus of control children ($M=44.2, SD=11.2$) and low health locus of control subjects ($M=42.7, SD=11.9$) did not significantly differ $F(1,83) = .39; p>.05$. The correlation between MYTH scores and health locus of control was in the positive direction but was also not significant $r(85) = .04; p >.05$.

In summary there was no significant relationship between Type A behavior and health locus of control.

RELATED INFORMATIONType A and Health-Related Behaviors

In order to provide further construct validation data on the SIRS and an elaboration of Type A children's health-related behavior, analyses of pre and post-surgical school attendance data and post-surgical medication useage were carried out. Correlational analyses on the entire sample indicated a significant positive relationship between symptom reports on the SIRS and the number of school days missed following surgery. Greater symptom reports by children (SIRS-C) were associated with more days missed from school following surgery $r(69)=.22$; $p<.05$. There was a positive trend in greater school absence with parent reports (SIRS-P) of children's symptoms $r(66)=.19$; $p=.06$. Pain medication useage also correlated significantly with symptom reports using the SIRS. Greater self-reported clinical symptoms were associated with higher medication use by the child, both at one day post surgery $r(80)=.23$; $p=.01$ and 7 days post surgery $r(81)=.27$; $p<.01$. Similarly, medication useage also increased with parent reports of children's symptoms at one day post surgery $r(79)=.24$; $p<.01$ and 7 days post surgery $r(82)=.31$; $p<.01$. There was a a significant positive relationship between the number of school days missed and medication use one day after surgery $r(50)=.39$; $p<.01$, but not with medication use one week later $r(48)=.07$; $p>.05$. The correlational data indicates that as the number of symptoms reported on the SIRS increase so too does the amount of pain medication and the number of days

missed from school.

It was also of interest to examine the relationships between Type A and health-related behaviors. Type A children missed on average 6.1% of school days due to illness prior to surgery compared to 7.4% for Type B's $F(1,78)=1.55$; $p>.05$. Following surgery, Type A's missed 6.5 days before returning to school compared to 7.75 days for Type B's $F(1,78)= 1.58$; $p>.05$. There was a significant interaction between Type A/B behavior and the sex of the child on post-surgical attendance $F(1,78)= 4.69$; $p<.05$. There was no significant main effect due to sex on post-surgical attendance $F(1,78)=.014$; $p>.05$. A post hoc Duncans Multiple Range (critical range=1.57) confirms that it was the Type A boys who missed significantly less school post-surgery than other children. (Table 21).

TABLE 21

Post-Surgery Absences (in days) for Type A and B Boys and Girls

	TYPE A	TYPE B
BOYS	x =6.64 sd=1.9	x =8.8 sd=2.4
GIRLS	x =7.6 sd=2.1	x =7.17 sd=2.4

There were no differences between Type A and B children in their pain medication useage, either one day post surgery $F(1,78)=.19; p>.05$ or one week later $F(1,78)=.98; p>.05$.

Analyses of the effects of parents Type A behavior on their own and their children's health behavior were also carried out. Type A parents sent their children back to school significantly earlier following surgery than Type B parents $F(1,78)=5.08; p<.05$. Type B parents kept their children home on average 8.5 days post surgery, while Type A parents kept their children home on average only 6.5 days. There were no differences in children's pre-surgery school attendance between Type A and B parents. $F(1,78)=.12; p>.05$. Interestingly, despite parental differences in post-surgical school attendance, there were no differences in how many analgesics they gave their children $F(1,78)=.78; p>.05$. Type A and B parents did not significantly differ in reporting their own symptoms on the PILL $F(1,92)=.34; p>.05$.

Effects of Family and Social Factors on Symptom Reporting and Health-Related Behaviors

Children with a history of previous hospitalizations, compared to those with no previous history, did not differ on any of the symptom report measures, clinically or in the laboratory, nor on attendance or medication useage.

Only children, those with no siblings living at home, had a tendency to take less pain medication than children with siblings living at home $t(74)=16.95; p=.07$). The amounts of

medication taken by both groups were so small however (1.4 pain tablets compared to 2.8 tablets over a 5 day period) that this trend should be interpreted with caution. On all other health-related and symptom reporting behaviors, only children were no different than children with siblings. Similarly, first born children demonstrated no significant differences from later born children on any health-related or symptom reporting behavior.

As previously reported; sex had no effect on symptom reporting behavior. Similarly, boys and girls did not differ in the amount of pain medication taken one day after surgery $F(1,78)=2.2$; $p>.05$ nor one week after surgery $F(1,78)=.04$; $p>.05$, nor did boys and girls differ in the number of days missed from school prior to surgery $F(1,78)=2.2$; $p>.05$ or after surgery $F(1,78)=.014$; $p>.05$.

Multiple Regression

In order to identify the best predictors of symptom reporting behavior a multiple regression analysis was carried out on both clinical and laboratory measures. As there was no a priori assumption regarding any causal ordering among the various predictor variables, a standard regression strategy using forward (step-wise) inclusion was used. Step-wise procedures permit evaluation of predictive strength and accuracy among a number of predictor variables (i.e. overall contribution to the regression equation) and decomposition of explained variance into orthogonal components, in order to

evaluate the independent contribution of a particular variable with the influence of other variables controlled for (partial regression coefficients).

Three separate regressions were done. The first was on children's self-report measures of clinical symptoms (SIRS-C), using as predictor variables scores on the MYTH, JAS, and PILL, age, laboratory fatigue scores and parent ratings of child symptoms (SIRS-P). As seen in Table 22 the six predictor variables accounted for 62% of the variance, and 60% of this was due to two variables, parent ratings and childrens' MYTH score. Parental ratings were the single best predictor of childrens' post surgical symptomatology. Both parent ratings and MYTH scores made significant statistical contributions to the prediction equation.

A second regression was done on parent ratings of children's symptom reports using as predictors children's age, family income and education, MYTH, JAS and PILL scores, and children's self-reports (Table 23a). This regression was then repeated without childrens' self-reports (Table 23b). When children's self-reports were included the prediction of parent ratings accounted for 50% of the variance, but dropped to 32% when excluded. While the latter was a less powerful prediction, it is more interesting as it points to other important contributions to parent ratings. Table 23b indicates that the child's age, family income and parents PILL scores all made significant contributions to parent ratings, and accounted for 30% of the variance.

The third regression was done on laboratory fatigue and used self and parent symptom reports, age, MYTH, JAS and PILL scores as predictors (Table 24). This combination accounted for 31% of the variance, with parent PILL scores making the single biggest contribution to the prediction equation. Childrens' MYTH scores and age also made significant contributions to the prediction.

TABLE 22

Multiple Regression of Clinical Symptom Self-Reports (SIRS-C)

Variable	Beta	SE	F	Δr^2	p
SIRS-P	.66	.10	48.2	.55	sig.
MYTH,	-.23 -	.07	6.5/	.05	sig
JAS	-.10	.04	1.3	.01	no
Age	.07	.39	.76	.005	no
PILL	-.02	.04	.06	.0004	no
Time 1	.01	.28	.01	.0001	no

MULTIPLE R = .786

 $r^2 = .62$

TABLE 23a

Multiple Regression on Observer Rating
 (SIRS-P) of Child Clinical Symptoms

Variable	Beta	SE	F	Δr^2	p
SIRS-C	.39	.09	.79	.30	sig
Age	.30	.41	8.4	.11	sig
Income	.17	.97	1.9	.04	no
PILL	.12	.04	2.9	.01	no
JAS	-.10	.04	.88	.009	no
MYTH	-.07	.07	.37	.004	no
Education	.05	.64	.26	.002	no

MULTIPLE R = .71

 $r^2 = .50$

TABLE 23b

Multiple Regression on Observer Ratings of Child Symptoms
(SIRS-P) Without Childrens' Self-Reports

Variable	Beta	SE	F	Δr^2	P
Age	.32	.46	10.1	.15	sig
Income	.23	1.10	3.0	.08	sig
PILL	.22	.05	3.2	.07	sig
MYTH	-.14	.08	1.5	.01	no
Educ	.09	.72	.57	.007	no
JAS	.05	.05	.17	.002	no

MULTIPLE R = .57

$r^2 = .32$

TABLE 24

Multiple Regression on Childrens' Laboratory Fatigue Rating

Variable	Beta	SE	F	Δr^2	P
PILL	.35	.02	7.5	.19	sig
MYTH	.29	.04	8.6	.06	sig
Age	-.22	.26	5.3	.05	sig
JAS	.07	.03	.45	.003	no
SIRS-C	-.07	.05	.17	.001	no
SIRS-P	.04	.06	.05	.000	no

MULTIPLE R = .55

$r^2 = .31$

CHAPTER 6
DISCUSSION

Subject Information

Several important overall findings were revealed regarding this sample of Type A children and adults. Of foremost significance was the finding that the surgical sample of patients obtained a significantly lower MYTH score (i.e. were more Type B) than a matched sample of same-aged, same-sex children from the general population. Of the possible explanations for this finding 2 warrant consideration. First it might be argued that as subjects were not randomly selected, but were chosen by virtue of a non-random event, namely tonsil and adenoid surgery, that a distribution bias may have occurred. This argument is countered however by the matching procedure used in sampling the control group from the general population. This procedure serves to render the 2 groups homogeneous on a number of important variables. While the use of a matched group design (or random block design) may always suffer from some unknown source of variance not controlled by matching, the current samples are demographically homogeneous. Thus, differences in MYTH scores between surgical and non-surgical samples are not likely due to selection problems.

The second more compelling explanation comes from the Type A construct itself. It may be that, if in fact, Type A subjects do under-report symptoms, then they may be less likely to present themselves for medical attention. Inasmuch as the decision to seek medical attention for children rests on symptom reports by

the child to the parent, Type A children may be less likely to report symptoms to parents and therefore to receive medical care. Thus, the lower Type A distribution of the surgical sample may reflect a health outcome of Type A under-reporting.

The Type A distribution of both the surgical and non-surgical samples was considerably lower than normative samples of American children drawn from the general population. This was a rather surprising finding and no immediate explanation is evident. One might speculate that there are cultural differences between American and Canadians that resulted in lower Canadian MYTH scores. For example, the aggression/hostility component of the Type A construct may be less well developed early in the lifespan of Canadian children. This line of reasoning may also help account for the absence of Type A/B sex differences in this sample, unlike most previous American work finding boys to be significantly more Type A than girls (Lawler et al 1981; Lundberg 1983; Matthews and Angulo, 1980; Sweda et al, 1986). Since boys are often considered more aggressive than girls (Macoby and Jacklin, 1974) any cultural diminution of aggression may reduce sex differences between Type A and B's. However, this speculation awaits empirical validation. The present sample was consistent with previous research in the direction of sex effects, with boys obtaining higher MYTH scores (more Type A) than girls. As with previous research there was no difference between older and younger children in their MYTH scores.

Among adults Type A parents in the study had significantly

more education and significantly greater incomes than Type B parents. To the extent that education and income measure achievement, this sample of Type A adults has greater achievement striving, a core characteristic of the Type A construct, and provides validation for the sample of parents. The study found no relationship between the Type A scores of parents and their children, adding to the equivocal nature of aggregation data.

There were no sex differences in adult Type A/B scores, also adding to the equivocal findings on this issue. However the current finding of no sex differences may in part reflect an unintended sample bias. The fathers who participated in the study (only 17 out of 104 parents) were those who took their children to the post-surgical medical follow-up. These fathers may have been more family rather than career oriented, and consequently less Type A, than those fathers who did not participate. Had both parents been assessed it is conceivable that a sex difference between adults might have surfaced. Future work of this kind would therefore do well to obtain Type A data for both mother and father.

Validity and Reliability of the S.I.R.S.

The study of symptom reporting is predicated on the accurate measurement of children's symptom reporting behavior. For reasons outlined earlier, a scale was specifically designed for this study, namely the Symptom Inventory Rating Scale (SIRS).

Preliminary empirical support for the use of the SIRS was found in the pilot study and was reported earlier. Results from

the current study provide further reliability and validity data. The reliability of the SIRS was assessed using measures of inter-rater and test-retest reliabilities. Inter-rater reliability was determined by comparing the SIRS scores of children with those of their parents who independently completed the SIRS during the same time interval. The correlations for these ratings were uniformly high, ranging from .64 at one day post surgery to .79 one week later. This level of concordance, accounting for 41% to 62% of the shared variance, is typical or slightly better than that found with other parent-child health measures (Andrasik et al, 1984). Test-retest reliabilities were also uniformly significant. While the level of the correlations were modest, ranging from .55 to .58 over a one week period, this is expected given the changing nature of the symptoms being measured. It is noteworthy in fact that despite changing symptom levels, test-retest scores were as consistent as they were. Remarkably, even pre and post surgery correlations among pilot subjects were significant, ranging from .46 to .61, confirming that the SIRS provides a consistent assessment of symptom reporting behavior.

The validity of the SIRS was determined by comparing the scale with other measures of symptom reporting, by measuring changes in the scale as a result of surgery and recovery, and by measuring illness behaviors associated with SIRS scores. The latter provides a measure of criterion validation while the others are measures of construct validation.

In order to establish the construct validity by examining the

relationship between the SIRS and other symptom report measures, children in the pilot study were administered a visual analogue scale assessing the degree of distress currently experienced, and all children in the study completed an experimental laboratory measure of symptom reporting. Both measures provided external ratings of the symptom reporting construct against which SIRS scores were compared. Using the visual analogue scale ratings provided a test of the assumption that children who reported many symptoms were in more distress than those who reported few symptoms. Similarly, if the SIRS tapped symptom reporting in a clinical context it ought to correlate with laboratory measures of the same construct.

Correlations between SIRS scores and visual analogue scores were significant and modestly high ranging from .52 at post surgery to .70 pre-surgically. Hence the greater the symptom reports, the greater was the associated subjective distress. Stated differently, the higher the SIRS score, the more discomfort the child experienced. This finding supports what would be expected from a scale measuring physical symptom reporting. Hence, in the sense that SIRS scores are sensitive to subjective levels of distress due to symptomatology, construct validity support for the scale may be taken.

This is consistent with the data measuring changes in SIRS scores as a result of surgery and recovery. There were significant differences in subjects SIRS scores before and after surgery, and from one day to seven days post surgery. Hence, the SIRS is sensitive to changes in symptom severity. The

dovetailing of the distress and severity data supports the validity of the SIRS in its ability at minimum, to detect changes in clinical symptomatology following tonsil and adenoid surgery.

Correlations between the SIRS scores and the laboratory measure of the symptom reporting construct were also significant. Children who reported few symptoms on the SIRS also tended to under-report on the laboratory measure, a rating of fatigue induced by holding a weight. Similarly, high SIRS scores were associated with high scores in the laboratory.

Using the laboratory method to provide construct validation is a particularly strong validation strategy for clinical health measures (Chapman et al, 1985). It can demonstrate that assessment measures in one context predict behavior in another context, and therefore permits one to make generalizations about the accuracy and meaningfulness of the measure. In this regard, the strong concordance between the clinical scores on the SIRS and the laboratory measure of the construct, illustrates that the SIRS has meaning beyond the specific context of tonsil and adenoid symptoms and can predict reporting behavior of other symptoms. The findings suggest that the SIRS measures symptom reporting propensities, not simply the level of symptomatology. Hence, the data supports the use of the SIRS as a measure of the symptom reporting construct. One of the limitations of previous health measurement research has been the failure to integrate work in the laboratory and clinical arenas (Chapman et al, 1985). The use in this study of clinical and laboratory

measurements of symptom reporting behavior helps to bridge the gap between these contexts, in order to facilitate a comprehensive understanding of the construct.

Validation for the SIRS was also assessed by examining illness behaviors associated with symptom reporting, namely the number of school days missed following surgery and the amount of analgesic medication used by the patient. Both illness behaviors were significantly correlated with SIRS scores, such that the more symptoms reported on the scale, the greater was the post-surgical school absenteeism and pain medication usage. These results provide criterion-related validation for the SIRS. That is, independent criteria or medical end-points are predicted by scores on the scale.

In summary, there is good empirical support for the use of the SIRS as a measure of children's symptom reporting behavior. Good criterion-related and construct validity evidence was found, excellent inter-rater reliability and good test-retest reliability were established. In all there was a convergence of evidence to provide good support for the use of the SIRS as a clinical measure of children's tendency to report symptoms. Future factor analytic work is planned for the scale.

Hypotheses 1 and 2

Hypothesis 1 predicted that Type A's would report fewer clinical symptoms than Type B's following tonsil and adenoid surgery, as measured by both self-report and parent ratings. Hypothesis 2 predicted that Type A's would report less fatigue in the laboratory than Type B's.

Both hypotheses received full support. Under controlled laboratory conditions Type A children took significantly longer, on average 3 minutes longer than Type B's to report the maximum level of fatigue from holding a weight. Moreover, Type A's reported significantly less fatigue over the last 3 fatigue ratings than did Type B's. This relationship was confirmed by the regression data. Type A behavior made a significant contribution to the prediction of laboratory symptom reporting behavior and in fact, was the second best sum predictor variable. Thus, relative to Type B children, Type A's under-report laboratory fatigue.

These findings are consistent with previous research. Matthews and Volkin (1981) found, using the identical task, that grade 6 boys under-reported laboratory induced fatigue. A similar pattern has been demonstrated among Type A adults (Matthews and Brunson, 1979; Carver and Coleman, 1976). According to the authors, Type A subjects so behave in order to devote full attention to excelling at the task at hand. It would also appear to account for the present findings. While Type A's did not hold the weight any longer than Type B's, they did exert considerably more energy, as measured by the amount of work they expended. That is, gram for gram of weight, Type A's worked 30% harder than Type B's, and in the service to do so under-reported their subjective fatigue. Interestingly, when anecdotally asked after the procedure was over, how hard they tried on a 1 to 7 scale, Type A's stated they tried harder than Type B's. Thus, not only do current results replicate previous data, they also

support the notion that achievement striving may mediate Type A's symptom under-reporting.

As Matthews and Volkin's (1981) research used only grade 6 boys, it was unclear whether girls and younger children would demonstrate similar behavior. This study permitted an answer to this question, and indicated that the Type A under-reporting phenomenon occurs in both sexes and across age groups. Hence the study not only replicates but extends previous data, and permits a generalization that under-reporting occurs in Type A girls as well as boys across young and old ages.

An important aspect of laboratory research is its ability to model, under controlled conditions, real-world behavior. In this regard changes or stages in children's symptom reporting may be examined as an analogue of the stages of decision making in seeking medical care. Such an analogue is meaningful for heart disease research in pointing to sources of delay in seeking medical treatment following symptom onset, particularly since treatment delay may be fatal. Seventy percent of infarction deaths occur within 4 hours of symptom onset but patients often delay going to the hospital for more than 3 hours (Hackett and Cassem, 1969). Moreover, the most important component of delay is the time taken to decide treatment is necessary (Moss et al, 1969). Thus, reducing patient delay in treatment decision making may be beneficial. Using the 3. stage delay model of Safer et al (1979), an analysis of analogue data was done. It was found that Type A children took significantly longer than Type B's to recognize their fatigue was at its maximum or most serious.

However, once recognized as such, Type A children were much quicker than Type B's to take remediative action to alleviate their symptoms, ~~metaphorically~~ to seek treatment for their symptoms. By analogy then, Type A children have much longer illness decision times but quicker treatment decision times.

The pattern of delay found in this sample of Type A children is similar to that found in Type A adult myocardial infarction patients whose inordinate delay in seeking treatment for their symptoms contributes to increased coronary risk (Matthews et al, 1983; Green et al, 1974). This parallel in illness behavior is suggestive that differences in illness decision making and its associated risk for coronary heart disease may begin early in life. It would be interesting to know whether the health outcomes of these behavioral differences also occur in children. Among adults this pattern of delay and symptom reporting is associated with a greater severity of heart disease (Matthews et al, 1983). Similar data does not exist for Type A children. One line of converging evidence may be the finding that Type A children were under-represented in the elective surgery population, suggesting that their under-reporting and delay is associated with under-utilization of medical services. Conceivably, this could lead to an exacerbation of symptoms and any underlying disease. Of course, some uncontrolled variable may have contributed to the observed relationship, such as a long distance to hospital, or parental work responsibilities, that created a barrier to health care. Hence a more direct examination of the actual extent or severity of illness in Type

A and B children would provide a strong test of the Type A-illness relationship in children.

Turning to the findings regarding the post-surgical symptomatology directly addresses the question of whether Type A children under-report symptoms in real life. No previous study has examined this question. The current data also permits examination of the continuity or stability of symptom reporting in children across various contexts.

Post-surgically, Type A subjects reported fewer symptoms than Type B's as measured by both self-report and parent ratings. Type A' under-reported across all time periods, across young and old age groups and across both sexes. Hence, there pervasive tendency, irrespective of time, age, or sex for Type A children to under-report clinical symptoms. Moreover, not only did Type A children self-report fewer clinical symptoms, but independent observational ratings of the children made by their parents concurred. These findings are confirmed by the regression data that Type A behavior made a significant contribution to clinical symptom reporting, as the second best predictor variable. The clinical data suggests that the Type A symptom under-reporting phenomenon is indeed meaningful, evident not only with ambiguous laboratory induced symptoms but also with important, clinically relevant ones. These data have several important implications.

The findings from parent ratings of children's symptoms are of special interest. First of all, their strong concordance with the children's own reports augers for their continued use in child health measurement. They provide important observational

data that is both reliable and valid (Andrasik, et al, 1984; McGrath et al, 1985). Secondly, and of more importance to the present study is the implication for children's illness behavior. Parent ratings were based essentially on observation of their children's behavior, the child's spontaneous verbal reporting, and minimally on direct questioning. Therefore, the finding that parents rated Type A children as they did suggests that the under-reporting of these children is apparent at the behavioral level as well as verbally. While not possible in this study, a systematic behavioral observation study of the post-operative or laboratory induced illness behavior of Type A and B children could examine this hypothesis. There is some evidence in adults that Type A subjects demonstrate fewer observable pain behaviors during clinical exercise testing than Type B's (Castell and Blumenthal, 1984).

The clinical implications of Type A children's illness behavior may be considerable. For example, if these children appear less symptomatic to parent observers they may be perceived as healthier and therefore receive less medical care, even when required. This may partly account for the observed under-representation of Type A children in the surgical sample. Post-operative implications may be more serious. Parents may perceive less symptomatic Type A children as fully recovered from surgery or illness when they may not be, thus permitting a quicker return to normal activity such as school. In fact Type A children in this study did return to school after surgery one day earlier on the average, than Type B's. Although this may be

admirable in terms of achievement one might be concerned about the risk of post-operative complications:

Another important health implication of Type A's under-reporting may be non-compliance with medical regimens. As symptoms are used by patients to represent their illness state, changes in symptom perception may lead to changes in defining illness and therefore the need for treatment (Leventhal, 1985). It is possible that the tendency to under-report symptoms would engender the Type A child with chronic disease to under-estimate illness and need for treatment, thus contributing to non-compliant behavior. There is evidence that post myocardial infarction Type A patients rapidly become non-compliant with exercise rehabilitation as the perception of their symptoms decrease (Rejeski, 1984) and that they are prone to under-reporting due to misattribution of symptoms during the rehabilitation period (Gastorf and Suls, 1982). There is also evidence that Type A behavior is associated with non-compliance and decreased glucose control among diabetics (Peycot and McMurray, 1985). It would be interesting to examine whether a similar phenomenon occurs in chronically ill children, such as the the diabetic adolescent whose insulin must be injected regularly, independent of self-perceived symptom levels.

The reasons that the Type A child under-report clinical symptoms are not immediately evident. However, the most parsimonious account, consistent with the laboratory observations, would suggest that their high achievement striving may be causal. Thus, they would be highly motivated to return to

school, sports and so on in order to continue their high achievements. Available data, albeit correlational, would support this interpretation. Type A children, particularly Type A boys did return to school on the average one day sooner than their Type B peers. The observation of one mother was interesting, in that she claimed she had to force her Type A child to stay home despite his eagerness to return to school 3 days post-surgery.

As with the laboratory data, it was somewhat surprising to find no sex differences in children's reporting of clinical symptoms. While there are no comparable data with other studies of Type A and B children, as the only previous report was restricted to boys only, the literature suggests that girls report more symptoms than boys (Lewis and Lewis, 1982). The data are not unequivocal however, as some studies do not find sex differences (Gochman, 1971). For the purposes of this paper, perhaps the more important implication is that the Type A-Type B difference in symptom reporting is independent of sex.

Comparing the clinical results with those obtained in the laboratory has broader implications for the symptom reporting construct. It was found that concordance between laboratory and clinical reporting behavior was uniformly high, such that individuals who under-reported in the laboratory environment tended to under-report clinically. In addition, correlations between clinical symptom reports across the 3 rating periods were uniformly significant, and had over 30% in shared variance. Similarly, pilot testing found remarkable consistency in

children's symptom reporting. In fact correlations of symptom reports before surgery with those afterward were rather high accounting for 20% to 37% of the variance. The observed continuity of behavior suggests that childrens' symptom reporting behavior may be a relatively stable trait variable (Pennebaker, 1982; Leventhal, 1985). This interpretation converges with the data from the adult literature that individuals have characteristic symptom reporting styles that function across settings. Moreover, the data suggests that this behavioral disposition emerges early in life.

There is also suggestive evidence in both children and adults that symptom reporting has a dimensional, continuous quality (Costa and McRae, 1985; Mechanic, 1980). That is individuals range in their characteristic reporting behavior from stoical under-reporting to frank hypochondriasis. For example reporting after surgery may have more to do with this reporting style than the actual medical procedure, although the surgery may influence the kind of symptoms reported. As such, clinicians should always call into question the veridicality of physical symptom reports and health assessments (Linden et al, 1986; Costa and McRae, 1985) and recognize that in children as well as adults they may be biased by characteristic reporting styles.

While the current study treated symptom reporting as a categorical variable for the purposes of facilitating classification, the obtained distribution was clearly dimensional. Clinical use of the symptom reporting construct should therefore take this into account. It may be somewhat

arbitrary to divide individuals into under and over-reporters without specific criteria. For example, simplistic psychiatric models of symptom reporting assume that patients' physical symptom reports up to some point are real, after which they are seen as signs of mental not physical illness (Costa and McRae, 1985). Clinicians and researchers may find it more useful to consider the adaptiveness of the reporting behavior in determining whether it is under or over-reporting (Pennebaker, 1982). For example, chronic pain patients may under-report the severity of their symptoms but in doing so may cope better with their disability than over-reporters, who take more medication, miss more work, are less active and so on (Fordyce et al, 1985). In contrast this study in its concern with coronary heart disease, used criteria related to issues of medical under-utilization, treatment delays and non-compliance in defining under and over-reporting. There may be illness specific issues that render under-reporting adaptive or functional for one disorder and maladaptive or dysfunctional for another.

In summary, the data fully support Hypotheses 1 and 2. Relative to their Type B peers, Type A children under-report the frequency and severity of physical symptoms in a laboratory environment as well as in a clinical context. This behavior is externally validated by parent-observer ratings. Type A under-reporting is independent of age and sex, occurring across all age groups studied and among both boys and girls. It is suggested that the under-reporting behavior in both contexts is in the service of achievement striving. Moreover, the illness

behaviors associated with Type A adult under-reporting are also demonstrated in the study. In light of these data it seems reasonable to suggest that the behavioral risk factors for coronary heart disease emerge early in life and that they may be reliably identified in Type A children. The apparent continuity of symptom under-reporting and illness behaviors in Type A children and coronary patients suggests a developmental pathway linking Type A behavior in childhood with coronary heart disease in adults. This implies, with empirical support, that early intervention programs for the primary prevention of coronary heart disease may do well to target populations of Type A children and focus on their associated health and illness behaviors. For example recent data from the Bogalusa Heart Study (Croft et al, 1986) found decreases in adolescent cardiovascular risk status were derived from lifestyle alterations in childhood. In addition, prospective clinical trials have demonstrated that Type A behavior is modifiable; at least in adults, with improved coronary outcomes (Friedman et al, 1984). However, a proviso is noted in that the costs and benefits from similar trials with children have yet to be done.

Hypothesis 3

This hypothesis predicted that symptom under-reporting among Type A's would increase with age. It was expected that as the Type A behavior pattern became consolidated over time, older Type A subjects would be more motivated to under-report their physical symptoms, that is more interested in and capable of focusing attention on their high achievement striving than their

younger counterparts. The results of post-surgical symptom reporting did not support the hypothesis. In fact, older A's reported more clinical symptoms than younger A's. As it turned out, age had a similar effect on Type B's clinical symptom reports.

The most likely explanation for this finding is that tonsil and adenoid surgery is simply more difficult for older children than younger children. This view is consistent with the popular belief that having tonsils out is easier at the younger ages. It would seem that if Type A under-reporting does increase with age, it is overshadowed in this case by the greater difficulty of the surgical procedure at the older ages.

Findings from the laboratory tasks were somewhat more complex. Initial examination of the data suggested a similar pattern to the clinical reports, that older Type A's reported more fatigue, not less, than younger Type A's. However, this was due to the fact that older A's held 3 times the amount of weight than younger A's, making comparisons unfair, particularly as weight contributed significantly to fatigue scores. When the fatigue reports were examined in the context of the actual amount of work expended, using a simple mathematical transformation, older Type A's were found to report considerably less fatigue than younger A's, taking almost 3 times as long to report maximum fatigue. While this difference was in the predicted direction, it failed to reach statistical significance, and may best be considered a trend. Given the exploratory nature of this work, it may be useful to tentatively

consider the implications of this finding, and test these against replication data.

From a clinical perspective the data implies that with some symptoms, such as fatigue, Type A's under-reporting may increase with age in order to make greater efforts to excel. However, the relationship with age may not be present where the underlying motivation to achieve is absent. A clinical implication from this data is that under-reporting may become more difficult to alter or modify as it becomes consolidated with age in the Type A behavior repertoire. As health promotion and intervention becomes more expensive and often less effective in adulthood (Evans, 1982) these data might suggest further evidence for beginning behavioral risk factor intervention programs early, during the childhood years.

The findings may also have theoretical implications. It is now evident that symptom under-reporting occurs among Type A's in adulthood, adolescence and childhood. Thus there appears to be a developmental continuity across the lifespan. While cross-sectional data is not as strong an inference strategy as longitudinal data, these results are consistent with the stability of other Type A behaviors. Overt Type A characteristics such as impatience, aggression, and achievement striving have also been found to remain stable across the childhood years (Matthews and Avis, 1983) consolidate during middle childhood (Steinberg, 1986) and continue into adulthood (Bergman and Magnusson, 1986; Steinberg, 1985, 1986). Hence the under-reporting phenomenon may become an enduring characteristic

associated with the Type A pattern that persists throughout the lifespan.

Hypothesis 4

This hypothesis predicted an interaction between Type A behavior and the time of clinical reporting. It was expected that at 7 days post surgery symptoms would be less salient and that children would have returned to school, and as a result, A-B reporting differences were expected to be greater than day 1 post-surgery. This expectation was grounded in the experimental and clinical literature that salience and environmental challenge differentially effect Type A and B symptom reporting. Under conditions of high environmental challenge Type A's suppress attention to symptoms in order to excel (Pardine et al, 1984; Schleigel et al, 1984). Similarly, low salient symptoms elicit symptom under-reporting for Type A's but not for Type B's (Stern et al, 1981; Gastorf and Suls, 1982). Hence, Day 7 was expected to represent a low symptom salience/high environmental challenge condition, while Day 1 was a high salience/low challenge condition.

The results provided no support for the hypothesis. There was no interaction between Type A behavior and the time of rating. Symptom reporting differences between Type A and B children were consistent across Days 1 and 7. The failure to find support may have occurred for 3 reasons. For one, salience and environmental challenge may in fact have no effect on symptom reporting. This however is unlikely as both the experimental and clinical literature are consistent in this regard, using a variety of

symptoms in a variety of settings. While it may be possible that these effects are less important for children than they are in adults, there is no data at present to support this view. A second possibility is that the methodology did not provide a strong enough manipulation of salience or environmental challenge. Ratings on Days 1 and 7 were thought to provide a natural and unobtrusive manipulation of salience, as the natural history of post tonsil and adenoid surgery symptoms is a rapid decrease in severity (Paradise, 1983). Moreover, it was expected on this basis that children would have returned to school by Day 7 providing a naturally occurring environmental challenge that would compete for individual's attention. Unfortunately this did not occur as surgeons conservatively instructed parents to keep their children home for at least 10 days. As a result, there was no school work competing for children's attention that might have differentially effected Type A and B subjects and their reporting of low salient symptoms. A later rating time after return to school might have provided a stronger test of the hypothesis. The third possibility is a "floor effect". It is possible that Type A subjects had under-reported as much as possible given the nature of the symptoms and the symptom rating scale.

The findings do confirm that Type A symptom under-reporting is stable over time and across widely differing situations, such as the home and hospital. Moreover, the demonstrated decrease in symptom reporting for both Type A and B children over time supports the construct validity of the SIRS, to the extent that

it is able to detect changes due to the natural history of post-surgical symptoms.

Hypothesis 5

It was expected that children of Type A under-reporting parents would be the lowest symptom reporters in both the laboratory and clinical situations. The hypothesis was derived from a social learning model of symptom reporting behavior development that suggests parents influence their children to label and report symptoms in ways similar to their own (Pennebaker, 1982; Mechanic, 1980). The model predicts that symptom reporting would be lowest among children whose parents are both Type A and under-reporters since this behavior would be modelled by the under-reporting parent and reinforced directly, as parents reward behavior similar to their own, as well as reinforced indirectly as a means to an end of achievement encouraged by the Type A parent. Hence a cumulative effect on childrens' symptom reporting would be expected . . .

The findings provided some support for the hypothesis. In the laboratory the least fatigue was reported by children of Type A under-reporting parents. In fact these children took from 3 to 5 minutes longer, or 20% to 30% more time than other children to report the same level of maximum fatigue. The regression data supports this view as the single best predictor of childrens fatigue reports was the parents own symptom reporting behavior. This parent variable accounted for 19% of the variance in childrens fatigue reporting. In addition, while parental Type A did not make a significant independent contribution to childrens

fatigue, it did interact with childrens Type A behavior. Specifically, Type A children of Type A parents took considerably longer than all other children, from 5 to 6 minutes longer (30% to 40% longer) to report maximum fatigue. Hence, in the laboratory the hypothesis was fully supported, and judging from the size of the group differences it seems that parent variables had a considerable impact.

A converging but less robust set of results occurred clinically. Children of under-reporting parents reported significantly fewer clinical symptoms than did children of over-reporting parents. There was no significant interaction between parent Type A and symptom reporting. While the results could not confirm the expected relationship between parent variables and childrens clinical reports, the regression data does suggest that something important in this area is going on. The single best predictor of childrens' clinical symptom reports was the parents' observational rating, which along with parents' Type A accounted for 56% of the variance. Moreover, two of the top three predictors of the parent ratings had to do with the parent, not the child. These variables were parent income and parents own symptom report scores and accounted for 23% of the observed variance. In other words, the single best predictor of how children felt after surgery was the parents rating, and the best predictors of these ratings were other parent variables, not child variables. The only child variable that significantly entered the regression equation to influence parent ratings was childrens age. Hence the regression data indicates that parental


variables impart a significant clinical effect on childrens symptom reporting, providing conceptual support for the hypothesis.

Overall the clinical results dovetail with those from the laboratory. There was a tendency for Type A under-reporting parents to have the lowest symptom reporting children, providing some support for the social learning hypothesis. Parents own symptom reporting behavior had the most consistent impact on childrens' reporting, as it was the single best predictor of childrens reporting in the laboratory and the third best clinical predictor, and suggests that overall it is the most important contributing parent variable. Parental Type A made a more modest contribution to childrens' reporting, and was more important in the laboratory than clinically, particularly among Type A children. In general, parent variables imparted a significant effect both clinically and in the laboratory, on childrens' symptom reporting.

Given the correlational nature of the data it is of course impossible to ascribe causality to the observed association between childrens symptom reporting and parent variables. However, it seems unlikely that the direction of the relationship is from child to parent, but rather that parent variables such as Type A and symptom under-reporting, influenced childrens symptom reporting. This interpretation is consistent with previous research findings. Mechanic (1980), Pennebaker et al (1981), Campbell (1978) and others have all found that the strongest predictors of childrens symptom reports and illness

behaviors were parental variables, such as parents' self-reported level of symptomatology. In fact two recent studies have found that mothers' attitudes and behaviors make a stronger contribution to children's illness behavior than the child's own age or sex (Campion and Gabriel, 1985; Maiman, Becker, and Katli, 1986). The data from this and other studies suggest that if one is interested in children's symptom reporting, one should learn about their parents' attitudes and behaviors.

The current data replicates previous research in the area of children's illness behavior and lends some support to the idea that symptom reporting is learned through a process of socialization (Cambell, 1978; Mechanic, 1980). Mechanic (1980) found that parent variables acted on children's symptom reporting in both an indirect fashion, through the influence on perceived health, and in a direct way by influencing children's tendency to monitor inner sensations. A similar phenomenon may have occurred in this study. Parents' symptom reporting behavior, given its consistent effect across both settings, may have directly influenced children's behavior to the extent of providing an available model and reinforcement for similar behavior. Parents' Type A behavior may have acted more indirectly, altering health perception because of parental emphasis on achievement. For example, children of Type A parents missed significantly less school after surgery, returning on average 2 days before the children of Type B parents. It is possible that in their eagerness to encourage children's school achievement (Matthews 1977; Copeland et al, 1984) Type A parents indirectly encouraged



their children to minimize distracting health problems and illness symptoms in order to get back to school quickly. The cumulative effect of parental Type A and parental under-reporting behavior in lowering childrens' symptom reporting is consistent with a social learning model of symptom reporting development.

The interpretation of a social learning pathway must really be considered tentative given the correlational nature of the study data. It is possible that parent variables imparted their effect in other ways, such as genetic transmission of pain threshold or pain tolerance differences. Similarly, under-reporting parents may simply be healthier than over-reporters, and have healthier offspring who have less symptoms to report. This may not account however for much of the variance in the present study as childrens' symptom levels were directly manipulated. It is hoped that future research will investigate these and other possible mechanisms using a prospective clinical design or an experimental laboratory method. For example, a laboratory analogue study using parents and co-actor children, and children with co-actor parents (caretakers) could help tease apart the direction of any transmission effects of parental variables on childrens' symptom reporting behavior.

The parent data have several important clinical implications. The first of these is that any effort to alter childrens symptom reporting behavior, whether it be intervention for under or over-reporting, must take into account parental reporting

behavior and to some extent parental attitudes such as Type A behavior. In other words, modification of childrens illness behaviors must attend to both child variables and parent behaviors and attitudes. Failure to do so would be an error of omission, neglecting a significant mediating influence. For example, simply teaching a child new reporting behavior in ones office or clinic may not generalize to environments or settings where opposing reporting models and contingencies, such as parents reporting behavior, continue to operate, or where motivational systems and attitudes underlying the behavior (such as parents Type A) persist. Hence introducing parents as teachers and models of new reporting behavior in a social learning context (Patterson, 1975) may enhance intervention efficacy.

The data also have implications for Type A behavior and coronary risk reduction. This is the first known study to examine the illness behaviors of the offspring of Type A adults, and extends the data base regarding this population. The data suggests that Type A adults not only put themselves at risk by adopting coronary prone behaviors, but may transmit certain behaviors to their children, notably the propensity to ignore physical symptoms in order to achieve and return to work (school) more quickly after illness. The present findings replicate previous research that Type A parents are more directive and pushing than Type B's of their childrens' achievement striving (Matthews, 1977; Copeland et al, 1984), and extends the implications to childrens' health. It is possible

that part of the contribution to heart disease risk made by family history is the transmission to offspring of risk behaviors such as symptom under-reporting, since genetic factors do not entirely explain family aggregation variance (Kate 1982). If children in fact acquire illness behaviors via parental modelling, then the offspring of Type A adults, particularly those with existing disease, may be at risk for acquiring maladaptive illness behaviors. Further family aggregation study is needed to examine this possibility and to replicate the current health-related findings.

In terms of coronary risk reduction the above findings suggest that the offspring of Type A's, particularly Type A coronary patients may have learned maladaptive symptom reporting behaviors. Hence it may be useful for risk reduction programs that operate through cardiac rehabilitation and prevention settings to include the children of patients for certain educational and treatment components. In particular, teaching modules aimed at disseminating information on the nature of symptoms or on illness decision making could easily include children, at least at the older ages. Similarly, rehabilitation programs aimed at modifying adult Type A behavior should include a family session regarding illness behavior, both for the patients benefit, and as a preventative measure for offspring. Piggybacking a primary prevention program on top of existing tertiary prevention, provides economic advantages, saving both time, and money, as well as the opportunity for healthy individuals to observe the negative outcomes of maladaptive

illness behavior. Social psychological research suggests that a certain amount of fear, in combination with information on effective behavioral strategies, facilitates intention to acquire new behaviors (Leventhal, 1982).

Hypothesis 6

This hypothesis predicted that symptom reporting would be lowest among children with internal health locus of control. The expectation was derived from research data that perceived lack of control increases symptom reporting among adults (Pennebaker et al, 1977) and children (Gochman, 1971; Mechanic, 1982), and that children with lower internal locus of control perceive themselves as more vulnerable and more willing to report symptoms and use health care services (Gochman, 1971).

The data failed to provide any support for the hypothesis. No differences in symptom reporting were found between children with internal and external health locus of control. Neither was there any interaction between Type A behavior and health locus of control in symptom reporting, nor any relationship between childrens MYTH scores and their health locus of control. Type A's were no more internal in their health locus of control than were Type B's.

The findings cast some doubt on the ability of health beliefs to predict illness behaviors. In part this difficulty may reside in the fact that the health locus of control construct, derived from the Health Belief Model (Becker, 1974) was designed to account for preventative health behavior not illness behavior. However the problem may be as much a methodological one. The

Health Belief Model has received little empirical validation among children. In addition, assessment methodologies of childrens health beliefs are in their infancy (Jordan and O'Grady, 1982). It is possible for example that the Children's Health Locus of Control scale used in this study, while providing a valid assessment of the construct as it is seen in adults, may not tap the unique variance in childrens health locus of control.

The absence of a relationship between Type A behavior and health locus of control was somewhat surprising as Type A's usually seek and therefore have a perception of greater internal control than Type B's (Glass, 1977; Wolf et al, 1979). It is possible that the Type A's internalized locus of control is less evident in childhood or does not extend to health until adulthood. The findings do confirm though that the effect of Type A behavior on childrens symptom reporting is independent of health beliefs.

Summary and Limitations of the Study

The major purpose of this research was to examine the validity and generalizability of symptom under-reporting in Type A children by studying male and female Type A and B subjects across young and old age groups, in both a laboratory and clinical context. The study was able to confirm the primary hypothesis that Type A children, like their adult counterparts, do under-report physical symptoms relative to their Type B peers. It was also possible to address the questions of whether

Type A under-reporting extends to girls and younger children, and to important clinical symptoms. The study found that under-reporting was evident in girls as well as boys, in younger as well as older children, and on clinical symptoms as well as laboratory fatigue. In addition the study also found that for some types of symptomatology Type A under-reporting may increase with age. Hence, the results were able to directly address the major research questions and provided support for the primary hypotheses of the study.

A second purpose of the study was to examine the relationship of childrens' symptom under-reporting to other healthy related behaviors such as parents' symptom reporting and Type A behavior, health locus of control and symptom salience. Only some of these secondary hypotheses were supported. As predicted, it was found that the lowest symptom reporting occurred in children whose parents were both Type A and symptom under-reporters themselves. In general, childrens' symptom reporting was strongly predicted by parent variables. The study could not provide evidence regarding the effects of salience or environmental challenge, nor of health locus of control on symptom reporting. However, the study found that childrens' symptom reporting was associated with their school attendance and medication use, and that Type A's under-reporting was associated with a similar pattern of illness decision making and illness behavior as seen in adult coronary patients. A noteworthy serendipitous result was also found, Type A children were under-represented in a surgical sample.

The study has several important clinical and theoretical implications. An important thrust of this research has been to address the question of when and how Type A behavior becomes coronary prone. While previous data has established that the behavior pattern emerges early in life it is not at all clear when it becomes associated with coronary risk. The current data suggests that at least one aspect of what makes the Type A pattern coronary prone in adults, emerges early in life and can be reliably identified in children. In this regard, Type A behavior has coronary prone components during childhood. These findings auger for the continued investigation of the origins of other coronary risk factors, in order to plan a comprehensive (i.e. multiple risk factor) primary prevention program.

The findings also suggest that a continued focus on components of Type A behaviors may be particularly beneficial. Not all Type A children under-reported symptoms, and among adults not all Type A's develop coronary heart disease. Hence, it may be useful to identify within a target population for intervention, subjects who possess coronary prone components, rather than say all Type A children or all offspring of coronary patients.

The findings from the parental data suggest that any intervention must consider parental symptom reporting behaviors and attitudes. Coronary risk reduction strategies that piggyback primary prevention on top of adult cardiac rehabilitation may provide economic advantages as well as effective therapeutic leverage in modifying childrens' risk related behaviors. The

current data suggests that a social learning approach using parents as models for behavior change, may provide a starting strategy for child-oriented intervention. The cross-sectional evidence finding developmental continuity in symptom under-reporting and illness behaviors across the lifespan, suggests that behavioral intervention aimed at risk reduction ought to begin early in life.

The findings have other health implications in addition to coronary heart disease. It is possible that the pattern of illness behavior seen in this sample of Type A children, in particular their tendency to delay and under-utilize medical services, may place them at risk for more advanced symptomatology and disease severity. Moreover, their propensity to ignore physical symptoms in order to achieve, while admirable on the one hand, may jeopardize their recovery from illness and surgery, and contribute to non-compliance with ongoing medical regimens.

Theoretically, the results have implications for the symptom reporting construct. Children are relatively stable in their symptom reporting behavior across time, across settings and across various types of symptomatology. From a cross-sectional perspective, Type A individuals demonstrate a pervasive tendency to under-report across the lifespan. The developmental and cross-situational stability supports a trait interpretation of the symptom reporting construct, and suggests that children are like adults and have a characteristic reporting style or disposition that may range from stoicism to hypochondriasis. A

continuous rather than categorical classification scheme of symptom reporting may therefore be more appropriate for clinical practice. In addition, practitioners need to recognize that every health assessment may be biased by childrens' reporting disposition.

The study has attempted to extend the knowledge base in several areas; the Type A construct itself, behavioral prevention of heart disease and the symptom reporting construct. Nonetheless there are several limitations to the study that warrant mention. First of all, the data was correlational in nature and despite the effort to control various confounds through matching procedures, strict inclusion and exclusion criteria, and the combined use of clinical and laboratory methods, it is always possible that some uncontrolled third variable contributed to the relationship between Type A behavior and symptom reporting. One possibility is that Type A children were simply healthier than than Type B's and had less symptomatology to report. This would seem unlikely in this case given that symptoms were directly manipulated. Another possibility is that Type A's have higher pain thresholds than Type B's. This position would suggest that Type A's would be less sensitive in perceiving symptoms. This too seems unlikely as Type A's reported as much fatigue as Type B's when the laboratory procedure was over, under-reporting only during task completion. As was previously suggested, future research using a prospective clinical design or an experimental laboratory design may provide a stronger test of the Type A under-reporting

hypothesis.

A second limitation of the study is that parental data was limited to only one parent. This restricted the generalizability of the family aggregation data on Type A behavior. In addition, the predominance of mothers in the study, while consistent with previous research which shows they bear the major responsibility for childrens' health care, restricts the social learning interpretation regarding symptom reporting development to a largely maternal effect. Hence, future work of this kind would do well to include both parents even though it may result in slightly more non-compliance among participants.

A third limitation concerns the actual model directing the study, that being a psychological or behavioral model of disease prevention. The Type A risk for coronary heart disease is well established and it is assumed that identifying the behavior pattern early in life may permit remediation or prevention of its pathogenic components. However, it is not at all clear that Type A children in fact grow up to be the Type A adults who develop heart disease. The relationship of childhood Type A to coronary end points has not yet been established. It is prospective epidemiological work such as the ongoing Bogalusa Heart Study (Croft et al, 1986) that may shed light on this issue. The current study has simply established that the behavior pattern does become associated with risk-related components at an early age, and contributes to illness behaviors that are similar to those seen in coronary adults. No causal connection to heart disease has been established. As such, the

support generated by the current data for early intervention and primary prevention, while compelling, is still indirect and awaits confirming epidemiological data. It has been argued elsewhere (Sereganian, 1985) that the marriage or juxtaposing of the epidemiological and psychological models may promote the most effective prevention and rehabilitation programs.

Finally, the current study was based on children's reporting of laboratory induced and post-surgical symptoms. It is possible that more serious illnesses with longer recovery periods or chronic illnesses may not elicit the same behavioral responses seen here. Thus the clinical implications may not extend beyond the specific symptoms studied. While this limitation may be countered by the strong concordance between the laboratory and clinical data and the replication by this study of previous laboratory work, it may be prudent to consider the findings in a heuristic sense. That is, as a way to generate further hypotheses for future studies. Replication and extension of these results, particularly the clinical data, would provide greater confidence in the generalizability and meaningfulness of the findings.

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APPENDIX A

MATTHEWS YOUTH TEST FOR HEALTH - FORM O

Name of Child: _____ Age: _____

extremely uncharacteristic

I68.

extremely characteristic

Instructions. Please circle the number that best describes this child.

- | | | | | | |
|--|---|---|---|---|---|
| 1. When this child plays games he/she is competitive. | 1 | 2 | 3 | 4 | 5 |
| 2. This child works quickly and energetically rather than slowly and deliberately. | 1 | 2 | 3 | 4 | 5 |
| 3. When this child has to wait for others he/she becomes impatient. | 1 | 2 | 3 | 4 | 5 |
| 4. This child does things in a hurry. | 1 | 2 | 3 | 4 | 5 |
| 5. It takes a lot to get the child angry at his peers. | 1 | 2 | 3 | 4 | 5 |
| 6. This child interrupts others. | 1 | 2 | 3 | 4 | 5 |
| 7. This child is a leader in various activities. | 1 | 2 | 3 | 4 | 5 |
| 8. This child gets irritated easily. | 1 | 2 | 3 | 4 | 5 |
| 9. He/she performs better than usual when competing against others. | 1 | 2 | 3 | 4 | 5 |
| 10. This child likes to argue or debate. | 1 | 2 | 3 | 4 | 5 |
| 11. This child is patient when working with children slower than he/she is. | 1 | 2 | 3 | 4 | 5 |
| 12. When working or playing he/she tries to do better than other children. | 1 | 2 | 3 | 4 | 5 |
| 13. This child can sit still long. | 1 | 2 | 3 | 4 | 5 |
| 14. It is important for this child to win, rather than to have fun in games or schoolwork. | 1 | 2 | 3 | 4 | 5 |
| 15. Other children look to this child for leadership. | 1 | 2 | 3 | 4 | 5 |
| 16. This child is competitive. | 1 | 2 | 3 | 4 | 5 |
| 17. This child tends to get into fights. | 1 | 2 | 3 | 4 | 5 |

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JENKINS ACTIVITY SURVEY

by C. DAVID JENKINS, STEPHEN J. ZYZANSKI, RAY H. ROSENMAN

THE PSYCHOLOGICAL CORPORATION

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SYMPTOM INVENTORY RATING SCALE
CHILD FORM

Please tell us how you feel today by entering the number that best fits you in the space beside each question. (Note to parents: Any assistance should be limited to reading and defining words and writing, only when necessary). For all items use this scale:

1	2	3	4	5
no	a little bit	some	a lot	really bad

DO YOU FEEL/HAVE

- | | | |
|-----|-------|----------------------------|
| 1. | _____ | Dizzy? |
| 2. | _____ | Itching or painful eyes? |
| 3. | _____ | Ringing in your ear(s)? |
| 4. | _____ | Hard to hear? |
| 5. | _____ | Lump in your throat? |
| 6. | _____ | Choking sensation? |
| 7. | _____ | Sneezing spells? |
| 8. | _____ | Runny nose? |
| 9. | _____ | Stuffy nose? |
| 10. | _____ | Bleeding nose? |
| 11. | _____ | Sore nose? |
| 12. | _____ | Asthma or wheezy? |
| 13. | _____ | Out of breath? |
| 14. | _____ | Hurt to swallow? |
| 15. | _____ | Sore throat? |
| 16. | _____ | Hurt to talk? |
| 17. | _____ | Scratchy throat? |
| 18. | _____ | Tiredness? |
| 19. | _____ | Hard to sleep? |
| 20. | _____ | Toothache? |
| 21. | _____ | Upset tummy? |
| 22. | _____ | Pain or cramps in stomach? |
| 23. | _____ | Heartburn? |
| 24. | _____ | Sore muscles? |
| 25. | _____ | Diarrhea? |

1
no

2
a little
bit

3
some

4
a lot

5
really
bad

DO YOU FEEL/HAVE

- 26. _____ Constipation?
- 27. _____ Pain in throat?
- 28. _____ Sore joints?
- 29. _____ Stiff muscles?
- 30. _____ Rash?
- 31. _____ Itchy?
- 32. _____ Popping sound in ear?
- 33. _____ Ear feel warm inside?
- 34. _____ Sore ear?
- 35. _____ Pressure in ear?
- 36. _____ Earache?
- 37. _____ Stiff neck?
- 38. _____ Weakness?
- 39. _____ Pain in neck?
- 40. _____ Feeling of pressure in head?
- 41. _____ Headaches?
- 42. _____ Feeling hot or sweaty?
- 43. _____ Chills?
- 44. _____ Feel like throwing up?
- 45. _____ Have you been throwing up?
- 46. _____ Feel faint, like passing out?
- 47. _____ Twitching of eyelid?
- 48. _____ Twitching other than eyelid?
- 49. _____ Hands feel shaky or tremble?
- 50. _____ Feel sore?

Date: _____

Time: _____

SYMPTOM INVENTORY RATING SCALE
PARENT FORM

The following list contains a number of symptoms that children may normally experience after having their tonsils taken out. Please indicate how severe each symptom has been for your child today by entering the number that best fits in the space beside each symptom. Please complete this list before you assist your child, and do not discuss it with him/her. For all items use this scale:

1	2	3	4	5
absent	a little bit	some	a lot	extreme

- | | | |
|-----|-------|---------------------------|
| 1. | _____ | Dizziness |
| 2. | _____ | Itching or painful eyes |
| 3. | _____ | Ringing in ears |
| 4. | _____ | Hard to hear |
| 5. | _____ | Lump in throat |
| 6. | _____ | Choking sensation |
| 7. | _____ | Sneezing spells |
| 8. | _____ | Runny nose |
| 9. | _____ | Stuffy nose |
| 10. | _____ | Bleeding nose |
| 11. | _____ | Sore nose |
| 12. | _____ | Asthma or wheezy |
| 13. | _____ | Out of breath |
| 14. | _____ | Hard to swallow |
| 15. | _____ | Sore throat |
| 16. | _____ | Hard to talk |
| 17. | _____ | Scratchy throat |
| 18. | _____ | Tiredness |
| 19. | _____ | Can't sleep |
| 20. | _____ | Toothache |
| 21. | _____ | Upset tummy |
| 22. | _____ | Pain or cramps in stomach |
| 23. | _____ | Heartburn |
| 24. | _____ | Sore muscles |
| 25. | _____ | Diarrhea |

1
absent

2
a little
bit

3
some

4
a lot

- 26. _____ Constipation
- 27. _____ Pain in throat
- 28. _____ Sore joints
- 29. _____ Stiff muscles
- 30. _____ Rash
- 31. _____ Itchy
- 32. _____ Popping in ear
- 33. _____ Warm ear
- 34. _____ Sore ear
- 35. _____ Pressure in ear
- 36. _____ Earache
- 37. _____ Stiff neck
- 38. _____ Weakness
- 39. _____ Pain in neck
- 40. _____ Feeling of pressure in head
- 41. _____ Headaches
- 42. _____ Feeling hot or sweaty
- 43. _____ Chills
- 44. _____ Feel like throwing up
- 45. _____ Throwing up
- 46. _____ Feeling faint
- 47. _____ Twitching of eyelid
- 48. _____ Twitching other than eyelid
- 49. _____ Hands feel shaky or tremble
- 50. _____ Feel sore

Name of Child: _____ Date: _____ Time _____

Days off School (since surgery) _____

Medication record (Pain killers, at home only):

Date _____	Time _____	Amt. _____
Date _____	Time _____	Amt. _____
Date _____	Time _____	Amt. _____

ADULT SYMPTOM INVENTORY

The following list contains a number of commonly-experienced symptoms or bodily sensations. Please indicate how often you experience each one by entering the appropriate number into the blank space beside each symptom. For all items, use the following scale:

1	2	3	4	5
Have never or almost never experienced the symptom	Less than 3 or 4 times per year	Every month or so	Every week or so	More than once every week

- | | | |
|-----|-------|--|
| 1. | | Eyes water |
| 2. | | Itching or painful eyes |
| 3. | | Ringing in ears |
| 4. | | Temporary deafness or hard of hearing |
| 5. | | Lump in throat |
| 6. | | Choking sensations |
| 7. | | Sneezing spells |
| 8. | | Running nose |
| 9. | | Congested nose |
| 10. | | Bleeding nose |
| 11. | | Asthma or wheezing |
| 12. | | Coughing |
| 13. | | Out of breath |
| 14. | | Swollen ankles |
| 15. | | Chest pains |
| 16. | | Racing heart |
| 17. | | Cold hands or feet even in hot weather |
| 18. | | Leg cramps |
| 19. | | Insomnia |
| 20. | | Toothaches |
| 21. | | Upset stomach |
| 22. | | Indigestion |
| 23. | | Heartburn |
| 24. | | Severe pains or cramps in stomach |
| 25. | | Diarrhea |
| 26. | | Constipation |
| 27. | | Hemorrhoids |
| 28. | | Swollen joints |
| 29. | | Stiff muscles |
| 30. | | Back pains |
| 31. | | Sensitive or tender skin |
| 32. | | Face flushes |
| 33. | | Severe itching |
| 34. | | Small bumps or spots in rash |
| 35. | | Acne or pimples on face |
| 36. | | Acne or pimples on other face |

1	2	3	4	5
Have never or almost never experienced the symptom	Less than 3 or 4 times per year	Every month or so	Every week or so	More than once every week

- 37. _____ Boils
- 38. _____ Sweat even in cold weather
- 39. _____ Strong reactions to insect bites
- 40. _____ Headaches
- 41. _____ Sensation of pressure in head
- 42. _____ Hot flashes
- 43. _____ Chills
- 44. _____ Dizziness
- 45. _____ Feel faint
- 46. _____ Numbness or tingling in any part of body
- 47. _____ Twitching of eyelid
- 48. _____ Twitching other than eyelid
- 49. _____ Hands tremble or shake
- 50. _____ Stiff joints
- 51. _____ Sore muscles
- 52. _____ Sore throat
- 53. _____ Sunburn
- 54. _____ Nausea

APPENDIX F

CHILDREN'S HEALTH LOCUS OF CONTROL

179.

Please circle YES or NO for each question.

- | | | |
|---|-----|----|
| 1. Good health comes from being lucky | Yes | No |
| 2. There is nothing I can do from getting sick | Yes | No |
| 3. Bad luck makes people sick | Yes | No |
| 4. I can only do what the doctor tells me to do | Yes | No |
| 5. Getting sick just happens | Yes | No |
| 6. People who never get sick are just plain lucky | Yes | No |
| 7. It is my mothers job to keep me from getting sick | Yes | No |
| 8. Only a doctor or a nurse keeps people from getting sick | Yes | No |
| 9. I can make very few choices about my health | Yes | No |
| 10. Accidents just happen | Yes | No |
| 11. I can do many things to flight illness | Yes | No |
| 12. Only the dentist can take care of my teeth | Yes | No |
| 13. The only way I can stay healthy is to do what other people tell me to do. | Yes | No |
| 14. I always go to the nurse right away if I get hurt at school. | Yes | No |
| 15. Its the teachers job to keep me from having accidents at school. | Yes | No |
| 16. I can make many choices about my health | Yes | No |
| 17. If I feel sick, I have to wait for other people to tell me what to do. | Yes | No |
| 18. Whenever I feel sick, I go to the school nurse right away. | Yes | No |
| 19. There is nothing I can do to have healthy teeth | Yes | No |
| 20. I can do many things to prevent accidents | Yes | No |

CONSENT

Mr. Leikin of the School of Psychology, University of Ottawa, and Drs Firestone, McGrath and Bernard of the Department of Psychology and Division of Otolaryngology of the Children's Hospital of Eastern Ontario are conducting a study to investigate the relationship between behavioral factors and children's experience with physical symptoms. Children undergoing a Tonsillectomy have been selected because they normally experience some physical symptoms following surgery. Prior to their surgery, children will have a brief visit with one of the researchers to complete an exercise task, and parents and teacher will be required to complete some questionnaires. For a one week period following surgery, all children and parents will complete another questionnaire while at home. In all, the study will take approximately 1.5 hours of time. There are no known negative effects nor any benefits associated with any of the questionnaires.

All information collected will be kept confidential and if the results are published the child will not be identified in any way. You may withdraw from this study at any time without affecting the availability of treatment to your child.

I am informed of, and agree to have my child, participate in this study.

Date

Signed

Witness

Relationship

I have explained the study to the patient and believe he/she understood it.

Name

Date

APPENDIX H

Distribution of Boys and Girls in Type A and B Groups (N = 85)

	Type A	Type B	
BOYS	n = 25 (29%)	n = 15 (18%)	n = 40
GIRLS	n = 18 (21%)	n = 27 (31%)	n = 45

APPENDIX I

Distribution of Young and Old Children in Type A and B Groups


(N = 85)

	Type A	Type B	
YOUNG	n = 30 (35%)	n = 25 (30%)	n = 55
OLD	n = 13 (15%)	n = 17 (20%)	n = 30

APPENDIX J

ANOVA With Repeated Measures Comparing Self-Report Post Surgery
Symptom Scores (SIRS-C) Across Type A/B, Sex, and Time

	SS	F	df	p
Type A/B	4701.7	10.68	1,83	.0016
Sex	1514.9	3.55	1,83	.063
Time	24268.3	93.72	2,83	.000
Type A/B X Sex	478.1	1.12	2,83	.29
Type A/B X Time	396.5	1.58	2,83	.20
Sex X Time	300.9	1.19	2,83	.30
Type A/B X Sex X Time	53.4	.21	2,83	.80

APPENDIX K

ANOVA With Repeated Measures Comparing Parent Reports of
Childrens Symptoms (SIRS-P) on Type A/B, Sex and Time

	SS	F	df	P
Type A/B	1959.9	71.52	1,82	.0076
Sex	.44	0.0	1,82	.96
Time	13361.8	160.9	1,82	.000
Type A/B X Sex	1.9	.01	1,82	.93
Type A/B X Time	324.7	3.91	1,82	.055
Sex X Time	16.9	.93	1,82	.65
Type A/B X Sex X Time	3.17	.04	1,82	.84

APPENDIX L

Effects of Age on Child Symptom Reports (SIRS-C)
and Parent Symptom Reports (SIRS-P)

	df	SS	F ^o	p
<u>SIRS-C</u>				
Age	1,83	2302.2	5.48	.02
Age X Type A/B	1,83	33.0	.08	.78
Age X Time	2,83	39.1	.15	.85
Age X Time X Type A/B	2,83	125.6	.49	.61
<u>SIRS-P</u>				
Age	1,82	1346.1	5.42	.02
Age X Type A/B	1,82	69.0	.28	.59
Age X Time	2,82	1.48	.02	.89
Age X Time X Type A/B	2,82	173.8	2.1	.15

APPENDIX M

Effects of Age on Weight Held and
Laboratory Fatigue for Type A Subjects

	SS	df	F	P
Weight	25508	1,41	29.8	.000
Time to 1	369.2	1,41	8.27	.007
Weight Covariate on Time to 1	2140	1,41	4.65	.04 ⁰
Time to 1 without Weight Covariate	157.4	1,41	3.42	.08

APPENDIX N

Effects of Parent Type A and Parent Symptom Reporting
on Children's Clinical Symptom Reporting.

	SS	df	F	P
<u>SIRS-C</u>				
Parent Type A/B	495.2	1,97	.85	.36
Parent PILL	3262.1	1,97	5.57	.019
PILL X Type A/B	589.9	1,97	1.01	.32
<u>SIRS-P</u>				
Parent Type A/B	101.8	1,98	.33	.57
Parent PILL	951.7	1,98	3.31	.07
PILL X Type A/B	54.8	1,98	.18	.67

APPENDIX O.

ANOVA and Duncan's Multiple Range Test Comparing Fatigue
Ratings of Parent's Type A/B, Lo/Hi PILL Groups

	SS	df	F	P
<u>ANOVA</u>				
Type A/B	214.03	1,92	1.89	.17
PILL	.768	1,92	.026	.871
Type A/B X PILL	188.56	1,92	6.47	.013

Duncan's Multiple Range

Between	(M.S.=104.5)	313.13	3		
				34.5	.019
Within	(M.S.=30.2)	2781.1	92		

Non-Homogeneous Type A-Lo PILL and Type B-Lo PILL (p < .05)
 Sub-Sets)

Type A-Lo PILL and Type A-Hi PILL (p < .05)

APPENDIX P

Effects of Health Locus of Control on Clinical Symptom Reports

	SS	df	F	P
SIRS-C				
Locus of Control	311.5	1,120	.54	.46
Locus X Time	293.1	2,120	1.37	.25
Locus X Type A/B	490.6	1,83	.69	.41
Locus X Type A/B X Time	56.7	2,83	.28	.75
SIRS-P				
Locus of Control	133.3	1,120	.44	.51
Locus X Time	372.8	1,120	4.6	.03
Locus X Type A/B	27.2	1,82	.11	.74
Locus X Type A/B X Time	145.1	1,82	1.9	.17

APPENDIX Q

Effects of Health Locus of Control on Fatigue Reports

	SS	df	<u>F</u>	<u>p</u>
Locus of Control	654	1,120	.17	.89
Locus X Type A/B	45.2	1,83	1.33	.25

i

ABSTRACT

Symptom under-reporting has been postulated as one mechanism by which the Type A behavior pattern exerts its influence as a major risk for coronary heart disease. The main purpose of this study was to examine the symptom reporting behavior in a cross-sectional sample of Type A and B children in order to extend knowledge about this risk mechanism and to study its developmental origins. A secondary purpose was to examine the relationship of childrens' symptom reporting to other health-related variables such as parental Type A and symptom reporting behaviors, symptom salience and health locus of control.

Eighty-five male and female children from 9 to 14 years of age were studied on both a laboratory fatigue induction task and in a clinical context of post-surgical recovery. Subjects were taken from the upper and lower tertile of the Type A/B distribution of a surgical sample. The results indicated that Type A children under-reported both types of symptoms relative to their Type B peers, as measured by self-report and parent ratings, and that in general this phenomenon was independent of age and sex. However, it was found that on some types of symptoms Type A under-reporting may increase with age. In addition, under-reporting was greatest among children whose parents were both Type A and under-reporters themselves. Parental reporting behavior was the most consistent predictor of childrens' symptom reporting across contexts. Regardless of Type A/B status children were found to demonstrate a characteristic symptom reporting style that was stable across

settings, symptom type and over time. The study found no evidence for the effects of symptom salience or health locus of control on childrens' reporting behavior.

The results were discussed in terms of their implications for the prevention of adult-onset heart disease and for general illness behavior in children. A serendipitous finding that Type A children were under-represented in the surgical sample, converged with their under-reporting style and suggests that these children may under-utilize medical services. The parallels in the illness behavior of Type A children and their adult counterparts implied a developmental pathway in risk factor development and one route of transmission was speculated. Specific intervention strategies using a social learning model were discussed. The results also had theoretical implications for the symptom reporting construct as well as clinical practice in health psychology.

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Finally, but most importantly, I thank my wife Penny. In fulfilling a dream, my own Type A behavior was neglectful of many family obligations. Yet, Penny stood by throughout with quiet strength and loving compassion. This dissertation is dedicated to her.

OK Penny, now I can change Ben's diaper!