CARDIO-RESPIRATORY RESPONSES TO MENTAL CHALLENGE:
HIGH, MODERATE, AND LOW HEART RATE REACTORS

By

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Abstract

Three issues were examined in this study: (1) the extent to which psychological challenge or stress elicits changes in the breathing patterns of normal subjects; (2) whether breathing pattern changes covary with cardiovascular arousal; and (3) whether individuals identified as being potentially at-risk for developing hypertension respond to mental challenge tasks with breathing pattern and cardiovascular changes that are reliably different from those of lower risk individuals. Subjects were 100 healthy young men divided into reactor quintiles on the basis of their heart rate (HR) changes to a 1-minute cold pressor test. Those in the upper quintile were designated as being at-risk for developing hypertension. Their cardiovascular and respiratory changes to two counterbalanced versions of a 5-minute mental arithmetic test (Easy & Hard) were compared with those of the third and fifth quintile subjects. Marked individual differences were evident in the direction and extent of breathing changes. Overall, the rate, amplitude, variability, and predominant mode of breathing increased substantially over resting levels in response to the math tasks. Only breathing rate and variability reliably covaried with task difficulty. Little correspondence was found between breathing changes and cardiovascular arousal. The data did indicate a trend for breathing to shift towards greater ribcage dominance as task difficulty increased. This was especially true for the at-risk group and least true for the low reactor quintile. The expected
group differences in cardiovascular reactivity were not found however, implying that the HR reactivity to cold stimulation is not a good predictor of reactivity to acute mental challenge or stress. Overall, the results suggest that breathing patterns change in response to psychological stress but are not clearly associated with cardiovascular arousal. The attempt to identify subgroups of aberrant breathers on the basis of HR reactivity also yielded equivocal results.
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A case will be made for quantifying stress-induced changes in the breathing patterns of a healthy young population and comparing these changes with cardiovascular indices of sympath­etic arousal. Of particular interest is the extent to which the predominant breathing mode of cardiac hyperreactors (subjects who show excessive heart rate increases to a cold pressor test) can be differentiated from those of low and moderate reactors. A review of the literature will focus on four central issues: (1) the lack of an adequate empirical rationale for teaching diaphragmatic breathing as a stress management technique; (2) the relevance of simultaneously assessing a variety of respiratory and cardiovascular responses to acute psychological stressors; (3) the types of respiratory measures included in previous stress studies; and (4) the utility of measuring the relative contribution to ventilation made by the thoracic and abdominal compartments. Following this review of the literature, information pertinent to the choice of experimental protocol will be presented, along with the hypotheses of this study.

Respiratory control training: Rationale and Empirical Support.

Voluntary modification of one's rate and depth of breathing is considered an important aspect of most relaxation and medita­tion procedures (Beary & Benson, 1974; Bernstein & Borkovec, 1973). The breathing pattern generally prescribed consists of
slower, deeper inspirations achieved primarily by abdominal excursion (Faling, 1986). This pattern reportedly involves a more efficient use of the primary muscle of inspiration, the diaphragm, than does thoracic-predominant breathing. It is therefore commonly referred to as diaphragmatic breathing (Tobin, 1986). Attenuation of autonomic arousal, and even the induction of a transient "hypometabolic state", have been attributed to the practice of diaphragmatic breathing (Fenwick, Donaldson, Gillis et al., 1977; Heide & Borkovec, 1983; Singh, 1984; Wallace & Benson, 1972; Wallace, Benson & Wilson, 1971).

However, there is as yet little empirical evidence to substantiate these initial claims (Holmes, 1984). In fact, several laboratory studies have failed to verify that respiratory control procedures can attenuate acute autonomic arousal during laboratory challenges (Cappo & Holmes, 1984; Harris, Katkin, Lick, & Habberfield, 1976; McCaul, Holmes, & Solomon, 1979). On the other hand, a number of treatment studies utilizing respiratory control training as a primary or auxiliary component of treatment have reported significant positive outcomes for a number of disorders characterized by autonomic over-arousal. These include idiopathic seizures (Fried, Rubin, Carlton, & Fox, 1984), panic attacks (Salkovskis, Jones, & Clark, 1986), hyperventilation (Lum, 1976) and hypertension (Bali, 1979; Datey, Deshmukh, Dalvi et al., 1969; English & Baker, 1983; Jacob, Kraemer, & Agras, 1977; Patel, 1977).
The discrepant findings of these two lines of research raise a number of important questions regarding the methods for inducing respiratory pattern change and how these respiratory changes impact other physiological systems. In particular, the mechanism needs to be clarified by which slow, diaphragmatic breathing would more effectively reduce somatic and/or subjective arousal than some other form of respiratory control or even the individual's natural pattern of quiet breathing. Understanding and clearly stating the physiological mechanism by which a particular strategy of respiratory control is believed to reduce arousal would seem essential to the success of future efforts at verifying and/or maximizing the clinical usefulness of diaphragmatic breathing.

A necessary first step in determining whether or not any breathing control strategy can effectively reduce arousal is to establish that different patterns of breathing occur at different levels of arousal. In other words, the rate, depth, and/or predominant mode of breathing (i.e. thoracic versus abdominal) during high arousal states should differ in characteristic ways from the pattern observed during periods of low arousal or relaxation. If such differences are found, the rationale for voluntary alterations of breathing patterns will appear more tenable. To test for arousal-related differences in respiration, samples of breathing patterns should be obtained across a variety of conditions, including quiet resting, responding to psychological stressors, or engaging in physical exercise.
To date, only a handful of well-controlled psychophysiological studies have looked at respiratory changes under these conditions (e.g. Allen, Sherwood, & Obrist, 1986; Carroll, Turner, & Hellawell, 1986; Suess, Alexander, Smith et al., 1980; Svebak, Dalen, & Storfjell, 1981; Turner, Carroll, & Courtney, 1983). Only one study has investigated changes in the amplitude of ribcage and abdominal excursions during short periods of psychological stress (Svebak et al., 1981). No study has as yet looked at the relative contribution that either mode of breathing makes to overall ventilation in stressful situations. In addition, the cardiovascular correlates of stress-induced respiratory changes have typically been limited to heart rate and blood pressure. Other measures of cardiovascular change, such as T-wave amplitude and blood volume pulse, would add to our knowledge of how the cardiovascular and respiratory systems interact during periods of mental or physical stress.

Why study acute stress responses?

At present, psychophysiologists are only beginning to appreciate the importance of cardio-respiratory interactions in reactions to acute stressors. In the past, investigators focused almost exclusively on cardiovascular and subjective responses to short-term psychological stressors in their attempts to understand the physiological processes involved in the etiology of chronic cardiovascular disorders such as essen-
tial hypertension and coronary heart disease (Krantz & Manuck, 1984).

The importance of studying physiological responses to acute psychological stressors has its basis in the increasing body of evidence linking psychological and behavioral factors to disease. According to one model, excessive physiological responsivity to emotional stress might be a marker of some underlying pathological process or processes involved in the development of essential hypertension or coronary heart disease. While it can be argued that potentially pathological reactions to chronic stress conditions are more complex and variable than acute responses, there is evidence to suggest that reactions to acute stressors are related to the development of chronic disorders. It has been found, for instance, that some individuals exhibit a characteristic, temporally stable hyperresponse of heart rate and blood pressure to certain tasks and situations (Allen, Obrist, Sherwood, & Crowell, 1986; Elliot, Buell, & Dembroski, 1982; Frankish & Linden, 1985; Light, 1981; Obrist, 1981; Schulte & von Eiff, 1985; Wood, Sheps, Elveback, & Schirger, 1984). There is some overlap between this hyperreactivity subgroup and the offspring of hypertensive parents (Hastrup, Light, & Obrist, 1982; Light, 1981; Manuck & Proietti, 1982; Warren & Fischbein, 1980). Based on epidemiological evidence, the latter group carries a substantially greater risk of developing essential hypertension than do individuals with normotensive parents.
According to Steptoe (1985), the fact that hyperreactivity to certain stress tasks appears to precede the development of stable hypertension is one of the most important considerations in determining which task(s) to include in one's study. He notes that challenging, problem-solving tasks have consistently elicited excessive systolic blood pressure increases in hypertensives and borderline hypertensives, and that such increases are sustained primarily through augmented heart rate and cardiac output. Increased heart rate and systolic blood pressure are known to reflect strong beta-adrenergic influences on the heart (Obrist, 1981; Sherwood, Allen, Obrist, & Langer, 1986). Furthermore, heightened sympathetic reactivity has been implicated in the onset of the hypertensive process. Because of their ability to provoke sympathetically-mediated cardiac changes, tasks that require subjects to actively engage in some form of coping behaviour (e.g. mental arithmetic, video games, reaction time tests to avoid shock or earn monetary bonuses) can therefore be useful in analyzing the psychophysiological processes in essential hypertension. However, as yet, no longitudinal studies have been completed which directly link acute autonomic hyperreactivity to the development of hypertension or coronary heart disease.

While the predictive validity of active coping task responses is still not known, this is not the case for cardiovascular reactivity to the cold pressor test, a form of physical
challenge. In a 45-year follow-up study of 142 school children originally tested in 1934, Wood et al. (1984) found that children whose systolic or diastolic blood pressure responses to the cold pressor were in the 90th percentile or higher showed a much higher incidence of hypertension later in life than did normoreactors. Other studies have shown that individuals with essential hypertension similarly respond to cold pressor tests with significantly larger increases in blood pressure than normotensives do (Buhler, Bolli, Hulthen et al., 1983; Greene, Boltax, Lustig et al., 1965).

From a physiological standpoint, it is not clear why blood pressure reactivity to the cold pressor should be predictive of future hypertension in individuals who are presently normotensive. Schulte & von Eiff (1985) reported that males with resting blood pressure levels in the borderline hypertensive range do not show the exaggerated systolic blood pressure reactivity that established hypertensives show in response to a cold pressor challenge. They also found few differences between normotensive, borderline, and essential hypertensive men in the degree of heart rate, stroke volume, and peripheral resistance changes to the cold pressor test. In contrast, hypertensives showed significantly greater blood pressure, heart rate, and cardiac output reactivity to a mental arithmetic task than did the other two groups. Such findings led Schulte and von Eiff to conclude that increased reactivity during beta-adrenergic cardiac stimulation is one of the critical markers of hyper-
tension pathogenesis, and that only tests which provoke predominantly cardiac reactions would prove useful in predicting the development of hypertension. However, in light of Wood et al.'s results—one of only two completed prospective studies linking some form of laboratory stress response with risk for later hypertension—this conclusion is rather tenuous. The cold pressor test that Wood and his associates found so useful for predicting hypertension development has its primary effect on the peripheral vasculature rather than on cardiac output. If, as Schulte and von Eiff maintain, acute beta-adrenergic reactivity is the important predictor of hypertension development, how can one explain the strong correlation between excessive blood pressure reactivity to the cold pressor and later development of hypertension? Clearly, at this stage in the research, it is premature to conclude that hypertension development proceeds by one specific psychophysiological pathway.

Other lines of research suggest that the stability of cardiac hyperreactivity across stress conditions and over time may be an important factor in the development of hypertension. Light (1981), for instance, found that fully 50% of the normotensive men showing the highest heart rates during a shock avoidance reaction time test also had the highest heart rates during a cold pressor test. She also found that men with a family history of hypertension had significantly higher heart rates at rest and in response to a reaction time task than did
men with normotensive parents. Furthermore, subjects with the largest systolic blood pressure increases to a psychological stressor also showed the largest heart rate increases. A similar finding was reported by Frankish & Linden (1985). In this study, a 100% overlap was found between the systolic blood pressure reactivity and heart rate change of subjects in the top tercile of reactivity to mental arithmetic.

Overall, research suggests that cardiovascular hyperresponsivity is affected by predisposition and type of stimulus eliciting the response (Faulstich, Williamson, McKenzie et al., 1986; Linden, 1985). According to Manuck, Krantz and Prolefrone (1986), stimulus factors such as task difficulty, performance incentives, and stimulus novelty all can affect the pattern and magnitude of cardiovascular stress response. To the extent that the cardiovascular changes induced by laboratory stress tasks reflect levels occurring in the challenges of daily life, they may prove to be useful predictors of cardiovascular disorders.

Why measure respiration during stress?

The rationale for measuring respiratory activity during acute stressors has not been as well developed as that for recording stressor-induced cardiovascular changes. Nevertheless, there are many reasons for doing so. Perhaps the most important of these is the fact that the respiratory and cardiovascular systems interact in the critical task of maintaining
gas exchange between the atmosphere and body tissues. Together, they ensure that the levels of oxygen, carbon dioxide, and blood pH throughout all tissues are kept within the narrow parameters necessary for the continuation of normal tissue functioning (Sheperd & Vanhoutte, 1979). Both systems interact at a number of levels.

Respiratory-cardiovascular interactions.

Presently, four types of mechanisms have been uncovered by which respiration can modulate cardiovascular activity. These include; (1) mechanical effects of inspiration on venous return to the heart; (2) lung inflation receptors which reflexively trigger heart rate acceleration during inspiration; (3) chemoreceptors in the carotid and aortic bodies sensitive to changes in oxygen and carbon dioxide tensions; and (4) interconnections between the brain stem centers that control respiration and cardiovascular activity via descending efferent fibers. The interaction of these four mechanisms must be considered when analyzing cardiovascular responses (Grossman, 1983; Shepherd, 1981).

The action of these mechanisms on heart and blood vessel functioning is observed primarily through five respiratory parameters. Breathing rate is one of these parameters. Together with respiratory depth or tidal volume, it determines the volume of air ventilating the lungs in a given period of time. At resting levels, healthy adults breathe 12 - 17 times
or cycles per minute (cpm) (Linden, In press; Tobin, Chadha, Jenouri et al., 1983). Within certain limits, breathing rate has been found to covary with heart rate and be inversely related to heart heart rate variability (Angelone & Coulter, 1964; Hirsch & Bishop, 1981; Porges, McCabe & Yongue, 1982; Sroufe, 1971). Maximum heart rate variability (also known as respiratory sinus arrhythmia) occurs at a breathing rate of 6 cpm: it quickly and steadily decreases as respiration rate increases. Secondly, increased depth of respiration results in increased heart rate and heart rate variability, along with decreased blood flow to hands and feet (Shepherd & Vanhoutte, 1979; Sroufe, 1971). Manzotti (1958) showed that heart rate changes are directly proportional to intrathoracic pressure: increased pressure associated with inspiration results in increased heart rate. Thirdly, pauses between inspirations and expirations are known to produce a pronounced and rapid drop in heart rate, with a concomitant increase in blood flow to the heart and brain (Daly, Angell-James, & Elsner, 1979; Hirsch & Bishop, 1981; Hurwitz, 1981). The decrease in heart rate apparently results from the spontaneous activity of parasympathetic vagal efferents that remain silent during inspiration. In contrast, inspiration is associated with sympathetic fibre firing and consequent heart rate increases (Porges et al., 1982). The ratio of inspiration time to total respiratory cycle time (Ti/Ttot) is also related to sympathetic nervous system activity (Leischow & Allen, 1986; Nochomovitz, Supinski & Kelsen, 1986). Lengthy inspirations, particularly of thoracic
origin, require greater muscular exertion that consequently results in increased cardiac output. Fourthly, one's mode of breathing - either the predominance of thoracic or abdominal excursions - has a notable effect on cardiac output. Hurwitz (1981) found that when the depth and predominant mode of inspirations were voluntarily and independently manipulated, greater increases in heart rate and peripheral vasoconstriction occurred with thoracically dominant inspirations than with abdominal-diaphragmatic breathing. The cardiac cycle T-wave amplitude (TWA), a rough index of sympathetic nervous system control of the myocardium, was also found to be greater during abdominal than during thoracic breathing (Hurwitz, 1981). Finally, the concentration of carbon dioxide in the blood is a critical factor in cardiovascular regulation, and is controlled primarily via the rate and depth of respiration. A drop in the arterial concentration of this potent vasodilator, brought on by overbreathing (i.e. breathing in excess of the body's acute metabolic requirements), can result in vasoconstriction and decreased oxygen availability. Increased heart rate and cardiac output, T-wave flattening or inversion as well as peripheral vasoconstriction and dilation of muscle tissue vessels have been observed with hyperventilation (Grossman, 1983).

The preceding list of respiratory influences on cardiovascular activity is by no means exhaustive. It does, however, indicate the interrelatedness of the two systems. By implication, it also suggests that both respiratory and cardiovascular
parameters should be measured if one is to adequately study physiological stress responses.

Cardio-respiratory measures of stress.

A study by Suess, Alexander, Smith et al. (1980) illustrates the importance of recording several measures of cardio-respiratory activity during psychological stress. The stated purpose of the study was to accurately characterize the extent of stress-induced hyperventilatory responses in normal individuals. To this end, 29 undergraduates (13 female) completed a perceptual judgement task under the threat of electric shock for suboptimal performance. Recordings of carbon dioxide output, respiration rate, heart rate, and self-reported anxiety were obtained both before and during the stress task. A significant pre-task decrease in end-tidal carbon dioxide levels (indicative of hyperventilation) occurred concomitantly with increased heart rate and subjective anxiety levels. Only heart rate showed further significant change from pre-task to task. Respiration rate, on the other hand, was considered an inadequate measure of respiratory system response to stress because of its low correlation with end-tidal carbon dioxide levels. Respiratory mechanisms other than rate must therefore have been acting to increase the amount of carbon dioxide leaving the lungs. Suess et al. concluded that future efforts to document respiratory system reactivity must include additional measures of respiration change, such as tidal volume,
oxygen consumption, and length of inspiration.

Recent studies that did include comprehensive measures of cardio-respiratory activity have yielded some potentially important findings. Carroll et al. (1986) and Turner et al. (1983), for instance, have found that cardiac output during mentally challenging tasks can exceed metabolic demands, a finding which suggests that cardiac and metabolic functioning can become dissociated during periods of psychological challenge or stress. In the latter study, 24 young males played two versions of a "Space Invaders" computer game while their heart rate and various respiratory parameters were monitored. Relative to baseline levels, heart rate was found to be much greater during "Space Invaders" than during the control condition. In comparison, the task-induced increases in respiratory rate, depth of breathing (tidal volume), breathing volume per minute (minute ventilation), oxygen consumption or uptake and carbon dioxide production were considered modest although significant. A significant difference in the latter two respiratory measures prevented the investigators from concluding that an overall cardiac-metabolic independence had occurred during the game. However, the fact that a post-hoc analysis revealed no differences between high and low heart rate reactors in either oxygen uptake or carbon dioxide production suggests that the stressor-induced heart rate changes experienced by the high reactors were metabolically exaggerated.
Carroll et al. reported similar findings in a study comparing heart rate and respiratory responses during two mental challenges (mental arithmetic and Raven's matrices) that varied in level of difficulty (easy, hard, impossible). Physiological activity was also recorded while subjects engaged in graded isotonic exercise on a bicycle ergometer. From this information, regression equations were derived for each subject relating heart rate to oxygen consumption across three exercise loads. These regression equations were then used in conjunction with oxygen uptake values obtained during mental arithmetic and Raven's matrices to predict task-elicited heart rate values for each subject. The difference between actual and predicted heart rate was designated "additional heart rate" or metabolically unjustified heart rate. For most subjects, predicted heart rate values were considerably less than actual heart rate values. Analysis of these "additional heart rate" scores revealed a significant effect for task difficulty, with smaller values occurring during the easy condition compared with the hard and impossible conditions. Conversely, no such difficulty effect was obtained for most measures of respiration change. However, these measures did show significant changes with time on each task, unlike the measure of additional heart rate.

Considering the significant effect of task difficulty on heart rate, but the lack of such effect for respiratory indices of metabolic demand, it appears that psychologically challenging tasks can elicit heart rate increases unrelated to energy
expenditure. In addition, such increases are paralleled by subjects' self-reports of active engagement and arousal in the task. The apparent cardiac-metabolic independence is not, however, a global response. This is evident from the fact that, while heart rate and oxygen uptake both declined significantly with time on task, no such time effect was evident for "additional heart rate". The impact of psychological and physical challenge on respiratory variables was also found to vary considerably. For instance, whereas increased minute ventilation during exercise was a function of increases in both tidal volume and respiration rate, changes in ventilation during psychological challenge were largely a function of tidal volume variations. Respiration rate, on the other hand, was the only respiratory variable influenced by the level of task difficulty. To summarize, Carroll et al. found cardiac-respiratory covariation to be a complex phenomenon: heart rate variations with task difficulty appear to be independent of changes in oxygen consumption, carbon dioxide output, and overall ventilation, but they covary with breathing rate.

Simultaneous recording of both cardiovascular and respiratory measures during acute psychological and physical challenge was also done by Allen et al. (1986). Comprehensive measures of physiological change in response to a twominute cold pressor, a five-minute reaction time task with the threat of electric shocks for suboptimal performance, and three load levels on a bicycle ergometer (five minutes at each load) were
obtained for 22 male undergraduates. The cardiovascular measures included heart rate, cardiac output (the beat-by-beat product of heart rate & stroke volume), pre-ejection period, and systolic and diastolic blood pressure. Indices of respiration change included respiratory rate, tidal volume, minute ventilation (the product of tidal volume & respiration rate), oxygen uptake, and end-tidal carbon dioxide. The means for Minutes 5 and 10 of a 15-minute adaptation period served as the baseline values for calculating change scores for each dependent measure. Single means were also calculated for each dependent measure from the minute-to-minute values recorded during each of the five conditions.

As expected, the one-way (conditions) ANOVAs executed for each measure showed that increasing exercise load yielded significant differences on all variables except diastolic blood pressure. Responses to the reaction-time and cold pressor tasks, however, varied across the different physiological indices. For instance, cardiac output was significantly greater, and pre-ejection period significantly less, during the reaction-time task than during the cold pressor. This task difference was attributed to increased beta-adrenergic activity in response to the reaction-time/threat condition. In general, cardiovascular responses to the stress task differed substantially from the responses observed when subjects were resting quietly.
Differences in the pattern of respiratory changes induced by the reaction-time and cold pressor tasks were also found. For instance, only the cold pressor elicited a significant above-baseline increase in tidal volume. Respiratory rate, on the other hand, was greater during the reaction-time task than during the cold pressor. Although the difference was not statistically significant, respiration rate in both conditions significantly exceeded baseline levels. As expected from the above findings, the total volume of air inspired per minute was significantly greater for the cold pressor task than for the reaction-time task, with both tasks producing significantly higher ventilation levels than occurred during baseline. Also as one might expect from this pattern of results, end-tidal carbon dioxide decreased significantly more during the cold pressor than during shock threat. In fact, subjects were apparently hyperventilating during the cold pressor. Oxygen uptake, however, did not differ across the two conditions although it was significantly higher in both conditions relative to baseline.

In order to further assess the relationship between cardiovascular and respiratory variables during stress, Allen et al. computed regression equations for each subject, much as Carroll et al. (1983) had done. In this case, however, regression equations based on rest and exercise values were computed for four different pairs of variables: 1) cardiac output/minute ventilation; 2) cardiac output/oxygen uptake; 3)
heart rate/minute ventilation; and 4) heart rate/oxygen uptake. The extent to which each of these cardio-respiratory pairings showed an "uncoupling" during the cold pressor and reaction-time/threat conditions could then be seen by looking at whether or not these two means lay outside of the 95% confidence interval for the respective regression line. Any significant deviation from the linear relationship at rest and during exercise was seen as evidence for cardio-respiratory uncoupling.

Just as Carroll et al. had found, heart rate and oxygen uptake showed an uncoupling during the two stressors. A nonlinear relationship between cardiac output and minute ventilation was also observed during the cold pressor. During the reaction-time task, a similar nonlinearity of responding occurred for cardiac output and oxygen uptake. Allen et al. concluded on the basis of these findings that the relationship between heart rate and minute ventilation is less susceptible to disruption by behavioral stressors than are the relationships involving cardiac output and oxygen uptake.

Because of substantial individual differences observed in the cardiovascular and respiratory parameters during the reaction-time and cold pressor tasks, Allen et al. also decided to examine the responses of extreme ventilation reactors and nonreactors to both stressors. A further purpose was to assess the impact these differences might have on other variables. Controlling for observed order effects in responses to the two
stress conditions, they executed two-factor repeated measures ANOVAs (reactor group X time on task) on each of the physiological measures. Reactor groups were based on the 3 highest and 3 lowest ventilation reactors in each task order, for a total of 6 subjects per reactor group. It was noted that only three subjects were common to the hyperreactor groups of both tasks, and three to the low reactor groups.

The results of these analyses revealed that ventilation hyperreactors had significantly higher minute ventilation and cardiac output values during the cold pressor than did the low reactors. This difference was due primarily to larger tidal volumes for the hyperreactors rather than significantly greater respiratory rates. Oxygen uptake was also substantially higher for the hyperreactors at the first minute of the cold pressor, but dropped off considerably between Minutes 1 and 2. During the reaction-time task, hyperreactors also showed significantly greater changes in carbon dioxide exhalation and oxygen uptake values than low reactors. In general, it was observed that those subjects who showed substantial minute ventilation increases and end-tidal carbon dioxide decreases during either of the two stressors also responded with exaggerated cardiac output and pre-ejection period changes associated with beta-adrenergic activity. Allen et al. concluded from these results that some of the sympathetic effects on cardiac functioning seen during their behavioral stressors may be due to hyperventilation.
The importance of the findings of Allen et al. and Carroll et al. extend beyond their respective conclusions about the existence of metabolically unjustified heart rate increases during acute psychological challenge. The fact that they measured changes in respiration as well as cardiovascular parameters such as heart rate is at least as important, since without such measures "additional heart rate" could not have been calculated. The respiratory measures also provide a more complete picture of how different individuals respond to psychological challenge. Using larger sample sizes than those used by the previous investigators, it may be possible to identify subgroups that show aberrant or excessive stress responses across a number of physiological systems, including the respiratory and cardiovascular systems. Identifying a subgroup of individuals whose respiration during stress deviates significantly from relaxation patterns and whose heart rate reactivity classifies them as being at risk for coronary heart disease or essential hypertension has important implications for treatment. To the extent that voluntary slowing and/or deepening of one's breathing can produce a concomitant decrease in heart rate, it may be considered an important treatment for such hyperreactors.

Respiratory control research.

A number of empirical studies have attempted to demonstrate
that voluntary control of one's breathing pattern can attenuate autonomic and self-reported arousal to acute psychological stress. These studies were conducted in response to the claims by meditation enthusiasts and behavioural therapists alike that the practice of diaphragmatic breathing could reduce arousal in both systems. In general, the results were not encouraging.

One of the earliest studies to specifically test the effect of respiration control on autonomic responses to psychological stress was conducted by Harris, Katkin, Lick and Habberfield (1976). In their study, 14 male undergraduates were asked to maintain a constant depth of respiration while matching their respiratory rate to the pace set by a flashing light. The other 28 subjects were either given no instructions regarding the pacing light (Baseline control) or instructed to press a switch on every tenth light flash (Attention control). A 10-minute practice period was allotted for each condition. Heart rate and skin conductance levels were monitored during four subsequent periods in which subjects waited for a signalled electric shock. One painful 2-second shock was administered after the first 60-second warning tone.

These investigators found that subjects who voluntarily reduced their breathing rate to match the pace set by a timing light during a 10-minute practice period showed significantly smaller increases in skin conductance levels during pre-shock and shock expectation periods than did subjects in either
no-treatment or attention control groups. Although such a finding suggests that paced respiration can attenuate autonomic arousal, no measures of respiration rate and tidal volume were obtained to verify that subjects had actually adopted the specified breathing pattern. Also questionable was the high heart rate mean (90.0 bpm) observed for paced breathing subjects at post-practice. In contrast, the mean heart rate for attention control and baseline control subjects was 84.1 bpm and 84.9 bpm respectively. This finding suggests that attempting to slow one's breathing rate down to approximately one-half of normal resting levels is arousing rather than relaxing.

Holmes, McCaul and Solomon (1978) reported similar findings in a study comparing idiosyncratic respiratory control with no-treatment and attention control conditions. In their study, 111 male and female students sat quietly for 30 minutes while a polygraph tracing of their respiratory rate and amplitude was obtained. Subjects spent the next five minutes either: (1) sitting quietly, (2) hand-copying their own respiration tracing from Minutes 25 - 30 of the adaptation period, or (3) attempting to reproduce this tracing pattern in vivo. After this, 50% of the subjects in each condition were led to expect a signalled electric shock in a 90-second interval. The remaining subjects received no threat of shock.

No group differences in heart rate were found for this latter condition. In the threat condition, however, subjects
who previously copied their adaptation phase respiratory tracing by hand or in vivo showed significantly greater heart rates and self-reported anxiety than subjects in the no-treatment group. One conclusion from these results is that newly learned coping strategies which require concentration, such as controlled breathing, increase rather than decrease autonomic activity, possibly because they interfere with one's usual, and apparently more effective, methods for coping with threat. The fact that the controlled breathing group showed increased arousal in the threat condition relative to resting subjects hardly seems surprising considering how little time subjects had to practice such an unnatural maneuver (subjects had 10 minutes to practice the control strategy). Although the investigators recognized this problem, they further undermined the manipulation's potential effectiveness by deciding not to instruct subjects to maintain the practiced breathing pattern during the stressor period.

In a more recent study, Cappo and Holmes (1984) attempted to redress some of the weaknesses of earlier breathing control studies by: (1) including a larger number of dependent measures, (2) recording the physiological changes that took place while subjects practiced one of three different breathing techniques, and (3) including attention and no-treatment control groups. The three different breathing techniques were fast-slow, slow-fast, and equivalent length inspirations and expirations. After 5 minutes of practice with a special pacing clock, subjects were
told to maintain the practiced breathing pattern until the end of the experiment. One-half of the subjects in each of the five conditions were then told to expect a signalled electric shock sometime following a 90-second "wait period". Conditions were identical in the no-threat condition except that no mention was made of electric shocks.

The results of this study confirmed Holmes et al.'s (1978) finding that altering one's breathing pattern to match a predetermined pattern, especially one in which subjects are to breath at roughly half their resting rate, increases rather than decreases arousal levels. All three breathing strategies were ineffective in reducing cardiovascular and self-reported arousal to threat although there was a trend for the fast inspiration-slow expiration method to contain the perception of arousal at pre-arousal levels.

Some general conclusions can be drawn regarding these three laboratory studies of controlled breathing. First, altering one's respiration rate and/or length of inspiration to some predetermined level increases arousal rather than limits it. However, in each of these studies practice of the control strategy was far too brief to fairly assess the potential of the various breathing techniques for attenuating arousal. Second, it is not clear why the different control conditions were expected to further reduce arousal levels after subjects had been resting quietly for up to 30 minutes. Floor effects may
have masked any beneficial effect the breathing techniques might have had. Third, the degree of arousal induced by the stress conditions was minimal and therefore inadequate for testing the efficacy of the breathing control procedures. Fourth, respiration activity during breathing control practice and the subsequent threat/no-threat periods was not measured; therefore, it is not known how accurately subjects performed the assigned breathing manipulations nor is it known whether or not the breathing patterns of trained subjects differed from control subjects during the anticipation and threat conditions. Without these data, the validity of these studies is open to question.

Why measure thoracic and abdominal movement?

The importance of monitoring changes in breathing mode has long been recognized by physiologists (e.g. Konno & Mead, 1968; Sharp, Goldberg, Druz et al., 1976). Psychophysiologicals, on the other hand, have paid relatively little attention to such changes, although there are some notable exceptions (e.g. Helbick, 1981; Svebak et al., 1981).

One reason for including measures of thoracic and abdominal excursion when studying stress responses is that they represent the body's principal method for replenishing its supply of oxygen and eliminating carbon dioxide. According to Konno and Mead (1967), the thorax or ribcage and the abdomen constitute the two moving parts or components of the respiratory system.
Furthermore, each of these parts moves independently of the other in the process of ventilating the lungs. Typically, however, thoracic and abdominal movement appear to be coupled to some extent during inspiration and expiration (Hirsch & Bishop, 1981). Underlying this apparent ribcage-abdomen coupling is the action of the diaphragm, the primary muscle of respiration.

The diaphragm is a dome-shaped sheet of muscle separating the abdominal and thoracic compartments. At its periphery, it inserts into the lower ribs, forming a zone of apposition (Nochomowitz, Supinski, & Kelsen, 1986). When contracted, as during inspiration, the diaphragm pushes downward on the contents of the abdomen. Since the abdominal viscera can be compressed very little, the downward force of this contraction causes both the abdominal and ribcage walls to displace outward. Contraction of the external intercostals, a second group of inspiratory muscles, also results in an outward displacement of the ribcage. This outward expansion of the ribcage and/or abdomen generates a negative intrathoracic pressure that results in lung inflation. Expiration is essentially the reverse process. Relaxation of respiratory muscles allows the elastic forces of the inflated lungs to return the lung to its resting volume, forcing air out in the process (McLaughlin, 1977; West, 1974).

To the extent that the abdominal wall is compliant (i.e. able to move outward), inspiration can be considered pre-
dominantly abdominal in origin. As the abdominal muscles contract, however, the abdominal wall becomes less compliant and inspiratory movement shifts towards ribcage predominance. The dominance of either source of intrathoracic pressure change is what is referred to by the term "breathing mode". Whatever the dominant breathing mode, volume changes in both respiratory compartments are a function of the pressure exerted by the associated respiratory muscles (Grimsby, Goldman, & Mead, 1976).

Because of the way in which the two respiratory compartments interact in the process of breathing, one can obtain a noninvasive, reliable estimate of inspiration volume, otherwise known as tidal volume. This is done by taking the sum of volume changes in both the ribcage and abdomen (Tobin, 1986). Dual silastic strain gauges or dual transducer bands of the respiratory-inductive plethysmography are used to obtain these data, one attached around the ribcage and the other around the abdomen. The output of the two strain gauges or transducer bands must be calibrated against the output of a spirometer, the traditional means for recording changes in respiratory rate and volume. While this process can be complicated and may require extensive subject training, Tobin argues that it is worth the additional effort because of evidence that the invasive spirometer apparatus (subjects breath into a mouthpiece or face mask) causes tidal volume to increase and respiratory rate to decrease relative to noninvasive methods (Gilbert, Auchincloss, Brodsky et al, 1972; Mead, 1960). Such findings call into
question the reliability of the respiration data obtained in recent stress response studies which used either mouthpieces or masks to obtain spirometer recordings (e.g. Allen et al. (1986); Carroll et al. (1986)). Spirometry poses additional problems for stress research in that the breathing apparatus precludes the use of stress tasks involving speech.

Evidence for different breathing modes.

The relative contribution of the ribcage and abdomen to breathing in normal subjects was extensively investigated by Sharp et al. (1975) using noninvasive monitoring devices known as respiration magnetometers. One magnetometer was attached to the subject's chest to measure anteroposterior distance changes associated with respiratory excursions. A second device fixed to the abdomen provided data on the amplitude of abdominal excursions during breathing. Recordings from both magnetometers were obtained as 81 male and female subjects of various ages participated in six breathing maneuvers. These maneuvers included relaxation (deep inspirations followed by slow expiration to functional residual capacity), quieting breathing, increasing tidal volume with each successive breath, forced inspiration and expiration, quiet speech, and loud speech. Because the magnetometers are sensitive to postural changes, each maneuver was done twice, once in the supine position and a second time with subjects seated upright. By dividing the thoracic data by the sum of thoracic and abdominal amplitude
scores, a measure of the relative motion of each respiratory compartment was obtained.

Sharp et al. found that for a given maneuver, breathing patterns varied somewhat across subjects of the same age and gender. Nevertheless, some patterns were far more common than others. For instance, in the relaxation-supine condition, 81% of the young men and 93% of the young women showed thoracic predominant breathing when tidal volumes (i.e., amplitudes) were small; with larger tidal volumes, abdominal displacement exceeded ribcage displacement. Essentially the reverse pattern was observed when subjects breathed quietly while seated upright. For 91% of the men and 83% of the women, breathing was accomplished almost exclusively by thoracic excursion, especially at lower tidal volumes. Given that the volume change or amplitude of a given compartment reflects the compliance of that compartment, these findings suggest that the ribcage is more compliant than the abdomen-diaphragm when subjects perform a deep breathing-relaxation maneuver in the upright position.

When subjects were asked to simply rest quietly, the effect of posture on the ratio of thoracic displacement to total tidal volume displacement was again apparent. In the supine position, thoracic amplitude accounted for 25% 4% (men) to 28% 4% (women) of the tidal volume. When seated upright, 72% 3% of the tidal volume in young men and 61% 4% in women was attributable to ribcage displacement. Age and sex had little
impact on breathing mode in comparison to the effect of posture. Nearly all subjects were abdominal breathers when supine and thoracic breathers when upright.

Greater inter-subject variation in breathing mode occurred when subjects purposely increased their depth of inspiration, breathed forcefully, or spoke aloud. Once again, breathing in the upright posture was predominantly thoracic for most individuals, and less so in the supine position. Age and gender, however, were generally unrelated to the marked differences in breathing pattern. Other factors, possibly of a psychological nature (e.g. learned anxiety responses), must therefore be considered as potential sources for these differences (see Haas (1980) for correlational data linking personality characteristics with respiratory behavior). Sharp and his colleagues did not attempt to uncover what these factors might be. They did note though that the ribcage muscles are capable of more rapid action than the diaphragm and abdominal muscles and that consequently thoracic excursions should be expected to predominate where breathing is deep and/or rapid.

Thoracic-abdominal stress responses.

Svebak et al. (1981) demonstrated that breathing mode does change in response to one type of acute psychological challenge. In their study, 13 male undergraduates played two counterbalanced versions (easy and hard) of a video game while changes in their heart rate, forearm EMG, skin conductance and
respiration were measured. Recording of breath by breath changes in both the circumference and amplitude of the ribcage and abdominal compartments were made possible by fastening one mercury-filled strain gauge around each subject's chest and another around his abdomen. By comparing the degree of amplitude change in each respiratory compartment, an estimate of breathing mode change could be obtained.

Analysis of the EMG data revealed that EMG levels had increased gradually during both versions of the 150 second long video game. Similar increases were found for respiration rate and thoracic circumference. In contrast, abdominal circumference decreased throughout the task. A different pattern of results emerged for respiration amplitude. Thoracic and abdominal excursions both dropped to their lowest levels at the beginning of the game performance, the difficult game eliciting the largest drop in amplitudes, especially for abdominal amplitude. By task's end, however, the scores for both game versions had increased to within approximately 90% of baseline levels.

On the basis of these findings, Svebak et al. concluded that respiratory changes associated with acute psychological challenge parallel increases in somatic activity. Furthermore, the roughly monotonic increase in physiological activity occurring throughout this task took place only for skeletal muscle and not for the viscera (i.e. heart rate and skin
conductance). In contrast, these latter measures reached their highest levels shortly after the task began. Such results suggest that a performance-related dissociation of cardiac functioning and somatic activity occurs in tasks where outcomes can be positive or negative, depending on the individual's performance. Of greater relevance, they provide preliminary evidence that acute stressors can induce changes in the circumference and amplitude of the two independent components of the respiratory system. An effective means for measuring respiratory changes via dual strain gauges is also demonstrated.

Unfortunately, the significance of these findings is limited by the small sample size (n = 13). Given the considerable range of possible respiratory and autonomic responses, the existence of reactor subgroups could not be determined with this sample. As mentioned earlier, identifying individuals who show hyperresponses on a number of physiological measures, including respiratory mode, will provide a more convincing data base for teaching breathing control techniques in attempts to reduce exaggerated stress responses.

Conclusions and hypotheses.

In the preceding review, a rationale was developed for assessing both respiratory and cardiovascular changes in response to acute psychological challenge. It was first emphasized that sympathetically mediated reactivity of the
cardiovascular system to laboratory stressors may possibly be a predictor of cardiovascular disease. An outline of the main mechanisms by which various respiratory parameters alter cardiac and blood vessel functioning provided some initial support for measuring stress-induced respiratory changes concomitantly with cardiovascular responses. Further support came from studies by Allen et al. (1986) and Carroll et al. (1983) which demonstrated the value of including comprehensive cardio-respiratory measures in stress research. After briefly considering how ventilation changes are accomplished physiologically and how traditional assessment methods tend to alter breathing responses, it should be clear that the respiratory data reported in these studies are inadequate. Monitoring changes in the amplitude and/or circumference of the respiratory system's two independent compartments, the chest and the abdomen, was considered as an alternate source of information on breathing responses. Svebak et al.'s (1981) study is of particular relevance in that it demonstrates that these latter measures are sensitive not only to the effects of psychologically challenging tasks but also to the level of task difficulty. Given that breathing patterns shifted towards greater thoracic dominance throughout the challenging tasks, these findings provide some initial support for teaching diaphragmatic breathing as a technique for reducing arousal.

One purpose of the present study is to attempt to replicate Svebak et al.'s findings using a larger subject sample and a
different task of longer duration. By doing so, the extent to which breathing pattern changes and cardiac hyperreactivity covary in normal subjects should become clearer. The inclusion of additional measures of autonomic and somatic arousal, such as T-wave amplitude, blood volume pulse, pulse transit time, and respiratory movement variability, will provide further, as yet untapped, information on cardio-respiratory interactions during psychological challenge. Another measure included in this study which previous laboratory stress studies have not assessed is the task-induced change in thoracic excursions relative to abdominal excursions. The one other study to include this measure (Sharp et al., 1975) found it to be a useful index of breathing mode variations. Based on the Svebak et al. data, it might be expected that breathing mode will shift towards thoracic dominance with the onset of stress tasks.

The difficulty level of a challenge task like mental arithmetic may also have a differential effect on breathing mode given Svebak et al.'s finding that easy and hard versions of their active coping task (a video game) elicited significantly different levels of ribcage and abdominal amplitude. In fact, it can be argued that, in comparison to a video game stressor, easy and hard versions of a mental arithmetic task represent a better test of breathing mode sensitivity to stress load. Little physical exertion is required in mental arithmetic tasks; therefore, response differences between difficulty conditions are likely to be the result of cognitively mediated arousal
rather than by-products of increased muscular activity.

A second purpose of this study is to determine whether or not individuals whose cardiac adjustments to a cold pressor test may be considered excessive relative to other subjects also respond to subsequent mental challenge tasks with significantly different respiratory and cardiovascular changes than those experienced by low or moderate heart rate reactors. On the basis of Wood et al.'s (1983) findings, the high reactor group may be considered to be at greater risk for developing hypertension or coronary heart disease. Research by Allen et al. (1986) also suggests that hyperreactors to a cold pressor test breathe more deeply and/or more rapidly than hyporeactors. In addition, a considerable proportion of the cold pressor hyperreactors also experienced the greatest cardiovascular and respiratory arousal to a shock avoidance reaction time task. Whether or not such findings can be replicated with a larger sample of healthy young males remains to be seen. Furthermore, it needs to be demonstrated that this apparent cardiorespiratory covariation occurs under different levels of task difficulty and across different types of active coping tasks. The results of several of the studies reviewed here suggest that active coping to avoid aversive consequences or gain monetary bonuses causes an uncoupling of the cardiac and respiratory responses which ordinarily operate in synchrony to meet metabolic requirements (e.g. Carroll et al., 1983; Svebak et al., 1981). The possibility exists that such uncoupling of otherwise
parallel responses in the two systems is limited to a subset of subjects (Allen et al., 1986; Turner et al., 1981). Unfortunately, the sample sizes in these post-hoc comparisons were unacceptably low, ranging from n = 3 to n = 6. A study that employs a larger sample size would clear up this ambiguity. To the extent that cardiac hyperresponders also have a thoracically dominant breathing pattern during stress, they may benefit from training to breathe diaphragmatically prior to and/or during times of stress.

In summary, the following questions are addressed in the present study:

1) To what extent does an active coping task like mental arithmetic affect multiple indices of respiratory activity, including breathing rate, amplitude of ribcage and abdominal excursions, the variability of such excursions (i.e. breathing rhythmicity), and the predominant mode of breathing (either thoracic or abdominal)? In addition, are these changes sensitive to difficulty manipulations?

2) Are the respiratory measures mentioned above sensitive to adaptation with continued performance of a mental arithmetic task?

3) To what extent do task-elicited respiratory changes
correspond to changes in several indices of cardiovascular arousal, including heart rate, T-wave amplitude, blood volume pulse, and pulse transit time?

4) Are task-elicited changes in cardio-respiratory activity associated with subjective and behavioural indices of task difficulty?

5) If meaningful reactor subgroups can be formed on the basis of subjects' heart rate adjustments to an initial cold pressor test, will these subgroups differ significantly from each other in terms of the cardiovascular and respiratory changes experienced in response to easy and hard versions of a mental arithmetic task? In essence, the question being asked is "Do changes in breathing mode covary with expected group differences in cardiovascular reactivity?".
Method

Subjects

One hundred male undergraduates participated in the study for course credit. Subjects ranged in age from 18 to 30 (mean = 20.9, SD = 0.33). Prior to participation in the study, all subjects were instructed to avoid strenuous physical activity and the use of tobacco, alcohol and coffee for two hours preceding the experiment session (Jennings, Berg, Hutcheson et al., 1981). Due to recording equipment difficulties, complete data were available for only 92 of the 100 subjects.

The decision to employ only male subjects was based on two considerations. First, previous studies in this area have used male subjects almost exclusively and, in order to directly compare findings, a similar population was required. Second, using subjects of both genders could pose certain problems in data collection and interpretation. For instance, several investigators have reported gender differences in respiratory rate, amplitude and/or predominant mode of respiration (e.g. Linden, in press; Svebak, 1975). In order to facilitate interpretation of the results, and also to avoid the need for separate analyses for each sex (which would reduce the power of the test), it was decided to use only males.

Apparatus

Electrocardiographic (ECG) activity was recorded with
bipolar recording electrodes placed on lateral positions of the lower ribcage. A ground electrode was also attached to the back of the subject's neck. This configuration is known to yield relatively large T-waves and produces an ECG signal vectorially equivalent to standard lead I (Constant, 1981). The ECG signal was processed via a cardiotachometer coupler (Sensormedics, Model 9857) in the "Direct" position in order to obtain complete ECG signals. The cardiotachometer filter was set at 30 Hz with a standard ECG amplification of 50 uv/mm. These settings were recommended in the manual for the coupler and have been found in prior studies to generate the cleanest ECG signals. Blood volume pulse (BVP) was monitored via a Sensormedics photoplethysmograph which was placed on the tip of the index finger of the nondominant hand and then interfaced with a voltage/pulse/pressure coupler (Sensormedics, Model 9853A). A filter setting of 30 Hz and standard amplification of 5 uv/mm was used for this signal.

The method for measuring respiratory activity was based in part on the methods used by Svebak et al. (1981) and Linden (1986). In order to obtain recordings of both thoracic and abdominal amplitude changes, two Bellows strain gauges were attached to each subject, one around the chest at the level of the armpits and the other located at the level of the abdominal umbilicus. Resistance changes from one strain gauge were fed into a Sensormedics voltage/pulse/pressure coupler (Model 9853A) set at 10 uv/mm of amplification with a filter setting of 3 Hz.
Output from the second strain gauge was channeled into a Beckman strain gauge coupler (Model 9872) set at 100 uv/mm of amplification with a filter setting of 30 Hz. The two strain gauge/coupler pairings were counterbalanced with the two respiratory compartments to avoid a possible confound resulting from undetermined differences in the sensitivity and/or signal processing of the two couplers. For both couplers, the filter and signal gain settings were those that yielded the cleanest and most interpretable polygraph tracings in pilot testing.

The couplers used for both the cardiovascular and respiratory measures were integrated with a 4-channel Sensormedics Dynograph, Model R611, interfaced with an analogue-to-digital (A/D) converter and a Victor 9000 microcomputer. Signal processing software written for this system was set to sample data in 30 second intervals at predetermined times throughout the experiment. Heart rate was calculated on the basis of interbeat intervals, defined as the length of time between successive R-wave peaks. The peak voltage reading between successive R-wave peaks defined T-wave amplitude (TWA). Pulse transit time (PTT) scores were obtained by computing the time delay between each R-wave peak and the corresponding blood volume pulse peak. All cardiovascular data from the signal processing program were stored on a 10 megabyte hard disk and on real-time hard copy printouts from an interfaced high speed printer. Since only one respiratory signal at a time could be processed by the waveform analysis
software, data from both strain gauges were recorded on polygraph paper for manual, post-experimental analysis. The cardiac and BVP signals were also recorded on the polygraph paper to permit cross-validation checks.

**Procedure**

Throughout the experiment, subjects were seated upright in a recliner chair. This situation minimized extraneous body movements and changes in posture that otherwise might have affected the cardiovascular and respiratory recordings.

Upon entering the lab, subjects were asked to complete a brief questionnaire on their age, height, weight, smoking habits, frequency of physical exercise, family history of hypertension, and personal history of respiratory and cardiovascular disorders (see Appendix A). Any individuals suffering from asthma (symptomatic), respiratory infection, or pulmonary hypertension were excluded from the study because the respiration rate, tidal volume, and minute ventilation of such individuals differ markedly from those of normal subjects (Tobin, 1986). A brief overview of the experiment and physiological response apparatus was then given to subjects (Appendix B), after which they were asked to read and sign a consent form (Appendix C). Next, the ECG electrodes were placed on skin abraded and cleaned with rubbing alcohol. Finally, the ribcage and abdomen strain gauges were fastened around the subject. In order to ensure accurate recordings of thoracic and
abdominal amplitude changes over the entire range of tidal volumes, the position of these gauges was checked by having subjects breath in and out deeply one or two times. If either gauge shifted position, it was readjusted and rechecked. Cables from all devices were fed through a hole in the wall to the neighboring recording room.

A 10-minute adaptation period followed in which subjects were encouraged to relax and read Herman cartoons. No specific task instructions were given until the end of the adaptation period. This procedure has previously been found to facilitate adaptation to the laboratory while at the same time limiting the effect that anticipation can have on physiological response adaptation (Linden, McEachern & Frankish, 1985). The final minute of the adaptation period served as the baseline for the first stressor task.

Immediately following the adaptation period, detailed instructions for the first stress task (the cold pressor test) were provided to subjects (see Appendix D for a script of these instructions). During the last minute of a 10 minute recovery period, a second baseline recording was obtained. Detailed instructions for the mental arithmetic task were then given (Appendix E). The order of the two mental arithmetic trial blocks (easy math and hard math) was counterbalanced across subjects. Subjects were randomly assigned to task orders. A 5-second break separated the two trial blocks. Following the
second block of equations, subjects were given a further five minutes to relax before a final recording was obtained. They then completed a brief post-experimental questionnaire (Appendix F) before removal of the recording apparatus. Subjects were debriefed and thanked for their participation (see Appendix G for the debriefing script).

To summarize the procedure followed in this study, subjects first participated in a 10-minute adaptation period followed by a 1-minute cold pressor test, a 10-minute recovery period, two 5-minute blocks of arithmetic problems, and finally a 5-minute recovery period. In all cases, the cold pressor task preceded the arithmetic task. For the two math task conditions, 50% of subjects were presented with the easy equations first while the remaining 50% responded to the hard problems first. This procedure approximates a within-subject multiple treatment cross-over design: each of the three tasks (cold pressor, easy math, and hard math) constitutes a treatment condition, with only the latter two conditions being counterbalanced across subjects.

**Experimental Tasks**

**Cold Pressor.**

All subjects were asked to immerse their nondominant hand for one minute in a four-liter container filled with 4 - 6 Celsius ice water after being told that other subjects had
successfully completed this task. Previous studies have found that this task elicits significant increases in such indices as blood pressure, cardiac output, tidal volume, and respiratory rate (e.g. Allen et al., 1986). Other investigators have also reported considerable increases in peripheral resistance during this task (Murakami, Hiwada, & Kokubu, 1980).

Mental Arithmetic.

The mental arithmetic task consisted of two 5-minute long blocks of arithmetic equations pre-recorded on videotape and presented to subjects by video monitor. Findings from a pilot study (n=10) confirmed that one block of problems was difficult and the other easy: the difficult problems elicited heart rate increases approximately twice as large as those elicited by the easy equations. The 30 easy problems involved the addition, subtraction, multiplication, or division of one-digit and two-digit numbers. The 30 difficult problems involved the same mathematical operations but with two- and three-digit numbers. Previous research using a similar difficulty manipulation for mental arithmetic has shown that, on average, the more difficult problems elicit significantly greater autonomic responses than do easier ones (Carroll et al., 1983).

During the easy and hard arithmetic blocks, the respective arithmetic equations were displayed on a 51 cm video screen for 10 seconds each. A stopwatch function located in the lower left corner of the screen simultaneously marked the passage of time.
to 1/100ths of a second accuracy (Hitachi video recorder, Model VT-7P with an RCA videocamera, Model CC017). Subjects were instructed to write down their answers to each problem before the onset of the next equation. A prepared recording sheet, divided into two sections numbered from 1 to 30, was provided for this purpose. The importance of concentrating on the task was emphasized since the problems presented to the subjects by video were not numbered. As a further inducement to concentrate subjects were told that approximately 90% of other subjects had correctly answered at least 75% of the problems and that their own performance would be reviewed with them at the conclusion of the session.

Self-report indices on the perceived difficulty of the two mental arithmetic conditions were obtained immediately following the task. A 15 cm visual analog scale with labelled endpoints was used for this purpose (Appendix H).

Data Reduction

Samples of autonomic activity occurring over 30-second time intervals were obtained at the following times: (1) Minute 10 of adaptation (cold pressor baseline); (2) seconds 30-60 of the cold pressor task; (3) seconds 570-600 (Min. 10) of the cold pressor recovery period (mental arithmetic baseline); (4) seconds 30-60 (Min. 1), 150-180 (Min. 3) and 270-300 (Min. 5) of Easy and Hard mental arithmetic; and (5) seconds 270-300 (Min. 5) of the final recovery period. For all sampling times except
the final baseline, the raw data (beat to beat or breath to breath recordings) for each subject were averaged to yield single mean scores for the following variables: heart rate, T-wave amplitude, blood volume pulse, pulse transit time, respiration rate, thoracic amplitude and its variability, and abdominal amplitude and its variability.

A sixth measure of respiratory activity, the ratio of thoracic to abdominal amplitude (T:A), was also computed for each of the 10 sampling times outlined above. By definition, the amplitude of thoracic and abdominal excursions equalled the mean inspiration-expiration change in strain gauge resistance within a given measurement period. Respiratory amplitude variability was defined as the standard deviation of breath by breath amplitude changes for each recording period. The relative nature of these amplitude and amplitude variability scores meant that, for across-subject comparisons of task-induced changes, the raw data needed to be expressed as percentages of the appropriate baseline values. Obviously, mean amplitude, amplitude variability, and T:A scores could not be reported for the two baseline periods.

Before computing mean scores from the raw data, all data files were inspected for artifacts associated with extraneous body movements and recording equipment errors. Movement artifacts were defined as sudden, very large increases in respiration amplitude noted on the polygraph chart tracings by
the experimenter at the time of recording. Data associated with these artifacts were omitted from further analyses. Computer-generated errors in the recording of cardiovascular activity were more difficult to identify. For most subjects, the waveform analysis program produced only one type of error, that is, impossibly large heart rate estimates for the first second of the recording period. These errors were easily identified: they occurred because the program had no previous R-wave peak with which to compare the current R-wave peak. Comparison of polygraph ECG tracings with their respective digital data printouts revealed that for a minority of subjects, particularly those with very small S-waves, the program-generated output occasionally appeared to be erroneous. Whenever the digital recordings of blood volume pulse, pulse transit time and/or T-wave amplitudes deviated by 100% or more from both (a) their previous and subsequent values in a given recording interval and (b) their analogue counterparts on the polygraph tracing, the data were deleted. Extensive data editing was necessary for less than 15% of subjects and, within this subgroup, for fewer than 50% of the recording periods.
Results

Analysis of the response data will be reported in two major sections: (1) cardiovascular, respiratory, and subjective responses of all subjects to the two mental arithmetic conditions; (2) mental arithmetic responses of subjects in the first, third and fifth quintiles of heart rate reactivity to the cold pressor challenge.

In the first section, four questions will be addressed. First, to what extent do breathing mode, rate of breathing, the amplitude of ribcage and abdominal excursions, and the variability of these excursions vary as a function of the experimental task and the difficulty levels of that task? Second, what is temporal pattern of respiratory changes during such tasks? Third, do respiratory changes correspond to simultaneously occurring changes in cardiovascular indices of arousal? Finally, to what extent are task-related changes in cardiovascular and respiratory functioning correlated with subjective and behavioural assessments of task difficulty?

Analyses in the second section were devised to determine whether or not individuals in the upper, middle, and lower quintiles of heart rate reactivity to the cold pressor test also responded differentially to the subsequent mental challenge tasks. Of particular interest was whether or not changes in breathing mode covaried with expected group differences in
cardiovascular reactivity.

I. Mental Arithmetic Responses - All Subjects

The mean change scores for each physiological measure at each recording period of the two mental arithmetic conditions are presented in Figures 1 and 2. Mean change scores and standard deviations for the two difficulty conditions and for the three recording periods are presented in Tables 1 and 2. For three dependent measures (heart rate, breathing rate, and pulse transit time) change scores were computed as the difference between the task and baseline raw scores. For the remaining seven measures, this method of computing change scores was inappropriate since the raw scores included an unknown amount of error variance associated with individual differences in physiology and recording equipment settings. In order to compare response levels across subjects, each subject's task responses were expressed as percentages of the respective baseline values. A summary table of raw baseline means and standard deviations is included in Appendix I for those interested in comparing data collected in our laboratory with data collected by other investigators.

The analysis began by executing a 2 (Task difficulty) X 3 (Recording period) MANOVA (BMDP:4V) that included change scores for all 10 physiological measures. The multivariate test results for the Task ($F(10, 82) = 13.29, p < .001$) and Period
Table 1
Average change scores for Easy and Hard mental arithmetic (MA).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Easy MA</th>
<th>Hard MA</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (bpm)**</td>
<td>6.4 (7.0)</td>
<td>12.0 (10.0)</td>
</tr>
<tr>
<td>TWA (%)</td>
<td>-4.7 (10.8)</td>
<td>-6.3 (14.7)</td>
</tr>
<tr>
<td>BVP (%)**</td>
<td>-29.0 (33.6)</td>
<td>-34.7 (34.6)</td>
</tr>
<tr>
<td>PTT(msec)**</td>
<td>-11.9 (20.2)</td>
<td>-16.5 (21.4)</td>
</tr>
<tr>
<td>RR (cpm)**</td>
<td>2.3 (3.2)</td>
<td>4.2 (3.6)</td>
</tr>
<tr>
<td>T:A (%)</td>
<td>15.5 (54.6)</td>
<td>21.1 (56.2)</td>
</tr>
<tr>
<td>RA (%)</td>
<td>5.3 (33.0)</td>
<td>7.5 (34.8)</td>
</tr>
<tr>
<td>AA (%)</td>
<td>-0.4 (28.9)</td>
<td>-0.8 (33.2)</td>
</tr>
<tr>
<td>RAV (%)</td>
<td>86.3 (216.7)</td>
<td>89.8 (202.6)</td>
</tr>
<tr>
<td>AAV (%)*</td>
<td>41.7 (127.5)</td>
<td>72.3 (181.3)</td>
</tr>
</tbody>
</table>

F(10, 82) = 5.77, p < .001 factors confirmed that further analyses for Task and Period main effects were appropriate. A Type I error risk of .05 was adopted for each of the univariate ANOVAs. Testing for significant Period main effects was conducted multivariately to guard against violations of the sphericity assumption (Vasey & Thayer, 1987). Whenever a significant Period main effect was observed, post-hoc comparisons of recording period mean scores were executed using Scheffe's method (p < .05). Due to the nonsignificant multivariate test result for Task x Period interactions (F(10, 82) = 1.58, p = .08), significant univariate interactions were re-analyzed using the Greenhouse-Geisser correction for degrees of freedom.

Two of the six respiratory change indices were found to vary significantly in response to the easy and hard arithmetic conditions. Breathing rate changes to hard math were found to be significantly greater than those occurring to the easy task
(F(1, 91) = 58.60, p < .001). On average, the hard task elicited roughly twice as great an increase in breathing rates as did the easy condition (+4.2 cpm versus +2.3 cpm respectively). In comparison to breathing rates at baseline, the mean rates during each of the math tasks were also found to be significantly higher than resting levels (F(2, 90) = 58.5, p < .001). Variability in the amplitude of abdominal excursions was also significantly greater during the harder of the two arithmetic tasks (F(1, 91) = 5.75, p = .02). Once again, the ratio of change was approximately 2:1 in favour of the hard task. Task-related differences in abdominal and ribcage amplitude, ribcage amplitude variability, and breathing mode were not found to be significant, suggesting that these indices of respiratory change are insensitive to variations in stress load.

When differences between recording periods were analyzed, significant main effects were observed for changes in breathing rate (F(2, 90) = 3.26, p < .05) and ribcage and abdominal amplitude (F(2, 90) = 3.92, p < .05 and F(2, 90) = 7.79, p < .001 respectively). Post-hoc comparisons indicated that Minute 1 values for all three respiration indices differed significantly from their respective change scores at Minutes 3 and 5, while scores for these latter two periods did not differ reliably from one another (see Table 2). The amplitude of ribcage movements clearly decreased over time: from an average above-baseline increase of +10.5% at Minute 1, ribcage amplitudes decreased to +5.1% at Minute 3 and +3.6% at Minute 5. Abdominal amplitude,
Table 2

Average change scores for the three recording periods of mental arithmetic (MA).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Minute 1</th>
<th>Minute 3</th>
<th>Minute 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (bpm)***</td>
<td>11.4 (11.1)</td>
<td>7.8 (8.3)</td>
<td>8.4 (7.1)</td>
</tr>
<tr>
<td>TWA (%)**</td>
<td>-6.9 (14.2)</td>
<td>-4.9 (11.8)</td>
<td>-4.9 (12.7)</td>
</tr>
<tr>
<td>BVP (%)***</td>
<td>-36.9 (33.4)</td>
<td>-28.6 (34.2)</td>
<td>-30.1 (34.5)</td>
</tr>
<tr>
<td>PTT (msec)**</td>
<td>-16.4 (21.2)</td>
<td>-14.0 (19.7)</td>
<td>-12.2 (21.4)</td>
</tr>
<tr>
<td>RR (cpm)*</td>
<td>3.5 (3.5)</td>
<td>3.1 (3.3)</td>
<td>3.0 (3.7)</td>
</tr>
<tr>
<td>T:A (%)</td>
<td>18.3 (59.6)</td>
<td>17.8 (50.5)</td>
<td>18.9 (56.0)</td>
</tr>
<tr>
<td>RA (%)*</td>
<td>10.5 (38.7)</td>
<td>5.1 (32.1)</td>
<td>3.6 (30.0)</td>
</tr>
<tr>
<td>AA (%)***</td>
<td>3.0 (31.9)</td>
<td>-2.1 (30.0)</td>
<td>-2.8 (31.2)</td>
</tr>
<tr>
<td>RAV (%)</td>
<td>100.2 (264.1)</td>
<td>70.0 (158.9)</td>
<td>94.0 (191.7)</td>
</tr>
<tr>
<td>AAV (%)</td>
<td>51.3 (129.7)</td>
<td>54.3 (138.9)</td>
<td>65.5 (195.7)</td>
</tr>
</tbody>
</table>

*p < .05, **p < .01, ***p < .001

on the other hand, showed an initial average increase of 3.0% from baseline levels followed by a -2.1% decrease at Minute 3 and a further decrease to -2.8% by Minute 5. In other words, the amplitude of respiratory movements varied in response to relatively brief mental challenges, increasing to their highest levels within 2 minutes of task onset and decreasing thereafter. However, unlike ribcage amplitude, which remained above baseline levels for the duration of the math tasks, the average amplitude of abdominal excursions decreased to below baseline levels some time after the first minute or two and remained below baseline for the remainder of the task. Measures of breathing variability and the ratio of ribcage to abdominal amplitude change
did not show significant variation during task performance.

The effect of task difficulty on heart rate, T-wave amplitude, blood volume pulse, and pulse transit time change appears to have been more consistent across subjects than it was for respiratory change. Whereas only two of the six respiratory variables varied significantly in response to the easy to hard mental arithmetic, three of the four cardiovascular measures showed a significant main effect for task difficulty. Heart rate increases to the hard math condition (mean = +12.0 bpm) clearly exceeded the mean increase of +6.4 bpm elicited by the easy math problems (F(1, 91) = 101.27, p < .001). Significant task main effects were also obtained for blood volume pulse (F(1, 91) = 9.86, p < .01) and pulse transit time (F(1, 91) = 16.07, p < .001). On average, the difficult math problems elicited a larger decrease in blood volume pulse waves (-34.7%) and pulse transit times (-13.9 msec) than did the easy equations (-11.9 msec and -28.6% respectively). A similar response pattern was apparent for T-wave amplitude but was not statistically significant (F(1, 91) = 3.20, p = .08). Taken together, these data indicate that the harder of the two math conditions elicited the greatest increase in autonomic arousal.

Analysis of cardiovascular changes over time also revealed a marked degree of response consistency across subjects not evident for respiratory changes. Significant main effects for recording periods were noted for all four cardiovascular
measures: 1) heart rate ($F(2, 90) = 26.78, p < .001$; 2) T-wave amplitude ($F = 5.17, p < .01$; 3) blood volume pulse ($F = 23.03, p < .001$; and 4) pulse transit time ($F = 5.06, p < .01$). Post-hoc analyses of the heart rate change data revealed that across both difficulty conditions heart rate declined significantly from the first (+11.4 bpm) to the third minute (+7.8 bpm) of mental arithmetic and then remained stable until Minute 5 (mean = +8.4 bpm). Comparison of the recording period change scores for the other three cardiovascular measures also revealed that the response levels recorded at Minutes 3 and 5 did not differ reliably from one another. However, at Minute 1 response levels were significantly lower than they were at any other time during the task. Such findings are consistent with the breathing rate and ribcage amplitude changes in suggesting that adaptation to the task occurred within the second to fourth minute of responding.

From the pattern of results described above, it appears that cardiovascular functioning and, to a lesser extent, respiratory activity vary both as a function of time on a mental challenge task and the difficulty level of that task. As a means of determining the extent to which task-elicited changes in heart rate were related to respiratory changes, stepwise linear regressions were executed for each arithmetic condition. Bivariate scatterplots obtained prior to this analysis suggested that the indices of respiratory change were linearly related to heart rate changes.
In the easy math condition, two respiration variables accounted for 25.8% of the variance in heart rate changes. Approximately 14.7% of the variance was attributable to changes in breathing rate while the remaining 11.1% was associated with ribcage amplitude change. A further 14.3% of the overall variance was accounted for by two cardiovascular measures, T-wave amplitude and pulse transit time. In all, 40.1% of the heart rate variance in the easy condition could be accounted for. Of this total, approximately 64% was associated with respiratory changes. In contrast, only 19.2% of the variance in heart rate changes to the hard task could be attributed to respiratory changes. Abdominal amplitude variability, the first respiratory variable to enter the regression equation following pulse transit time and T-wave amplitude, contributed to 3.4% of the variance. A further 3.6% was attributable to breathing rate changes. Overall, 36.5% of heart rate variance was accounted for by the nine cardiovascular and respiratory measures.

From these analyses, it would appear that heart rate responses under increasing psychological load (stress) become dissociated to some extent from respiration adjustments. To the extent that respiration changes reflect metabolic activity or need, heart rate during the hard arithmetic condition appears to be exaggerated.

Further information on cardio-respiratory interactions
Table 3
Correlations between easy (E) and hard (H) mental arithmetic change scores: all physiological measures.

<table>
<thead>
<tr>
<th></th>
<th>T:A</th>
<th>RA</th>
<th>AA</th>
<th>RAV</th>
<th>AAV</th>
<th>RR</th>
<th>HR</th>
<th>TWA</th>
<th>BVP</th>
</tr>
</thead>
<tbody>
<tr>
<td>RA-E</td>
<td></td>
<td>0.63*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RA-H</td>
<td></td>
<td>0.54*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AA-E</td>
<td></td>
<td>-0.68*</td>
<td>-0.03</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AA-H</td>
<td></td>
<td>-0.78*</td>
<td>-0.07</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RAV-E</td>
<td></td>
<td>0.20</td>
<td>0.35*</td>
<td>-0.06</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RAV-H</td>
<td></td>
<td>0.12</td>
<td>0.28</td>
<td>0.01</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AAV-E</td>
<td></td>
<td>-0.11</td>
<td>0.11</td>
<td>0.24</td>
<td>0.54*</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>AAV-H</td>
<td></td>
<td>-0.15</td>
<td>0.19</td>
<td>0.29</td>
<td>0.56*</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>RR-E</td>
<td></td>
<td>-0.18</td>
<td>-0.29</td>
<td>0.08</td>
<td>-0.17</td>
<td>-0.12</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RR-H</td>
<td></td>
<td>-0.09</td>
<td>-0.20</td>
<td>-0.16</td>
<td>-0.22</td>
<td>-0.20</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR-E</td>
<td></td>
<td>0.17</td>
<td>0.27</td>
<td>0.09</td>
<td>0.20</td>
<td>0.15</td>
<td>0.34*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR-H</td>
<td></td>
<td>0.07</td>
<td>0.11</td>
<td>0.07</td>
<td>0.12</td>
<td>0.27</td>
<td>0.20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TWA-E</td>
<td></td>
<td>-0.01</td>
<td>0.04</td>
<td>0.01</td>
<td>0.01</td>
<td>0.18</td>
<td>-0.11</td>
<td>-0.33*</td>
<td></td>
</tr>
<tr>
<td>TWA-H</td>
<td></td>
<td>-0.01</td>
<td>-0.06</td>
<td>-0.07</td>
<td>0.01</td>
<td>-0.04</td>
<td>-0.08</td>
<td>-0.36*</td>
<td></td>
</tr>
<tr>
<td>BVP-E</td>
<td></td>
<td>-0.20</td>
<td>-0.29</td>
<td>-0.01</td>
<td>-0.09</td>
<td>-0.08</td>
<td>-0.16</td>
<td>-0.37*</td>
<td>0.18</td>
</tr>
<tr>
<td>BVP-H</td>
<td></td>
<td>-0.17</td>
<td>-0.19</td>
<td>0.07</td>
<td>-0.07</td>
<td>-0.08</td>
<td>-0.07</td>
<td>-0.27</td>
<td>0.17</td>
</tr>
<tr>
<td>PTT-E</td>
<td></td>
<td>0.12</td>
<td>0.10</td>
<td>-0.16</td>
<td>0.00</td>
<td>-0.10</td>
<td>-0.22</td>
<td>-0.33*</td>
<td>0.22</td>
</tr>
<tr>
<td>PTT-H</td>
<td></td>
<td>0.06</td>
<td>-0.02</td>
<td>-0.14</td>
<td>-0.11</td>
<td>-0.21</td>
<td>-0.10</td>
<td>-0.44*</td>
<td>0.27</td>
</tr>
</tbody>
</table>

* Significant at p < .001

during the two mental arithmetic conditions was obtained by correlating the mean change scores for each cardiovascular and respiratory measure within each difficulty condition. The resulting correlations are presented in Table 3. In response to the easy math task, only one respiratory measure, breathing rate change, correlated significantly with any measure of cardiovascular arousal, in this case heart rate change (r = .34). With an increase in task difficulty this relationship weakened
Heart rate change also showed moderate correlations with ribcage amplitude and its variability during the easy math problems but not during the hard condition. The reverse was true for abdominal amplitude variability. Correlations of .15 and .27 were obtained with heart rate changes to the easy and hard tasks respectively. The only other cardio-respiratory correlation to approach significance was between blood volume pulse and ribcage amplitude. Once again, the hard condition was associated with the strongest correlation (r = .29 versus r = .19 for the easy task).

In comparison to the moderate to weak correlations obtained for task-induced changes in cardiovascular and respiratory activity, the correlations among the respiratory measures were noticeably greater. Variability in the amplitudes of ribcage and abdominal movements, for instance, was found to be highly correlated in both difficulty conditions (r = .54 or greater). This suggests that people who breathe irregularly with their abdomen also do so with their ribcage. The amplitude of movements in each respiratory compartment also correlated moderately well with their respective variability measures (r = .24 or greater). Breathing rate, on the other hand, correlated moderately and negatively with all indices of amplitude and amplitude variability. To some extent, increases in breathing rate were associated with decreases in the magnitude of respiratory excursions and their variability. The more difficult the task, the more marked this relationship
appears to have been for all variables except ribcage amplitude and T:A ratio. In fact, changes in the abdominal and ribcage amplitudes to both math conditions were virtually unrelated. Such a finding might be expected for two relatively independent compartments. Contrary to this finding, the large correlations found between each amplitude measure and its composite index, T:A ratio, suggests that a strong inverse relationship exists between them.

The extent to which subjective and behavioral assessments of task difficulty corresponded with task-related changes in cardio-respiratory activity was assessed in two ways. First, two one-way ANOVAs were executed to determine whether or not the difficulty manipulation had yielded significantly different error rates and perceptions of task difficulty across the entire sample of subjects. The results of these analyses confirmed that the hard math condition was judged to be more difficult ($F(1, 99) = 19.91, p < .001$) and elicited more incorrect answers ($F(1, 99) = 150.94, p < .001$) than the easy condition. Mean scores for task performance and perceived difficulty are presented in Table 4. Second, correlations between the subjective, behavioural, and physiological change measures were examined for significant relationships among these variables. No significant correlations were found. However, the perceived difficulty level of the hard task did show a moderate inverse relationship with task performance ($r = -0.28$). In other words, the higher the difficulty rating, the fewer correct responses
Table 4
Performance levels and ratings of task difficulty Easy and Hard arithmetic.

<table>
<thead>
<tr>
<th>Arithmetic Condition</th>
<th>Correct Responses</th>
<th>Difficulty Rating **</th>
</tr>
</thead>
<tbody>
<tr>
<td>Easy</td>
<td>29.4 (5.4)</td>
<td>48.4 (37.2)</td>
</tr>
<tr>
<td>Hard</td>
<td>20.8 (5.9)</td>
<td>73.8 (37.6)</td>
</tr>
</tbody>
</table>

Maximum score is 30.

Difficulty ratings based on 150 mm visual analog scale with anchor points 0 (Not at all difficult) and 150 (Very difficult).

The perceived difficulty of the easy task also correlated moderately with breathing rate \( r = -0.28 \) and T:A ratio \( r = .25 \). No clear associations were found between physiological responses to the hard math condition and either subjective or behavioral measures of difficulty level.

II. Stressor Responses: Heart Rate Reactivity Subgroups

Before testing for group differences in mental arithmetic reactivity, it was necessary to determine that the cold pressor test elicited heart rate changes that were large enough to permit a subdivision of the entire subject sample into meaningfully different heart rate reactor quintiles. The three reactor groups were further defined by comparing their cold pressor response levels on the other nine physiological measures. The degree of association between heart rate change and respiratory
Table 5

Mean cold pressor change scores: High, Mid and Low heart rate reactors and all subjects combined.

<table>
<thead>
<tr>
<th>Reactor Group</th>
<th>Variable</th>
<th>Low (n = 18)</th>
<th>Mid (n = 17)</th>
<th>High (n = 19)</th>
<th>All Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HR (bpm)***</td>
<td>-5.4 (2.9)</td>
<td>2.8 (0.8)</td>
<td>13.9 (5.9)</td>
<td>3.5 (7.2)</td>
</tr>
<tr>
<td></td>
<td>TWA (%)</td>
<td>4.6 (11.0)</td>
<td>1.0 (8.5)</td>
<td>0.6 (12.5)</td>
<td>2.9 (10.6)</td>
</tr>
<tr>
<td></td>
<td>BVP (%)</td>
<td>-49.5 (22.9)</td>
<td>-63.3 (22.0)</td>
<td>-46.1 (21.7)</td>
<td>-55.0 (27.4)</td>
</tr>
<tr>
<td></td>
<td>PTT (msec)*</td>
<td>-7.0 (12.0)</td>
<td>-11.5 (12.0)</td>
<td>-17.9 (11.5)</td>
<td>-9.1 (14.8)</td>
</tr>
<tr>
<td></td>
<td>RR (cpm)</td>
<td>-0.3 (3.7)</td>
<td>0.5 (3.7)</td>
<td>0.3 (2.4)</td>
<td>0.1 (3.3)</td>
</tr>
<tr>
<td></td>
<td>T:A (%)</td>
<td>-11.0 (33.8)</td>
<td>-1.2 (68.2)</td>
<td>12.4 (56.1)</td>
<td>4.8 (95.8)</td>
</tr>
<tr>
<td></td>
<td>RA (%)**</td>
<td>-13.1 (27.4)</td>
<td>-0.3 (42.8)</td>
<td>39.1 (65.5)</td>
<td>6.8 (45.6)</td>
</tr>
<tr>
<td></td>
<td>AA (%)</td>
<td>6.0 (34.6)</td>
<td>25.0 (49.2)</td>
<td>29.7 (33.7)</td>
<td>21.3 (41.5)</td>
</tr>
<tr>
<td></td>
<td>RAV (%)</td>
<td>57.7 (327.7)</td>
<td>55.4 (121.5)</td>
<td>165.2 (354.7)</td>
<td>86.7 (260.3)</td>
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<tr>
<td></td>
<td>AAV (%)</td>
<td>23.2 (119.4)</td>
<td>101.6 (174.5)</td>
<td>127.5 (299.8)</td>
<td>115.8 (439.8)</td>
</tr>
</tbody>
</table>

*p < .05, **p < .01, ***p < .001

change was also assessed.

Cold pressor responses.

The mean change scores and standard deviations for each dependent measure are presented in Table 5. As mentioned in Section I, change scores for heart rate, pulse transit time and breathing rate were computed as the difference between task and baseline raw scores and are presented in their standard units of measurement. Task responses for the remaining seven variables had to be expressed as percentages of their respective baseline values due to the relative nature of these measures.
The first step in determining whether or not significantly different reactor groups could be formed on the basis of heart rate reactivity to the cold pressor test was to confirm that heart rate did change significantly in response to that task. A one-way ANOVA was conducted for this purpose, comparing baseline and cold pressor heart rate levels. The results indicated that task levels significantly exceeded baseline heart rates ($F(1, 99) = 24.02, p < .001$).

Information on which respiratory variables contributed significantly to the observed heart rate changes was obtained by executing a stepwise linear regression. In this analysis, a step-up procedure was used in which respiratory change variables were entered into the regression equation if their partial correlations with the independent measure (i.e. heart rate change) equalled or exceeded an F-to-enter value of 4.00. Bivariate scatterplots obtained prior to this analysis suggested that heart rate change was linearly related to change in each respiratory variable.

The regression analysis revealed that only two respiratory variables contributed significantly to the variance in heart rate change during the cold pressor test. The largest contribution was made by ribcage amplitude change. It alone accounted for 22.4% of the variance. A further 3.5% of the variance was attributable to abdominal amplitude change. In total, respira-
tory changes accounted for approximately 26% of the variance in heart rate change scores.

Subjects were then divided into reactor quintiles based on the magnitude of their heart rate change to the cold pressor test. These reactor subgroups were defined in the following way. Subjects whose heart rates decreased by two or more beats per minute (mean = -5.4 bpm, SD = 2.86, range = -2 to -12 bpm) formed the Low reactor group (n=21). The Mid reactor group (n=18) showed heart rate increases ranging from two to four beats per minute (mean = +3.4, SD = 0.87) while heart rate increases for the High reactor group (n=21) ranged from +9 to +33 beats per minute (mean = +13.9, SD = 5.87). The second (n=20) and fourth (n=20) reactor quintiles included subjects whose heart rate changes ranged from -1 to +1 bpm (mean = +0.1, SD = 0.77) and +5 to +8 bpm (mean = +6.2, SD = 1.15) respectively. In order to present effects from groups that have a greater likelihood of showing differences on other physiological responses besides heart rate, only data for the first (Low), third (Mid) and fifth (High) quintiles were included in subsequent analyses.

Before conducting a repeated measures analysis to determine whether or not the three reactor groups differed reliably from one another with respect to cardiac reactivity, a test for the equivalence of baseline heart rates was undertaken. The results of the one-way ANOVA confirmed that the three groups were not
reliably different in their baseline levels of heart rate ($F(2, 57) = 2.42, p > .05$). On the basis of this finding, change scores for heart rate and for the other nine physiological measures were used in a MANOVA that compared the responses of subjects in the high, mid and low reactor groups. By including the three other cardiovascular indices and the six respiratory change measures in the analysis, further information would be made available on possible group differences in cardiorespiratory response patterns. The mean change scores and standard deviations for each reactor group are presented in Table 5.

The results of the overall multivariate test indicated that significant between-group differences existed in cold pressor responses ($F(10, 47) = 9.03, p < .001$). Inspection of the univariate ANOVA results revealed a highly significant group difference in heart rate change to the cold pressor ($F(2, 55) = 125.30, p < .001$). Post-hoc analysis using Scheffe's method confirmed that subjects in the high reactor group experienced significantly larger increases in heart rate (mean = 13.9 bpm) than did Mid reactors (mean = 2.8 bpm) who, in turn, showed larger increases than the low reactors (mean = -5.4). Significant group differences were found for two other measures: 1) pulse transit time ($F(2, 55) = 4.51, p = .02$); and 2) ribcage amplitude ($F(2, 55) = 6.55, p < .01$). Post-hoc comparisons revealed that the high reactor group showed the largest above-baseline increase in ribcage amplitude (+39.1%) and the
largest average decrease in pulse transit time (-17.9 msec). In contrast, subjects in the low reactor group experienced the smallest average decrease in pulse transit (mean = -7.0 msec) and the largest decrease in ribcage amplitude (-13.1%). The Mid reactor group differed significantly from the other two groups in showing moderate changes on both measures (-0.3% change in ribcage amplitude and -11.5 msec decrease in pulse transit time). In summary, the above results confirmed that subdividing subjects into high, mid and low reactor quintiles yielded reliably different groups.

Mental Arithmetic Responses.

Testing for group differences in cardio-respiratory reactivity to the easy and hard math conditions constituted a 3 (reactor group) x 2 (task difficulty) repeated measures design. A MANOVA was conducted based on change scores for the 10 physiological measures. The Type I error risk was set at .05. Complete data for this analysis were only available for 18 subjects in the Low group, 17 in the Mid group and 19 in the High group. Change scores for these three groups can be seen in Table 6.

The overall multivariate test result for the grouping factor was nonsignificant, as were all 10 of the univariate ANOVAs. These nonsignificant results imply that the three reactor groups didn't show any reliable differences in their cardio-respiratory responses to the two difficulty levels of mental arithmetic.
Table 6
Changes in physiological activity from baseline during easy and hard mental arithmetic (MA): High, Mid & Low heart rate reactor groups.

<table>
<thead>
<tr>
<th>Heart Rate Reactor Group</th>
<th>Low (n = 18)</th>
<th>Mid (n = 17)</th>
<th>High (n = 19)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>HR (bpm)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Easy MA</td>
<td>7.3 (7.4)</td>
<td>4.4 (3.6)</td>
<td>7.7 (6.2)</td>
</tr>
<tr>
<td>Hard MA</td>
<td>13.1 (10.5)</td>
<td>9.6 (6.0)</td>
<td>15.5 (10.5)</td>
</tr>
<tr>
<td><strong>TWA (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Easy MA</td>
<td>-5.5 (10.3)</td>
<td>-1.3 (6.7)</td>
<td>-6.0 (10.8)</td>
</tr>
<tr>
<td>Hard MA</td>
<td>-7.7 (9.0)</td>
<td>-4.0 (11.9)</td>
<td>-6.4 (19.5)</td>
</tr>
<tr>
<td><strong>BVP (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Easy MA</td>
<td>-26.2 (44.2)</td>
<td>-34.0 (22.2)</td>
<td>-25.1 (34.4)</td>
</tr>
<tr>
<td>Hard MA</td>
<td>-27.3 (49.1)</td>
<td>-38.6 (25.8)</td>
<td>-32.6 (33.8)</td>
</tr>
<tr>
<td><strong>PTT (msec)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Easy MA</td>
<td>-12.9 (17.1)</td>
<td>-7.0 (15.9)</td>
<td>-15.3 (17.0)</td>
</tr>
<tr>
<td>Hard MA</td>
<td>-17.4 (18.7)</td>
<td>-13.0 (18.6)</td>
<td>-19.8 (15.9)</td>
</tr>
<tr>
<td><strong>RR (cpm)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Easy MA</td>
<td>2.4 (2.1)</td>
<td>3.0 (4.0)</td>
<td>2.3 (3.7)</td>
</tr>
<tr>
<td>Hard MA</td>
<td>4.4 (3.5)</td>
<td>5.3 (3.9)</td>
<td>3.9 (3.7)</td>
</tr>
<tr>
<td><strong>T:A (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Easy MA</td>
<td>10.5 (41.8)</td>
<td>2.5 (42.8)</td>
<td>31.2 (73.6)</td>
</tr>
<tr>
<td>Hard MA</td>
<td>7.5 (39.3)</td>
<td>26.3 (63.3)</td>
<td>35.6 (61.8)</td>
</tr>
<tr>
<td><strong>RA (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Easy MA</td>
<td>7.7 (27.8)</td>
<td>1.5 (27.7)</td>
<td>3.0 (32.2)</td>
</tr>
<tr>
<td>Hard MA</td>
<td>3.1 (29.4)</td>
<td>16.5 (37.3)</td>
<td>9.5 (30.1)</td>
</tr>
<tr>
<td><strong>AA (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Easy MA</td>
<td>3.3 (20.8)</td>
<td>7.2 (27.2)</td>
<td>-8.0 (32.7)</td>
</tr>
<tr>
<td>Hard MA</td>
<td>5.0 (31.9)</td>
<td>2.5 (31.0)</td>
<td>-6.6 (37.2)</td>
</tr>
<tr>
<td><strong>RAV (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Easy MA</td>
<td>60.4 (159.8)</td>
<td>47.8 (81.8)</td>
<td>87.5 (206.2)</td>
</tr>
<tr>
<td>Hard MA</td>
<td>66.4 (185.0)</td>
<td>56.5 (81.4)</td>
<td>91.1 (203.3)</td>
</tr>
<tr>
<td><strong>AAV (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Easy MA</td>
<td>30.7 (96.0)</td>
<td>51.7 (121.6)</td>
<td>42.8 (117.0)</td>
</tr>
<tr>
<td>Hard MA</td>
<td>98.4 (208.5)</td>
<td>62.0 (158.9)</td>
<td>54.8 (128.3)</td>
</tr>
</tbody>
</table>

Inspection of the multivariate and univariate ANOVAs for the difficulty manipulation, irrespective of reactivity grouping, revealed that the present results largely replicated those
reported in Section I. For this reason, they will not be presented here.

Of particular interest were the analyses for possible interactions between the grouping and task difficulty factors. Although the overall multivariate test was nonsignificant (F(20, 100) = 2.35, p = .09), the epsilon-corrected univariate ANOVAs for ribcage amplitude and T:A ratio did suggest that the three reactor groups responded differently to the two difficulty conditions (ribcage amplitude: (F(2, 51) = 3.36, p < .05); and T:A ratio: (F(2, 51) = 3.01, p = .058). Simple effects post-hoc testing revealed that the Mid reactor group experienced significantly greater ribcage excursion during hard math (16.5% above baseline) than they did during easy math (1.5% above baseline). The High and Low reactor groups showed no such effect for task difficulty. Looking at between-group differences in T:A ratio, a similar pattern was evident. Both the High and Low groups showed little variation in T:A ratio from easy math to hard math whereas the hard math condition elicited a greater T:A ratio in the Mid group. However, relative to baseline levels, the average change in T:A ratio elicited by the hard task appeared to be greatest for the High reactor quintile (+35.6%) and least for the Low reactor group (+7.5%). Contrary to expectations, no significant Group x Task interactions were found for any of the cardiovascular measures.

The unexpected absence of between-group differences on all
physiological measures suggested the possibility that heart rate reactivity to the cold pressor is a poor predictor of overall reactivity to subsequent mental challenge tasks. As a means of exploring this possibility, and also in order to determine how stable the other nine physiological measures were across tasks, change scores for each measure in each task (cold pressor, easy math, hard math) were correlated with one another. These correlations are presented in Table 7. It should be noted, however, that because the cold pressor task preceded the two counterbalanced math conditions for all subjects, the across-task correlations are confounded with order effect and are therefore difficult to interpret.

In general, the results indicated that there was relatively little correspondence between cold pressor responses and those elicited by mental arithmetic. However, reactivity to the easy and hard versions of mental arithmetic was remarkably stable across all measures, ranging from $r = .41$ (ribcage amplitude) to
r = .85 (pulse transit time). This finding suggests that cardio-respiratory responses are somewhat task specific. The fact that heart rate change to the cold pressor correlated poorly with changes elicited by either of the math conditions (r = .13 in both cases) is of particular interest since the literature suggested that heart rate reactivity to the cold pressor would be a good predictor of math task reactivity. While this did not prove to be the case, two other cardiovascular measures, blood volume pulse and pulse transit time responses, did show moderate to high levels of inter-task stability. In both cases, cold pressor changes were more strongly associated with easy task reactivity than with hard task responses. This was also true for ribcage amplitude variability, the only respiratory variable to show significant cold pressor-math task stability (r = .37). Ribcage amplitude, T:A ratio and T-wave amplitude showed the opposite trend: reactivity to the hard math task was more closely related to cold pressor responses.

Based on these correlations, it appears that respiratory changes to one type of task are generally poor indicators of the direction and/or extent of changes that may occur to a different type of task. In contrast, cardiovascular responses other than heart rate change show a moderate level of across-task stability.
Overall effects of task difficulty.

One of the central issues in this study was whether psychological challenge or stress elicits significant changes in the breathing patterns of healthy young males and whether these changes correspond to reactivity in the cardiovascular system. Overall, the data indicate that breathing rates increase significantly from resting levels when individuals respond to a continuous series of time-limited mental arithmetic problems. It was also apparent that the stress task elicited markedly more irregular and more thoracically dominant breathing than was evident when subjects simply sat quietly reading cartoons. Two measures of respiratory change, breathing rate and abdominal amplitude variability, were also found to have varied reliably with the difficulty level of stressor. In contrast, three of the four cardiovascular measures that were included in this study showed significantly different response levels to the easy and hard versions of this active coping task. In all cases where significant main effects for task difficulty were observed, the more difficult task elicited the greatest changes from baseline levels. In general, subjects breathed more rapidly and with more irregularity in their abdominal movements in response to the hard task than to the easy task. At the same time, they also experienced faster heart rates, increased peripheral resistance and higher systolic blood pressure. This
pattern of responses suggests that increasing mental challenge results in increased somatic and autonomic arousal.

However, the fact that four of the respiration measures (breathing mode, ribcage amplitude, abdominal amplitude, and ribcage amplitude variability) did not vary significantly with task difficulty suggests that breathing patterns are relatively unaffected by varying difficulty levels of cognitively mediated stress. Two observations undermine this conclusion. First, with the exception of abdominal amplitude, the average shift in response levels from rest to mental arithmetic was considerable, regardless of the difficulty level. Unfortunately, the relative nature of five of these measures precluded any testing for significant differences between rest and task response levels. Nevertheless, the magnitude of the mean change scores, even in response to the easy condition, argues against the conclusion that cognitive challenge has no effect on breathing patterns. Second, large individual differences were evident in the direction and/or extent of most respiration changes to the two math conditions. Across all six respiration measures, standard deviations exceeded their respective mean scores by a ratio of 12:1 (this value drops to 3:1 when the abdominal amplitude ratios are excluded). Given such great variability in responses, it is possible that a number of subjects responded strongly to the difficulty manipulation even though the mean response across subjects was insignificant. Furthermore, since the easy/hard comparisons were done separately for each respira-
tory variable, it is possible that notable changes did occur in the breathing patterns of most subjects but that these changes occurred in different parameters from one subject to the next. A casual inspection of the respiratory tracings suggested that for some individuals, the hard task elicited considerably higher breathing rates than did the easy task, but that little concomitant change occurred in breathing amplitudes. Other subjects appeared to show the opposite pattern of change. However, when these data are analyzed without regard to the interaction among these breathing parameters, the apparent effect for task difficulty is obscured. While testing of this possibility is of some interest, it was considered to be beyond the scope of the present study. Efforts were instead focused on testing for breathing pattern differences between cardiovascular reactivity subgroups.

Looking at the correlation coefficients for cardiovascular and respiratory changes to easy and hard mental arithmetic, the differential effect of mental challenge on respiratory and autonomic arousal becomes even more apparent. Of the 24 cardio-respiratory correlations obtained for each difficulty condition, only one (heart rate x breathing rate changes to easy arithmetic) was statistically significant. Another three correlations approached significance. Such findings imply that the two physiological systems adjusted to the brief mental challenges relatively independently of each other. The usefulness of studying stress-induced respiratory changes as
predictors of cardiovascular changes in healthy young men can therefore be considered low. The one exception to this conclusion, based on the stepwise regression analyses conducted in this study, is that breathing rate and ribcage amplitude changes to easy arithmetic are more closely related to heart rate increases than any of the three cardiovascular measures. Fully two-thirds of the 40.1% of heart rate change variance accounted for in the easy condition was attributable to changes in these two breathing variables. However, respiratory responses to the hard task were less valuable as predictors of heart rate responses, accounting for less than 19% of the 36.5% of explained heart rate variance.

One possible conclusion from these findings is that increasing stress loads result in a dissociation of heart rate and respiratory activity. In other words, the more difficult a task like mental arithmetic is, the smaller the correspondence between heart rate adjustments and ventilation efforts. This finding confirms Allen et al.'s (1986) and Carroll et al.'s (1983) conclusions. These investigators also suggested that, relative to the increases in overall ventilation that occurred during their active coping tasks, the observed heart rate increases were metabolically unwarranted. To some extent, the results of the present study are directly comparable with those obtained by these two groups of investigators. The heart rate and breathing rate changes reported in each study indicated that roughly equivalent levels of arousal had been elicited by the
different stressor tasks. This adds further credence to the argument that heart rate and respiration changes dissociated in the present study.

Temporal pattern of responses.

In comparison to changes associated with different levels of task difficulty, changes in respiration over time were generally more consistent across subjects. Three of the six measures showed significantly different response levels over the 5-minute arithmetic task. Significant task adaptation was also found for the four cardiovascular measures. On average, breathing rates increased to their highest levels with task onset. Thereafter, subjects' breathing gradually slowed, although rates still remained considerably above resting levels even at the end of the task. With task onset, subjects also tended to breath more deeply, as indicated by the increased amplitudes of both ribcage and abdominal movements. As time at the task progressed, a slight shift in breathing mode became apparent. In general, the contribution that ribcage movements made to tidal volume gradually returned to resting levels. At the same time, abdominal movements played even less of a role in filling the lungs with air than they did when subjects were resting quietly.

These data stand in contrast to the results reported by Svebak et al. (1981) in their study of breathing changes during a 2 1/2 minute video game stressor. Rather than increase in
response to task onset, ribcage and abdominal amplitude were reported to have decreased to their lowest levels. Both measures were also found to show a gradual and parallel return to near-baseline levels. The fact that the direction of ribcage and abdominal amplitude changes differed between the two studies may be the result of differing methods used to calculate respiratory amplitude. Svebak et al. expressed ribcage and abdominal amplitude responses to the lab stressor as percentages of the smallest respective amplitude values recorded at baseline. Although this procedure was designed to yield non-negative amplitude scores, basing the task scores on a single data value rather than on mean scores for the baseline period may have resulted in task scores that do not accurately represent actual increases or decreases from baseline levels. In order to avoid this problem, all amplitude responses in the present study were expressed as percentages of the average baseline values. An alternative explanation for the inconsistent amplitude results in the two studies is that video game performance may simply elicit a different pattern of physiological arousal than that produced by the less physically demanding task of answering arithmetic questions.

Reactivity subgroups and cardio-respiratory change.

The second major purpose of the present study, namely, to determine whether or not breathing pattern changes covary with cardiovascular reactivity in different reactor subgroups, on the
whole yielded no supportive findings. The three distinct reactor groups formed on the basis of heart rate responses to an initial cold pressor test did not show any overall group differences in cardiovascular and respiratory responses to subsequent mental arithmetic tasks. Stated differently, cardiac reactivity to cold stimulation, a putative marker of hypertension risk, was not clearly associated either with particular patterns of breathing changes to mental/psychological challenge or with differential cardiovascular reactivity to such challenge.

However, the data did indicate that different subgroups of cardiac reactors do respond to easy and hard versions of mental arithmetic with different levels of ribcage breathing and possibly also with distinct shifts in the predominant mode of breathing. In general, subjects in the third quintile for heart rate reactivity (designated as moderate reactors) breathed more thoracicly when the task was hard than when it was easy. Subjects in the highest and lowest quintiles, on the other hand, responded to the hard task with more or less the same degree of breathing change as they showed to the easy task. In other words, only the moderate reactors showed respiratory system sensitivity to manipulations of task difficulty. As far as changes in the predominant mode of breathing are concerned, it appears that subjects in the high reactor group experienced a much greater shift towards increased thoracic dominance than either of the other two groups and that this shift occurred irrespective of difficulty level.
Although the findings summarized in the preceding paragraph suggest that cardiac reactivity can be predictive of stress-induced respiratory pattern changes, several pieces of evidence challenge this conclusion. First of all, the task by group interactions for ribcage amplitude and breathing mode changes to the hard arithmetic task were not accompanied by similar results for cardiovascular reactivity. It can be argued that at least one of the cardiovascular measures should have shown a significant effect for the grouping variable or for a task by group interaction if, in fact, cardiovascular reactivity is a stable trait and is accompanied by characteristic patterns of breathing changes under conditions of mild to moderate stress. The question that arises is whether or not the current results were obtained because (a) cardiovascular reactivity across two laboratory stressors (cold pressor and mental arithmetic) is not a stable characteristic of most individuals or (b) respiratory changes to different difficulty levels of mental arithmetic are generally independent of cardiovascular changes.

The answer to the second part of this question has been considered already. The correlational data obtained for the sample as a whole suggest that changes in the way normal individuals breathing during mental challenge are relatively unrelated to how their cardiovascular systems adjust to the challenge. Nevertheless, these data were viewed as somewhat suspect given the large degree of individual differences observed for respira-
tory changes. It was for this reason that the sample was subdivided into distinct reactor subgroups.

The relevant question to answer, then, was whether or not cardiovascular reactivity to the cold pressor test predicts reactivity to mental arithmetic. Looking at the low correlations between cold pressor heart rate changes and heart rate changes elicited by the two mental arithmetic conditions (r = .13 in both cases), the answer is obviously negative. This finding implies that the subgroups formed on the basis of cold pressor responses were not homogeneous samples of either high, moderate, or low cardiovascular reactors. A homogeneous group of high reactors, for instance, would have included only those subjects showing exaggerated cardiovascular adjustments across a range of tasks and consistently over time. Such groups would be preferable when testing for covariation between breathing mode and cardiovascular reactivity. Instead, the reactor groups in the present study included a number of individuals whose heart rate responses to the cold pressor and mental arithmetic tasks did not reflect stable response tendencies. Rather, perceptions of task difficulty, prior success or failure at similar tasks, or a host of other state-specific factors may well have accounted for their differential reactivity to the two types of stress tasks.

One solution to this problem would have been to exclude from each of the three reactor groups those individuals whose heart rate changes to the cold pressor and two math tasks were not
consistent either in the direction of change or the magnitude of change. Alternatively, non-overlapping reactor groups might have been formed by including only subjects in the first, fifth and tenth percentiles of heart rate reactivity to the cold pressor. A similar strategy was adopted by Wood et al. (1984). These investigators demonstrated that young people whose blood pressure increases to a cold pressor test placed them in the 90th percentile for reactivity had a considerably higher risk of developing hypertension than did normoresponders. However, in the present study this strategy would have produced subgroups too small to yield reliable normative data. Another alternative would have been to execute a multiple regression analysis to predict cold pressor heart rate responses from respiratory and cardiovascular changes to easy and hard mental arithmetic. However, the emphasis in the present study was not simply to determine the extent to which breathing and cardiovascular responses covary during mental challenge, but to determine whether or not different reactor groups, one of which includes individuals at-risk for developing hypertension, respond to challenge with different patterns of cardio-respiratory change.

Problems associated with the way in which reactor subgroups were formed extend beyond the apparent heterogeneity of the groups and the general lack of significant differences between them in response to the mental arithmetic task. When one inspects the mean change scores for each of the three reactor groups, it becomes apparent that the terms 'low' and 'moderate'
to describe the first and third reactor quintiles may be misnomers. Low reactors to the cold pressor test in fact experienced noticeable decreases in heart rate. The designation 'low' reactor better suits subjects in the third quintile since they experienced heart rate increases of only two to four beats per minute. A more appropriate designation for the first quintile might then be inverse or hyporeactors.

These revisions appear justified when the mean change scores to easy and hard mental arithmetic are examined (see Table 6). For many of the cardiovascular and respiratory measures, the mean change scores for the first and fifth quintiles indicate that, on average, these two groups showed similar levels of arousal to the two difficulty conditions. In general, subjects in both groups showed greater reactivity than did subjects in the third quintile. The pattern of easy versus hard task change was also similar for the first and fifth quintiles and distinct from that of the 'mid' reactor group. For instance, neither group showed differential reactivity in ribcage amplitude and breathing mode changes to the easy and hard conditions whereas the 'mid' reactor group did. The degree of commonality between the upper and lower reactor quintiles suggests that the relationship between cardio-respiratory reactivity and varying levels of psychological challenge is quadratic rather than a linear.
Conclusions and Implications.

The results of this study support the tacit assumption of breathing control research (e.g. Cappo & Holmes, 1984; McCaul et al., 1978) that stress-induced cardiovascular arousal is accompanied by more rapid breathing. The data also indicate that active coping tasks such as mental arithmetic elicit more irregular breathing from most subjects with an evident trend for breathing to become more thoracically dominant with increased stress load. Underlying this shift in breathing mode is an apparent increase in ribcage movement and a concomitant decrease in abdominal excursions. On average, both compartments showed their greatest increases in amplitude soon after stressor onset.

Such findings suggest that breathing control strategies which aim at decreasing breathing rates while increasing abdominal excursions and the overall rhythmicity of breathing have correctly identified those aspects of one's breathing pattern that deviate most from resting levels in response to stress. This conclusion is based on mean responses obtained from a sample of healthy young men who showed marked variability in response levels to stress tasks. Furthermore, the tasks used in this study are analogues of the stressors most people encounter in everyday life. For these reasons, it would be inappropriate for clinicians to recommend breathing control training solely on the basis of these findings. The data do not clearly suggest that one group of potentially at-risk individ-
uals, cardiovascular hyperreactors, could benefit more from breathing control strategies than anyone else. It does appear, however, that regardless of task difficulty, cardiovascular hyperreactors tend to breathe more thoracically during challenging tasks than do normo-reactors. Further research comparing the breathing patterns of hyperreactors and normoreactors is necessary before unequivocal conclusions can be reached in this regard.

While the results of this study extend our current understanding of cardiovascular and respiratory changes in response to mental and physical challenges, a number of important questions still remain unanswered. It needs to verified that one's mode of breathing during stress is significantly different from resting patterns. This was demonstrated for breathing rate, but the technique used to record breathing amplitudes ruled out significance testing to confirm the apparent differences between baseline and stress response levels for amplitude measures. Nevertheless, the dual Bellows strain gauges permitted the non-invasive assessment of breathing mode, amplitude, and variability over time and across difficulty conditions.

Use of absolute measures of respiratory movements, made possible by a technique known as respiratory-inductive plethysmography (Tobin, 1986), would allow future studies to unequivocally describe the contribution made by each respiratory compartment to ventilation under various stress conditions. In
addition, this technique would enable researchers to obtain a noninvasive measure of tidal volume which, when multiplied by breathing rate, would provide a single index of ventilation changes. When recorded simultaneously with heart rate and other cardiovascular indices, this single measure of ventilation would permit a more detailed examination of cardio-respiratory interactions than is possible with the invasive procedures now in use.

A study is now needed which utilizes the respiratory-inductive plethysmography technique to assess changes in the breathing mode, volume, rate, and variability vary of different clinical populations as they respond to standardized psychological challenges or stressors. A comparison of essential hypertensives with matched normal subjects, for instance, could indicate whether or not particular breathing patterns are somehow associated with hypertension. Likewise, comparing the cardio-respiratory responses of anxiety disorder patients with those of normal individuals might suggest whether or not aberrant breathing patterns are characteristic of the former group. If so, the type of stress-induced respiratory changes observed in that study could be used in deciding on the most suitable form of relaxation/breathing control training for such individuals.

Whether relaxed breathing techniques can attenuate autonomic arousal is a question that still needs to be resolved
empirically. Once again, techniques like respiratory plethysmography or dual strain gauges fastened around each respiratory compartment could be used to noninvasively monitor the ability of individuals to produce the desired breathing changes. By simultaneously monitoring changes in cardiovascular arousal and the saturation of oxygen in the blood (Faling, 1986; Mueller, Petty, & Filley, 1970), as well as subjective reports of arousal, a more thorough assessment of breathing control effectiveness could be obtained than has been provided to date.
References


Appendix A

Health and Fitness Questionnaire

1. Age: .... years

2. Weight .... lbs/kg (Circle unit used)

3. Height .... ft. in./cm (Circle unit used)

4. Do you smoke? Yes/No (Circle answer)
   If yes, how much? Cigarettes/day
   0 - 10
   11 - 20
   21 - 30
   30 +

5. In the past 3 months, how often on average have you exercised vigorously (i.e. at least 20 minutes of continuous exertion)?
   ... 4 or more times per week
   ... 1-3 times per week
   ... seldom or irregularly

6. What activities does the above apply to? (Rank order for frequency of participation)

7. Do you suffer from any of the following physical disorders? Yes/No
   a) asthma (presently active)
   b) respiratory infection
   c) pulmonary or essential hypertension

8. Do either of your natural parents have a medical history of essential hypertension (i.e. high blood pressure), coronary heart disease or myocardial infarction (i.e. heart attack).
   Yes/No (Circle answer)
Appendix B

Experiment Introduction

Welcome to the lab ............... My name is ............ I'll be working with you today in this experiment. Why don't you make yourself comfortable in the armchair while I give you an idea of what we'll be doing over the next 45 minutes. As you'll recall from our phone conversation the other day, you were asked to participate in a study on how your body adjusts under conditions that some people consider stressful or challenging. One other thing I asked you at that time was to refrain from drinking any caffeinated or alcoholic beverages, smoking, or doing strenuous exercise in the 2 hours preceding the experiment. This is quite important as such things can give us a distorted picture of how your body might ordinarily respond to the 3 tasks of this study. Have you managed to follow these instructions? Great! It's also important for us to know whether or not you are presently bothered by any of the health problems listed on this questionnaire.

<Hand subject Health & Fitness Questionnaire>.

Also included on this inventory are some questions about your exercise and smoking habits and your family's history of cardiovascular disorders. Please respond to them as well.

**********

If the subject answers affirmatively to one or more of the questions re: respiratory problems, then thank him for volun-
teering but state that because of a possible risk to their health and contamination of the data the experiment must be discontinued.

**********

Fine! Now I'm sure you're wondering what this experiment involves. First of all, I'll be attaching the monitoring equipment that we'll use to record your body's responses throughout the experiment. I'll be in the room on the other side of this one-way mirror where the monitoring equipment is kept. After the equipment is attached and I know it's working correctly, you'll have 10 minutes to simply relax and get used to the equipment. You can read these Herman cartoons if you like. There's a clock on the table as well for your convenience.

At the end of the rest period, you'll receive some more detailed instructions on what the first task involves. I'll come into the room at that point to give these instructions to you and also to see that everything is going okay. The first task involves a brief physical challenge. You'll have a few minutes to rest after the first task before the second one - a mental challenge task - begins. Once again, more detailed instructions will be given before you start it. The same holds true for the third task. Between tasks you'll be given a few minutes to relax and also to complete a brief questionnaire. After the last rest period, I'll return to disconnect the
equipment and give you some feedback on what your responses to the different conditions were like. Any questions so far?

Okay. While I'm attaching the equipment, why don't you read over this consent form. If you feel comfortable about continuing with the experiment, go ahead and sign it. Again, if you have a question, ask me.

(State the following while attaching the equipment:)

1. In order to attach electrodes to your chest for monitoring your heart beat, I'll have to ask you to lift your shirt. I'll be cleaning the skin with alcohol and filling the electrodes with a conductive paste in order to obtain a clear signal. There is absolutely no danger of electric shocks with this apparatus.

2. This finger photoplethysmograph measures blood flow to your finger. While it's on, please keep your finger and hand movements to a minimum.

3. I'm attaching two strain gauges to you, one around your chest and the other around your abdomen. As you breath in and out, the amount of stretch in these tubes will be measured. Please breathe out deeply now so that I can fasten them. Good.
Now that all the equipment is attached, we're just about ready to begin the adaptation phase. For your information, the two rooms are connected by intercom: if you have any questions at any time, ask them and I'll respond over the intercom. Once again, you have the right to discontinue this experiment at any time if you so choose. Okay. I'm going to the other room to calibrate the equipment. Make yourself comfortable for the next few minutes. The Herman cartoons are there for you to read but please keep your movements to a minimum.
Appendix C

Consent Form

I, ........................., agree to participate as a subject in the research project entitled "Cardio-respiratory responses to mental challenge" conducted in the Cardiovascular Psychophysiology Lab, U.B.C., under the direction of Dr. W. Linden. The procedures of this 45-minute long study have been adequately explained to me. As I understand it, this study is concerned with cardiovascular and respiratory change during three brief physical and mental tasks.

I understand that I may refuse to participate or withdraw from this study at any time. I also realize that the data obtained from my participation in this study are strictly confidential: although it may be used in future studies, there will be no identification of me personally on any permanent records.

Furthermore, I have been given the opportunity to ask questions pertaining to the procedures of this study and my rights as a participant and I am satisfied with the answers received.

...............  ......................  ......................
Witness       Research Participant       Date
Appendix D

Cold Pressor Instructions

We are ready to begin the first task. This task involves putting your right hand in this bucket of ice water and keeping it there for 1 minute. So far, all of our subjects have managed to complete this task for the full minute but you may, if you wish, remove your hand at any time should you feel excessive discomfort. If you do wish to remove your hand from the water, please speak up and let me know. I'll hear you over the intercom. Please wait for my instruction before putting your hand in the water. I will enter the room again when the minute is up. At that time, I'll ask you to complete a brief questionnaire before beginning another 10 minute rest period. Do you have any questions? Okay, remember to wait for my signal before beginning.
Appendix E

Mental Arithmetic Instructions

Over the next 10 minutes, a series of arithmetic problems will be presented to you over the video screen in front of you. Each problem will be presented for 10 seconds. You must determine what the correct answer is and write it down on the answer sheet before the next problem is presented. There won't be any breaks between problems so you'll have to concentrate. So far, around 90% of our subjects have gotten scores of 75% correct or better on these problems. I'll let you know how well you did at the end of the study. If you don't respond to a problem, I'll have to consider it an error. Any questions so far? Good! At the end of the 10 minute task, I'll return and ask you to fill out the next questionnaire that you'll find face down on the table to your right. After completing this, you can continue to rest quietly for the remainder of the 5 minute break.
Appendix F

Post-experiment Questionnaire

You have completed all the tasks of this study. We would like to know what you thought of the experience. Please circle on the following scales the single number that best represents your view.

1. How do you rate your performance on the mental arithmetic tasks in comparison to other people of your age and background?

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
</tr>
</thead>
<tbody>
<tr>
<td>a lot</td>
<td>worse</td>
<td>equal</td>
<td>a lot</td>
<td>better</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

2. Please indicate with a "/" how stressful each task was.

<table>
<thead>
<tr>
<th>Task</th>
<th>Not at all stressful</th>
<th>Very stressful</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cold</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Math 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Math 2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

3. Did you experience any noticeable changes in your body during the 3 tasks? If so, indicate with an "X" which one's occurred in each of the tasks.

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Cold</th>
<th>Math 1</th>
<th>Math 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tense muscles</td>
<td>.....</td>
<td>.....</td>
<td>.....</td>
</tr>
<tr>
<td>Perspiring/Sweaty palms</td>
<td>.....</td>
<td>.....</td>
<td>.....</td>
</tr>
<tr>
<td>Yawning/Short of breath</td>
<td>.....</td>
<td>.....</td>
<td>.....</td>
</tr>
<tr>
<td>Rapid or pounding heart beat</td>
<td>.....</td>
<td>.....</td>
<td>.....</td>
</tr>
<tr>
<td>Dizziness/lightheadedness</td>
<td>.....</td>
<td>.....</td>
<td>.....</td>
</tr>
<tr>
<td>Trouble concentrating</td>
<td>.....</td>
<td>.....</td>
<td>.....</td>
</tr>
<tr>
<td>Tingling in hands, feet or face</td>
<td>.....</td>
<td>.....</td>
<td>.....</td>
</tr>
</tbody>
</table>
Appendix G

Debriefing Script

The experiment is now over. Perhaps you have some questions for me as I disconnect the recording apparatus? After I'm finished that, I'll give you a summary of what exactly we are looking at in this experiment?

One purpose of this study is primarily to correlate changes in the way people breathe in stressful situations with their cardiovascular responses, such as heart rate and blood flow to the extremities. We are especially interested in the contribution that both the ribcage and abdomen make to your breathing. It's known that these two compartments act independently yet to some degree in synchrony in the process of drawing fresh air into the lungs and then exhaling the used air. Most people appear to breathe more with their ribcage than with their abdomen when they're sitting or standing. The opposite is true when they're lying down. If you're breathing mostly by moving your ribcage in and out then your breathing mode is said to be thoracic. Some people appear to breathe more thoracically than others.

In this experiment, we expected people's breathing mode to shift towards even greater thoracic dominance as they continued at a challenging task. This effect was expected to be more
marked on the difficult mental arithmetic task than on the easier one. The reason for this change is that stress appears to elicit increased muscle tension throughout one's body—including the abdomen. When tense, your abdomen can't move as freely. In order to keep up the volume of inspired air that your body needs to function normally, we expect that your body will compensate by making greater use of ribcage movement and/or increasing the frequency of breathing. Both of these strategies are reported to require greater energy expenditure than breathing predominantly with one's abdomen.

A second purpose of this study is to determine whether or not people who show exaggerated heart rate increases to one type of stressor also experience greater thoracic dominance than other individuals during other stressors. The cold pressor test responses are used to separate people into high and low heart rate reactivity groups. The breathing patterns of these people are then compared to see if they differ to any extent. If we find that high heart rate reactors also breath more thoracicly than moderate or low reactors then we have a better basis for arguing that diaphragmatic breathing is a useful strategy to teach for reducing arousal. Any questions about what I've said so far?

I can show you some of your raw data if you like. Unfortunately, it will be difficult to understand in its present form but you can get an idea of what it looks like and what kind
of equipment we use for obtaining it.

I'd like to thank you for participating in this study. You've been a big help. Remember, all of your data are strictly confidential: your name will be omitted from all permanently records. I have one last request to make. Will you agree to keep the details of this study a secret for the next two months. During that time, we'll be asking other students like yourself to participate in this study. If they were to know what to expect ahead of time, their data would be useless to us. Thank you for agreeing to do so. Finally, if you would like to know what the results of this study are, you are welcome to contact Dr. Wolfgang Linden or Aaron Hait, a graduate student in this department, sometime in the fall of this year. Thanks again for participating today.
Appendix H

Post-task Questionnaire

Please answer the following questions by putting a slash ("/") at the point on the line that best reflects your feelings during the previous task.

1. How difficult did you find the task?

<table>
<thead>
<tr>
<th>not at all</th>
<th>very difficult</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

2. How well do you think you did at the task?

<table>
<thead>
<tr>
<th>not well</th>
<th>very well</th>
</tr>
</thead>
<tbody>
<tr>
<td>at all</td>
<td></td>
</tr>
</tbody>
</table>

3. How aroused or physically "keyed up" did you find yourself during the task?

<table>
<thead>
<tr>
<th>not at all</th>
<th>very much aroused</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix I

Average levels of physiological activity during baseline and easy and hard mental arithmetic (MA).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>Easy MA</th>
<th>Hard MA</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (bpm)</td>
<td>68.7 (9.6)</td>
<td>75.0 (10.8)</td>
<td>80.8 (13.1)</td>
</tr>
<tr>
<td>TWA (mm/mv)</td>
<td>413.0 (194.4)</td>
<td>392.8 (183.0)</td>
<td>383.5 (180.6)</td>
</tr>
<tr>
<td>BVP (mm/mv)</td>
<td>997.6 (395.7)</td>
<td>672.9 (367.6)</td>
<td>606.5 (356.6)</td>
</tr>
<tr>
<td>PTT (msec)</td>
<td>347.9 (24.6)</td>
<td>335.3 (28.3)</td>
<td>331.6 (29.1)</td>
</tr>
<tr>
<td>RR (cpm)</td>
<td>15.3 (3.5)</td>
<td>17.7 (2.9)</td>
<td>19.5 (3.3)</td>
</tr>
<tr>
<td>T:A (%)</td>
<td>100.0 (43.8)</td>
<td>115.5 (54.6)</td>
<td>121.1 (56.1)</td>
</tr>
<tr>
<td>RA (mm/mv)</td>
<td>20.7 (6.4)</td>
<td>21.8 (6.8)</td>
<td>22.3 (8.4)</td>
</tr>
<tr>
<td>AA (mm/mv)</td>
<td>23.1 (8.3)</td>
<td>22.9 (8.0)</td>
<td>22.0 (8.7)</td>
</tr>
<tr>
<td>RAV (mm/mv)</td>
<td>5.7 (4.2)</td>
<td>10.6 (4.7)</td>
<td>10.8 (4.0)</td>
</tr>
<tr>
<td>AAV (mm/mv)</td>
<td>4.7 (3.5)</td>
<td>6.7 (2.9)</td>
<td>8.1 (3.6)</td>
</tr>
</tbody>
</table>