ANGER AND DENIAL AS PREDICTORS
OF CARDIOVASCULAR REACTIVITY IN WOMEN

by

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(ABSTRACT)

Behavioral and physiological reactivity, and its relationship to cardiovascular disease has been studied in men for a number of years, and the expression of anger has been identified as a possible contributing factor. Few studies, however, have focused specifically on the reactivity of women, and those which have suggest that women are less reactive to laboratory tasks than men. For the present study, 45 undergraduate women, ages 19-21 were selected from a larger sample of 135 women to represent three discrete groups: (1) low anger/low denial, (2) high anger/low denial, and (3) low anger/high denial, based on their scores on the State-Trait Anger Expression Inventory, and the Marlowe-Crowne Social Desirability Scale. It was hypothesized that the three groups would show reliable differences in heart rate and blood pressure during presentation of a stressful laboratory stimulus, the Stroop Color and Word Test. Each subject received three counterbalanced conditions: (1) no feedback, (2) error feedback without observer present,
(3) error feedback with observer present. As hypothesized, women who reported a high level of denial and a low level of anger exhibited reliably greater systolic blood pressure to the no-feedback condition than subjects who reported low levels of denial and anger. The hypothesis that all groups would display greater reactivity in a condition which provided error feedback with observation was not supported.
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CHAPTER 1

Reactivity: The Relationship Between Emotional Stress
And Negative Health Consequences

The concept of reactivity, or varying degrees of physiological responsiveness, and its relationship to negative health consequences has been a focus of research over many decades. Engel and Bickford (1961) argued that there are widespread individual differences in autonomic nervous system response to perceived threat, moderated by both biological factors and psychological attributes. Accordingly, some individuals exhibit a characteristic "hyperreactivity," manifested by heart rate, pressor and/or endocrine responses which are elevated above normal range (Eliot, Buell & Dembroski, 1982; Obrist, 1981). The reasons for this are still somewhat unclear.

The Investigation of Hyperreactivity: Etiology

Investigations of the etiology and effects of hyperreactivity have followed three independent, yet seemingly related, lines of research. As early as 1911, Walter Cannon addressed the association between anger and the neuroendocrine mediation of the "fight-flight" response, the physiological reactivity associated with the mobilization of energy. Much later, in the early 1950's, investigators studying the relationship between cardiovascular reactivity and hypertension found that Cardiovascular Reactivity
compared to normotensives, hypertensive individuals manifested larger systolic blood pressure increases to stimuli which elicited both pain and anger (Schacter, 1957). Concurrently, other researchers utilized a harassment paradigm with men to study the effects of psychological stress on hypertension. They found that individuals could be grouped according to whether or not they expressed their feelings (especially those of anger and anxiety), and that these groups evidenced differential degrees of reactivity in blood pressure and heart rate (Funkenstein, King, & Drolette, 1954).

A third line of investigation focused on coronary heart disease. Friedman and Rosenman (1961) reported that male cardiac patients often demonstrated a particular constellation of behavioral characteristics which they termed the Type A Behavior Pattern. Since then, an enormous amount of data have been collected regarding both the behavioral and physiological hyperreactivity of Type A individuals, and the documented relationship between Type A behavior and coronary heart disease. Recent comprehensive reviews of this literature have been prepared by Matthews (1982), Glass and Contrada (1984), and Matthews (1986). A portion of this data will be discussed here.

Some investigators have argued for the importance of merging these lines of research into an integrated systems approach to cardiovascular reactivity.
approach to physiological function (e.g., Mason 1974; Schwartz, 1980). Recently, the possible advantages of considering such an approach, especially with regard to the reactivity premise, have been addressed specifically. In a comprehensive review of Type A research, Matthews (1982) reported that "the emerging strategy is to measure individual differences in behaviorally induced cardiovascular and neuroendocrine responses and to connect these measures to psychological processes that are more differentiated as well as broader than the Type A construct" (p. 315). Other researchers (e.g., Dembroski, MacDougall, Herd, & Shields, 1979; Glass, 1981; Goldband, Katkin, & Morell, 1979; Manuck, Corse, & Winkelman, 1979; Manuck & Garland, 1980) have also supported this approach. Dembroski and MacDougall (1983) acknowledged that the prediction of coronary heart disease may be facilitated by the measurement of reactivity. Likewise, Eliot and Buell (1983) concluded that hyperreactivity is presently regarded as the most plausible explanation of how Type A behavior is translated into coronary heart disease (CHD).

**Hyperreactivity and Sympathetic Function: Relation to Stress**

If it can be demonstrated that physiological reactivity is related to the psychological perception of stress, a more complete understanding of the developmental process of cardiovascular disease may become available. Stress has been
defined as an individual's perception of actual or potential harm, and the corresponding assessment that his or her resources are inadequate to cope with the threat (Glass, 1983); this conceptualization has directed the present study.

It is generally believed that activity of the sympathetic nervous system mediates the relationship between stress and cardiovascular disease (Kaplan, 1978a; Weiner, 1979). Activation of the sympathetic nervous system causes the adrenal medulla to excrete epinephrine, with the effect of increased cardiac output, heart rate and contractile force, and peripheral vasodilation. Norepinephrine is excreted by the adrenal cortex and nerve endings, and tends to augment peripheral resistance, or the contraction and relaxation of the smooth muscle walls of the small arteries and arterioles. This contractility affects vessel radius, the major determinant of change in total peripheral resistance. Existing data show that stressful tasks which require active coping increase sympathetic activity and enhance the excretion of epinephrine, norepinephrine, cortisol, serum cholesterol and hormones.

Arterial pressure is regulated by a number of neural and hormonal mechanisms, and involves both the sympathetic and parasympathetic branches of the autonomic nervous system. Briefly, systolic and diastolic blood pressure are...
indicative of different operations within the cardiovascular system (Sparks, Jr. & Rooke, 1987). Systolic blood pressure records the contraction of the heart in the pumping process. It is considered an indicator, primarily, of changes in activation associated with epinephrine. Conversely, diastolic blood pressure measures the dilatation of the heart cavities during which they fill with blood. It is considered indicative of peripheral resistance, changes primarily associated with norepinephrine. This series of events, in turn, affect blood pressure and cardiac output.

It has been hypothesized that high levels of arousal over a period of time may engender chronic changes in the cardiovascular system, including increases in serum lipids (e.g., cholesterol), myocardial lesions, arterial occlusion, and blood platelet aggregation, important factors in atherogenesis (Glass, 1983; Eliot, 1979; Haft, 1974; Raab, Chaplin, & Bajusc, 1969; Ardlie, Glew, & Schwartz, 1966). These effects have been observed in animals (Manuck, Kaplan, & Clarkson, 1983a; 1985a) and also in acute and chronic studies of humans (Frankenhaeuser, 1983; Henry, 1983; Herd, 1981; Schneiderman, 1983), primarily using male subjects. Coronary heart disease (i.e., angina, myocardial infarction) results when arterial obstruction or atherosclerosis becomes severe enough to block blood supply to the heart (Krantz & Manuck, 1984).
An important comparative study supported the above-noted progression of cardiovascular illness by demonstrating the adverse effects of increased competition and social uncertainty on heart rate reactivity in male cynomolgus monkeys (Manuck et al., 1983a), and later, on female cynomogus monkeys (Manuck et al., 1985a). Upon sacrifice, those monkeys whose psychosocial stress had resulted in high physiological reactivity showed the most advanced state of atherosclerosis. Likewise, in retrospective studies of men and women (e.g., The Framingham Heart Study, Haynes, Levine, Scotch, Feinleib, & Kannel, 1978), elevated physiological reactivity to laboratory stressors has been noted in patients with coronary heart disease or hypertension (Krantz & Manuck, 1984).

Hyperreactivity: The Heritability Premise

A number of investigators (Folkow, Hallback, Lundgren, Silverttson, & Weill, 1973; Von Eiff, 1970) have hypothesized that such reactivity may be heritable. Studies have found that young normotensive individuals tend to manifest greater elevations in blood pressure during stressful situations when there is a family history of hypertension (Briggs & Oerting, 1937; Brod, 1960; Shapiro, 1961). In addition, hypertensives often have a hypertensive parent (Thomas & Cohen, 1955). Other researchers (Falkner, Onesti, Angelakos, Fernandos, & Langman, 1979; Falkner, Cardiovascular Reactivity
Onesti, & Hamstra 1981; Manuck, Giordani, McQuaid, & Garrity, 1981) have observed that adolescents with hypertensive parents who eventually progressed from borderline (140/90 beats per minute; BPM) to sustained hypertension (160/110 BPM) also showed the largest cardiovascular responses to cognitive stress in earlier testing. These studies suggest that inherited differences in sympathetic functioning may play a causal role (Goldstein, 1981; Julius, Quadir, & Gajendragadkar, 1979; DeQuattro et al., 1981).

Additionally, Rosenman (1985) has noted that individuals at higher risk because of a hypertensive parent progress most frequently to sustained hypertension when they have shown previously the greatest physiological reactivity to anger-eliciting stressors; this is especially true when they have demonstrated that they are least able to express the anger. This finding is supported by other researchers as well (Kahn, Medalie, Neufeld, Riss, & Goldbourt, 1972; McClelland, 1979; Falkner et al., 1979). While admittedly speculative, it seems possible that in addition to inherited response tendencies, social learning might also play a part in the picture of increased intra-familial risk. Parents who are uncomfortable with openly sharing their own angry feelings may communicate to offspring, directly or indirectly, that it is not acceptable for them to show anger.
Hyperreactivity: Relationship to Anger and Hostility

A number of studies have found that reactivity, cardiovascular disease, and the frequency, intensity and expression of anger are positively interrelated (Schneider, Egan, Johnson, Drobny, & Julius, 1986; Dembroski, MacDougall, Williams, & Haney, 1984). Spielberger, Jacobs, Russel and Crane (1983), defined anger as "an emotional state that consists of feelings that vary in intensity, from mild irritation or annoyance to fury and rage" (p. 180). Equally as important as the frequency and amount of anger experienced is the expression of the feelings, often described as "anger-in," and "anger-out." Anger-in is defined as "an unwillingness in a variety of circumstances to express frustration-induced hostility and/or anger overtly, especially if the potential for interpersonal conflict would be heightened by such behavior" (Dembroski, MacDougall, Williams, Haney, & Blumenthal, 1985, p. 230). Conversely, anger-out is openly directed towards the source.

Across the studies of cardiovascular disease and negative health outcomes, the common denominators which have emerged are related to anger and the expression thereof. The relationship between hypertension and anger-in has been recognized for a number of years. Alexander, in 1939, proposed that hypertensives' efforts to suppress the
expression of angry feelings resulted in chronic activation of the autonomic and cardiovascular systems, eventually leading to chronically elevated blood pressure. Correlational studies have found a positive association between self-reported reluctance to express anger openly, and higher resting blood pressure (Diamond, 1982; Harrell, 1980; Harburg, Blakelock, & Roeper, 1979; Weiner, 1977; Baer, Collins, Bourianoff, & Ketchel, 1979), as well as elevations in baseline blood pressure under stress (Harburg et al., 1979; Gentry, Chesney, Gary, Hall, & Harburg, 1982).

The reluctance to express anger has also been observed in studies of coronary heart disease. The Israeli Ischemic Heart Disease Study (Kahn & Schill, 1972) found that self-reports of brooding and restraint of retaliation over being hurt by one’s supervisor were related to the incidence of hypertension among 10,000 male civil service workers. Likewise, anger-in was found to be related to an increased incidence of CHD in both the Framingham Heart Study (Haynes et al., 1978) and a 20-year prospective study of Harvard undergraduates (McClelland, 1979). Johnson (1984) found that anger-in was positively correlated with elevations in systolic (SBP) blood pressure for men (r=.47) and women (r=.27). Both were highly reliable at p < .001. Conversely, anger-out showed small negative correlations with SBP (r=-.13) for both men and women. Overall anger expression Cardiovascular Reactivity 9
scores were correlated with SBP for men (r = -0.45) and for women (r = -0.30). Again, these were highly reliable at p < 0.001. Correlations with DBP were of substantially less magnitude for both men and women. In an earlier study with male students, Funkenstein et al. (1954), found that pulse rate following stressful laboratory situations differed between an anger-in group and an anger-out group, with the anger-in group showing three times the elevation in rate of the anger-out group. Systolic blood pressure, however, did not differentiate the two groups in this study.

Hostility also has been linked to the severity of coronary atherosclerosis (Williams, Haney, Lee, Kong, Blumenthal, & Whalen, 1980; Shekelle, Gale, Ostfeld, & Paul, 1983; Barefoot, Dahlstrin & Williams, 1983), although the differentiation between anger and hostility in the literature seldom has been clear. Cook & Medley (1954) have described the hostile person as "has little confidence in his fellow-man. He sees people as dishonest, unsocial, immoral, ugly and mean . . . . Hostility amounts to chronic hate and anger" (p. 417-418). Spielberger, Jacobs, Russell, & Crane (1983), have proposed working definitions of anger (p. 12), hostility and aggression. Hostility has been differentiated from anger as a complex set of attitudes motivating aggressive behaviors directed toward destroying objects or injuring other people, while aggression has been
viewed as actual destructive or punitive behavior directed towards other persons, or objects (Spielberger, Jacobs, Russell, & Crane, 1983, p. 160). These researchers have pointed out the need to separate the fundamental properties of anger: (1) the intensity of angry feelings experienced at a particular time, (2) the frequency with which anger is experienced, (3) whether anger is held in or expressed outwardly, and (4) the degree to which a person endeavors to control anger.

**Hyperreactivity And Personality Characteristics**

Other studies have demonstrated elevated physiological reactivity in groups of individuals who measure high in denial and low in self-reported anxiety. Weinberger et al., (1979) found that men who scored high in denial, compared to those low in denial, showed marginally greater heart rate, increased spontaneous skin resistance and significantly greater frontalis region EMG. Scarpetti (1973), studied anxiety and denial in a sample of male and female undergraduates. It was found that subjects who scored high in denial, while self-reporting low anxiety, evidenced reliably greater galvanic skin resistance in response to threat of electric shock than a group which self-reported high anxiety and low denial. Likewise, Asendorpf and Scherer (1983) utilized a stressful phrase association task with male subjects and found that individuals who scored highest cardiovascular reactivity...
in denial also reported the lowest anxiety levels and displayed reliably greater physiological reactivity (in terms of heart rate and pulse volume amplitude) than subjects who reported both low anxiety and low denial.

Baer, Collins, Bourianoff, and Ketchel (1979) designed a brief self-report measure to differentiate essential hypertensives from normotensives, and to identify associated personality characteristics. They found that hypertensives tended to experience greater intensity of hostility and anxiety than normotensives, with longer latency of return to baseline. Manuck, Morrison, Bellack, & Polefrone (1985) have proposed that the anger component of hypertension may actually amount to an underlying deficit in assertiveness, evidenced by an inability to express feelings, stand up for one's rights and avoid mistreatment. The net result of this behavioral constellation, these investigators have suggested, is that the individual experiences anger and frustration as a result of not getting his or her needs met. Cumes-Raynor & Price (1989) have concluded that hypertensives prefer not to disclose information about personal life concerns. When social pressure to do so was increased in their study, however, accuracy of response improved. Apparently, at least for this sample, a conscious decision-making process was involved, rather than a total unawareness of the feelings themselves. Rosenman (1985)
showed that hypertensives may internalize emotions which result in exaggerated and prolonged cardiovascular responses. However, these responses may be diminished in the laboratory if subjects are given the opportunity to vent their anger (Hokanson, Burgess, & Cohen, 1963).

Additionally, a great deal of research has linked personality characteristics such as coronary-prone behavior, or the Type A Behavior Pattern (TABP) to incidence of cardiovascular disease, especially in the presence of anger. Rosenman & Friedman (1961), defined TABP as a combination of personality traits such as "excessive competitive drive, persistent desire for recognition, advancement and achievement, persistent inclination for multiple vocational and avocational involvements on one hand, and of chronic immersion in deadlines on the other hand" (p. 1173). In an early analysis of TABP components found in the Western Collaborative Group Study (Matthews, Glass, Rosenman, & Bortner 1977), competitive drive, impatience and hostility were shown to be the clearest predictors of CHD. Later studies however have found that the hostility/anger items of the Type A/B scales both differentiate between these personality types (Rosenman, Rahe, Borhani, & Feinleib, 1976; Chesney, Black, Chadwick, & Rosenman, 1981) and are correlated with the enhanced cardiovascular reactivity which Type A individuals tend to display (Williams, Lane, Kuhn, Cardiovascular Reactivity 13
Melosh, White, & Schanberg, 1982). In a component analyses of factors predictive of CHD, MacDougall, Dembroski, Dimsdale and Hackett (1985) found that Global TABP (substantially correlated with explosiveness of speech, rapid-accelerated speech and response latency) was completely unrelated to the severity of coronary artery disease. In this study, anger-in was the strongest predictor of CHD, and these results were consistent with an earlier study (Dembroski et al., 1985) in which potential for CHD was predictive only for those individuals who did not express their hostility openly. Anger-in was also found to be predictive of the incidence of CHD morbidity and mortality in both men and women in the Framingham population (Haynes et al., 1980).

Stress Induction: Laboratory Tasks

There have been a number of investigations of reactivity utilizing different laboratory stressors, which strongly suggest that personality factors interact with task variables to produce greater or lesser amounts of reactivity. In hypertensive individuals, large pressor responses have been associated with laboratory-induced emotions of anger and fear (Schacter, 1957), anxiousness over interviews centered on areas of personal conflict (McKeegney & Williams, 1967), participation in demanding cognitive tasks (Shapiro, 1989; Nestel, 1969), stressful Cardiovascular Reactivity
interpersonal situations involving negative evaluative feedback (MacDougall, Dembroski, & Krantz, 1981), and role-playing requiring assertive responding (Arkowitz, 1981; Bellack, 1979). In general, hypertensive men have been shown to be more reactive to tasks involving competition, reaction time, and cold pressor stimuli (Shapiro, 1961; Manuck, Giordani, McQuaid, & Garrity, 1981), while hypertensive women have demonstrated greater blood pressure elevations to Stroop stimuli (Golden, 1978), mental arithmetic (Falkner et al., 1979), and interpersonal interaction situations which call for aggressive or assertive responding (Arkowitz, 1981; Bellack, 1979). Investigators have found differing responses to active tasks such as reaction time, and passive tasks such as cold pressor or viewing of a pornographic film (Obrist, 1981). It has been argued that the active or passive nature of the task is a crucial variable in responsiveness, and that "sympathetic influences on the cardiovascular system are most pronounced when the subject is engaged in active coping behavior, particularly during cognitive tasks (Goldstein, 1981; Shapiro, 1961; Light & Obrist, 1980).

A parallel line of investigation of Type A/B individuals has produced similar results. For example, Type A women did not differ reliably from Type B women on cold pressor and reaction time tasks during which the difficulty
of the task was emphasized (MacDougal et al., 1981), but these same investigators found that Type A women showed reliably greater increases in systolic blood pressure to an oral history quiz. In another study involving abstract reasoning, Manuck, Harvey, Lechleiter, & Neal, (1978) observed differences in cardiovascular reactivity between Type A and B men, but found that Type A and B women did not differentiate reliably. Conversely, no differences in reactivity were found between Type A and B men and women subjected to a social interaction paradigm (Van Egeren, 1979a, 1979b). Additionally, there were no reliable gender differences in the behavioral responses of the groups. Thus Type A men and women were noticeably more aggressive and competitive than their Type B counterparts. Additionally, Dembroski, McDougall, Herd, & Shields (1979) found that Type A men responded with greater cardiovascular reactivity than Type B men to situations which involved interpersonal challenge or threat. A similar study using only women (MacDougall, Dembroski & Krantz (1981) reported that Type A women showed greater elevations than Type B women in situations involving demanding social interaction. In a final study (Jorgensen & Houston, 1981) the reactivity of type A's and B's, both men and women, was compared utilizing three different stressors: (1) the Stroop Color Word Test, (2) mental arithmetic, and (3) shock avoidance. No reliable Cardiovascular Reactivity
gender differences in reactivity were found for any of the tasks. Type A subjects showed reliably greater diastolic blood pressure than Type B subjects during the Stroop test, and greater elevations in systolic blood pressure than Type B's during the mental arithmetic task. Shock avoidance produced marginally reliable differences (p < .58) between Type A and Type B subjects. The authors hypothesized that the tasks utilized may have been free of gender role expectation, and additionally, that they may have engaged all participants equally.

Research on the relationship between laboratory stressors and cardiovascular reactivity, suggests that previously reported gender differences in systolic blood pressure may be more attributable to specific task effects; women do appear to show elevated physiological reactivity to certain tasks differentially from men, particularly traditionally-used laboratory stressors such as reaction time and cold pressor tests. Both men and women, however, appear to be reactive to situations involving interpersonal challenge. There is also a strong suggestion that personality variables such as Type A/B personality characteristics, passivity, and inability to cope successfully with situations requiring assertive or aggressive interpersonal responding interact with task characteristics to exacerbate these effects.
Despite the existence of a large body of research focused on the etiology and course of psychosomatic disease, there is comparatively little data regarding the reactivity of women, specifically. Data that does exist is equivocal. A review of studies involving women suggests, generally, that they are biologically less reactive than men to acute behavioral stress. For example, Stoney, Davis and Matthews (1987), did a meta analysis of psychophysiological studies published from 1965 up to and including 1986, which included women. They found a number of gender differences in reactivity. Relative to men, women had higher heart rates at rest and higher heart rate increases during challenge. Men had higher systolic blood pressure at rest than did women, and also exhibited higher elevations during challenge. Finally, men had larger urinary epinepherine output than women during stress, but not at rest. Diastolic blood pressure, urinary norepinephrine and cortisol in these studies showed non-significant gender effects. These investigators concluded that the results "provide some support for the hypothesis that men show greater physiological responses during acute behavioral stress than do women" (p. 129). They did note however that few investigations were available for analysis and little research had been programmatic, "making it impossible to Cardiovascular Reactivity

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conduct analyses of important issues that we had initially intended to address" (p. 129).

Other studies suggest that in certain situations women may be as reactive as men, or nearly so. Frankenhaeuser and associates, in a series of ongoing studies comparing neuroendocrine responses of men and women (e.g., Frankenhaeuser, Lundberg, & Forsman, 1980; Frankenhaeuser et al., 1978), have typically found that men excrete greater levels of catecholamines in stressful situations, particularly epinephrine. However, in situations where sex roles were reversed or role differentiation was minimized (subjects were more homogeneous), differences between men and women were greatly diminished (Lundberg, deChateau, Weinberg, & Frankenhaeuser, 1981). Likewise, when neuroendocrine responsiveness for groups of men and women engineering students was tested in a cognitive-conflict task, women increased their adrenaline excretion almost as much as men who were fellow students (Collins & Frankenhaeuser, 1978). These investigators suggest that male/female reactivity may be more dependent on situational factors and the amount of investment a given subject has in the situation at the time, than basic differences in biological reactivity.

In another study (Lerner & Kannel, 1985), working women and housewives were typed according to characteristics of Cardiovascular Reactivity.
the Type A Behavior Pattern. Those women who were classified as Type A scored significantly higher than Type B's on emotional lability, tension and anger symptoms, and also were found to be at much greater risk for CHD. Among women under 65 years of age, elevated scores on the Framingham Type A Scale and emotional lability remained significant predictors of cardiovascular disease when other factors, i.e., age, blood pressure, cholesterol, smoking and other psychosocial scales were controlled. Likewise, both white-collar Type A men and women were found to be at higher risk for CHD (e.g., Haynes, Feinleib, & Kannel, 1980). Waldren (1977) observed that Type A behavior was positively correlated with higher occupational status for women, and was more prevalent among women employed full time, leading to the conclusion that coronary prone behavior might also be related to upward educational mobility, contributing to success in vocational pursuits.

In another study of coronary-prone behavior using only women Lawler, Rixse and Allen (1983), investigated the relationship between Type A behavior, as measured by the Jenkins Activity Survey (JAS), and physiological reactivity, using a more homogeneous sample of 41 women aged 25-55. Half of these subjects were employed at a professional or executive level, and were categorized as strong Type A's; the remainder were housewives, some categorized as A's and

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some B's. In two tasks involving mental arithmetic and visual puzzles, Type A women evidenced significantly greater increases in heart rate, systolic blood pressure and diastolic blood pressure than did Type B women. However, a replication of this study with a group of younger women students, aged 18-25, who were studying in male-dominated fields failed to differentiate the two types (Lawler, Schmied, Mitchell, & Rixse, 1984). These investigators acknowledged that the result may have been due to methodological problems. This finding, however, is in keeping with several other studies which found Type A/B women more discernable from each other after age 25, when Type A's manifested more physiological reactivity, and a greater incidence of coronary heart disease. For example, Rosenman and Friedman (1961), in a sample of women aged 30-59, found four times the incidence of clinical heart disease for Type A women, as compared with Type B women, and also found a strong connection with type of employment. Whereas 35% of the total population studied were in professional and executive occupations, 62% of women with CHD were in these occupations. Likewise, Waldron (1977), found greater evidence of TABP in a sample of women aged 40-59, who worked full time, as opposed to women who didn't work or worked part-time. These women also had higher levels of education (at least some college) and worked at the management or Cardiovascular Reactivity 21
professional level. Women who worked full time had significantly higher SBP, on average, than those who were employed part-time or were housewives.

Similarly, in a study of employed men and women, Jenkins, Rosenman and Friedman (1967), found that more men in the 18-25 age range scored as Type A than did women; however at ages 26-44, women were as Type A as men. These investigators posited that women in the older group may have had more time pressure because of added responsibilities, or that the group of younger working women was likely to include more Type B women. It should also be noted that in both of these studies (Waldron, 1977; Jenkins et al., 1967), Type A scores were at the 50th percentile or below, actually in the Type B range. Thus the types may not have differentiated physiologically because they were not clearly differentiated typologically.

In a careful study of men and women aged 25-44 years, Davidson, Vandongen, Rouse, Beilin, & Tunney (1984), investigated differences between resting and "stressed" measures of heart rate, diastolic blood pressure, systolic blood pressure, plasma adrenaline and noradrenaline under four different stress conditions. While men showed higher resting baselines on measures of systolic blood pressure and adrenaline, both men and women exhibited significant increases under the various stress conditions, and tended to
vary proportionately. Additionally, women tended to show higher baseline measures of heart rate and noradrenaline, which were maintained under stress. This study strongly suggests that while men and women differ significantly in resting baselines, the amount of reactivity (demonstrated in change values from baseline) they display under stress is very similar. Whether or not it is the absolute value of physiological change, or the proportion of change from baseline that is damaging remains to be addressed.

Several studies involving women have viewed reactivity as a personality construct. Harris, Sokolow, Carpenter, Freedman, & Hunt (1953) rated female college students who were classified as borderline hypertensives by their responses to emotionally-provoking psychodramas. Behaviorally, they were described as hot-headed, daring, rash, tense and hurried. A follow-up of the same group as college seniors included personal interviews scored by a "blind" observer. Oral aggressiveness, excitability, self-defensiveness, hostility and anxiousness were included in the behavioral ratings (Kalis, Harris, Bennett, & Sokolow, 1981). These investigators concluded that the young women were unable to express hostility appropriately, resulting in emotional reactivity, anxiety and muscle tension. There was no follow-up to see whether these behaviors were actually predictive of hypertension. Another study rated female Cardiovascular Reactivity
hypertensives as interpersonally ineffective and inappropriate, with observers again suggesting conflict about assertion, and precarious control over hostility (Kalis, Harris, Sokolow, & Carpenter, 1957). These studies again suggest that "person" variables may be strongly related to how specific situations are perceived and acted upon, both behaviorally and physiologically, and that women may be more reactive to situations which have interpersonal involvement.

The foregoing investigations, taken together, underscore some of the major problems in assessing the vulnerability of women in connection with behavioral stress and cardiovascular disease. First, there have been comparatively few studies, especially of an experimental nature, involving women. Those which do exist typically have sought to contrast male/female reactivity per se, rather than investigating whether or not specific tasks are differentially arousing as a function of sex and/or age, or whether women value or perceive certain types of tasks differently than men. Additionally, few prospective studies were found which attempted to experimentally identify personality factors which may contribute to elevated reactivity, and which may allow prediction of cardiovascular disease in women. It is important to ascertain whether women, like men, differentiate from each other, not only in

Cardiovascular Reactivity
physiological reactivity, but also in personality characteristics, degree of motivation, levels of emotionality and other factors which appear to be associated with hyperreactivity in men.

There is data to show that women have less incidence of coronary heart disease than men. In a 26-year follow-up on the Framingham Heart Study population of men and women overall, women encountered half the amount of CHD as men did, only approximating the mortality rate of men in the +75yr cohort (Kannel & Abbott, 1984). It is striking, however, that women showed an increase of approximately 40 times the original morbidity between age 30 and age 75+. While this essentially reflects the very low incidence of CHD in women in the 35-44 age group (in 1978 when the study was updated), morbidity rates for women accelerated more quickly than those for men after age 45, and the men/women ratio of 6 to 1 was reduced to a factor of one by age 75. It is also important to note that women had no survival advantage once CHD became overt; in fact the risk of death for women during a coronary attack exceeded that of men in every age category (Lerner & Kannel, 1985).

The questions of whether or not women are less prone to cardiovascular disease and whether this is resultant from some gender-related variation in biological processes are compelling. Krantz and Manuck (1984) and Matthews and Haynes Cardiovascular Reactivity
(1986) have proposed that large magnitude cardiovascular and neuroendocrine responses to acute behavioral stress may elevate risk for coronary heart disease (CHD). To the extent that this is true, verification of lower reactivity in women could provide a valid explanation for the observation that women suffer lower incidence of CHD (Stoney et al., 1987; VanDoornen, 1986). Alternatively, however, other explanations for women's possible "lower reactivity" should be explored. These include variations in behavior and perception, differing social roles and values, small, poorly controlled samples and methodological artifact. Any of these factors may provide plausible explanations which, to date, have not been ruled out by adequate empirical study.

Historically, women have led a substantially different life style from men, that only recently has changed enough to affect large numbers of women. The number of working women in the United States is increasing: The projected number of women employed full-time in the civilian labor force by the year 2000 is expected to increase to 65.8 million, from 31.5 million in 1970 (U. S. Bureau of Labor Statistics, 1987). Likewise, a growing number of women are entering full-time white-collar management and professional positions. In 1988, 49.4% of professional positions were filled by women, as well as 43% of managerial positions, and 84% of technical, sales and administrative support
(U. S. Bureau of Labor Statistics, 1987). Additionally, substantial increases in the number of women enrolled in college, from 3,877,000 in 1972, to 6,554,000 in 1986 (U. S. Bureau of the Census, 1980) suggests that women are actively seeking a more substantial career role. As yet, there exists little data regarding the possible long-term effects on women of dealing with traditional "male" stressors. Additionally, many working women continue to deal with traditional "female" stressors such as primary responsibility for children's needs, care of the home, and so forth.

Another cause of the seemingly reduced reactivity of women may be methodological error. Few of the studies incorporating women have used samples of representative size. Additionally, most samples have been homogeneous only in that they were composed of college freshmen, or Type A/B women. Information as to medical histories, social backgrounds, career goals and expectations, personality traits, motivation, behavioral state at time of testing, or perception of the test itself has not been well reported in the literature.

Physiological indices in themselves may contain multiple sources of differential measurement, which are difficult to control if specific detailed measurement parameters are not included as part of a given study.
Seemingly small methodological details such as failing to size a blood pressure cuff to the circumference of a subject’s arm may cause large variations in blood pressure readings (Harrison & Kelly, 1987; Harrison, Gorelcenko, & Kelly, 1988), particularly between men and women. Investigators have also reported significant variations in blood pressure measures depending on whether a manometer, an automated occlusion device, or intra-arterial recording is used to measure BP (e.g. Harrison & Edwards, 1988; Melville & Raftery, 1981).

Other potential sources of variability include diurnal variations in catecholamine excretion, phasic variations during women’s monthly menstrual cycle, ingestion of substances (caffeine, nicotine, alcohol) prior to testing, and so forth. While a number of studies have controlled for some of these variables, few if any have considered all. More importantly, most have not provided details regarding these issues in the study itself. This has made it difficult to correctly assess the differential results that tend to prevail, or to replicate a given study successfully.

**Investigation and Hypotheses**

Differences in anger expression and the number and quality of situations which induce anger have been identified as possible contributors to hyperreactivity and cardiovascular disease in men. However, the impact of these cardiovascular Reactivity
variables on women has not previously been assessed. Additionally, several prior studies (Weinberger, Schwartz, & Davidson, 1979; Cottington, Matthews, Talbott, & Kuller, 1986) have suggested that denial may function as a negative coping mechanism in some individuals, actually exacerbating the physiological effects of stress by preventing resolution of the stressful situation. This study was designed to address these variables.

**Purpose of Investigation and Subjects**

While the effects on physiological reactivity of denial in association with anxiety have been investigated to some degree, no studies to date have addressed the possible effects of denial coupled with anger, particularly for women. Thus, the purpose of the present study was to evaluate specifically those women who self-reported very high or low levels of anger in combination with very high or low levels of denial. Undergraduate women between the ages of 19-21 with no prior history of medical problems were chosen as subjects. Since this population has not demonstrated differential reactivity in past studies, and since several studies (e.g., Lawler et al., 1983) have shown that cardiovascular reactivity increases with age, it was reasoned that evidence of differential reactivity in groups of young women would provide strong support for potential increases in the differential reactivity of older women.

**Cardiovascular Reactivity**
Measures of Anger and Denial

There is a good deal of research to support the premise that frequency, intensity and expression of anger are important contributors to physiological reactivity, and thereby to cardiovascular disease endpoints (e.g., Alexander, 1939; Funkenstein, et al., 1954; Averill, 1982; Tavris, 1982). Because the State-Trait Anger Expression Inventory (STAXI; Spielberger, et al., 1985; Spielberger, 1988) was designed to differentiate among these variables, it was selected as the primary measure of anger for this study. The STAXI is designed to measure state anger, trait anger and anger expression (derived from scores on three subtests: (1) anger-in, (2) anger-out, and (3) anger control. Trait anger has been defined by the author as "individual differences in the disposition to experience anger (range of situations which one finds anger-provoking) and the intensity of the anger" (Spielberger, 1988, p. 1). State Anger asks subjects to rate the intensity of their anger "right now," on a four-point scale. Spielberger has described state anger as "an emotional state or condition that consists of subjective feelings of tension, annoyance, irritation, fury and rage, with concomitant activation or arousal of the autonomic nervous system" (Spielberger, 1988, p. 1). S-Anger can vary in intensity and fluctuate over time. "Anger-in (AX/In) measures the frequency with which..."
angry feelings are held in or suppressed. Persons with high AX/In scores frequently experience intense angry feelings, but they tend to suppress them rather than expressing them, either physically or in verbal behavior. Some persons who are high in AX/In may also have high anger-out (AX/Out) scores, in which case they may express their anger situationally. AX/Out measures how often an individual expresses anger toward other people in the environment. AX/Out may be expressed in physical acts such as assaulting other persons or slamming doors, or it may be expressed verbally in the form of criticism, sarcasm, insults, threats, and the extreme use of profanity. Anger control (AX/Con) measures the frequency with which an individual attempts to control the expression of anger. Persons with high scores on the AX/Con scale tend to invest a great deal of energy in monitoring and preventing the experience and expression of anger. Over-control of anger may result in passivity, withdrawal, and depression in persons with high AX/CON scores who also have high T/Anger scores and low AX/Out scores. Anger expression (AX/Ex) provides a general index of the frequency with which anger is expressed, regardless of the direction of that expression" (p. 1).

The STAXI provides a way of measuring each component of anger separately, as well as determining a combined anger expression score based on levels of anger-in, anger-out and Cardiovascular Reactivity
anger control. Subsequent validation studies of this instrument (Spielberger, Krasner, & Solomon, 1988) have suggested that individuals who experience anger more frequently are also more likely to both express and suppress their anger (anger-out/anger-in) than persons who experience anger less frequently. The Trait Anger scale of the STAXI was utilized as one of the preliminary screening measures by which subject groups were formed.

The Cook-Medley Hostility (HO) Scale (Cook & Medley, 1954), a 50 item hostility inventory, has also been found frequently to be associated with coronary heart disease outcomes (e.g., Barefoot, Dahlstrom, & Williams, 1983, Williams et al., 1980; Williams et al., 1985; Shekelle et al., 1983). It will also be utilized in an attempt to assess whether the factors it measures are similar to or different from those of the STAXI. Williams et al. (1985), have argued that "persons with a strong attitudinal set of hostility will be likely to experience the emotion of anger more frequently and intensively than persons low in hostility."

Although instruments which are typically utilized to measure anger and/or hostility have been evaluated for reliability and validity, they still rely on self-report, and as such, may be less than accurate assessment tools. Regardless of how clearly the constructs of anger, hostility and aggression are set forth, some individuals may not.
respond accurately to such direct measurement. Studies of anxiety suggest that individuals with a high denial profile coupled with self-reported low anxiety or low aggression actually show heightened physiological reactivity to stress as compared to those who score in the range of high anxious, high neurotic (Parsons, Fulgenzi, & Edelberg 1969; Cohen, 1975; Scarpetti, 1973). These individuals may be prone to physical disease, including hypertension and cancer (Carroll, 1972; Blackburn, 1985; Davies, 1970; Kissen, 1986).

In recognition of the denial problem, several investigators (e.g., Weinberger, Schwartz, & Davidson, 1979) have used a multiple-measures approach including behavioral observation and physiological recordings, as well as self-report techniques. Through the use of a scale which has been frequently utilized to measure denial (Crowne-Marlowe, 1964), the existence of a denial style of coping with stress has been supported. Although initially developed to measure social desirability, the Marlowe Crowne Social Desirability Scale (MCSD; Crowne & Marlowe, 1964) has been used frequently as a measure of defensiveness (Cunes-Rayner & Price, 1988; Jamner & Schwartz, 1986; Kahn & Schill, 1971; Boor & Schill, 1967). In one study using this scale, subjects reported low amounts of anxiety, but scored high on measures of denial and showed elevations in reactivity.
(Weinberger, Schwartz, & Davidson, 1979). Several other investigators (e.g., Boor & Schill, 1967) have noted that the MCSD is particularly useful in differentiating individuals who self-report low levels of anxiety, yet display concurrent measures of relatively high physiological reactivity (i.e., blood pressure and heart rate). There were no studies found which had used the MCSD in conjunction with measures of anger. Crowne and Marlowe (1964) have also reported data to show that the scale measures "affect inhibition, defensiveness and protection of self-esteem." The MCSD correlates with the Lie scale of the MMPI at 0.54, lending validity to the assertion that it is measuring something other than the subject's true response. This scale was utilized as the second prescreening measure by which subjects were assigned to groups.

**Experimental Stressor**

The Stroop Color and Word Test (Golden, 1978) was specifically chosen as the stressor in this study for two reasons. First, past research has shown that the task can be performed by most subjects without extensive practice. Thus it was believed to be capable of providing a more or less consistent measure of stress across individuals, free from large differences in performance. Additionally, the Stroop test has been demonstrated to be an effective stressor for women. Jorgenson and Houston (1981) found that the Stroop Cardiovascular Reactivity
test was associated with reliable increases in systolic blood pressure for women, both with and without a family history of hypertension. Likewise, in a study of male and female university students Frankenhaeuser, Dunne, & Lundberg (1976) found reliable increases in heart rate attributable to the stress of the Stroop test. In the standard Stroop presentation, words naming colors are presented in non-congruent colors on stimulus sheets arranged in three columns per page. The subject is asked to read down each column of words in the presence of an experimenter, who verbally tells the subject when an error is made. For this study, the words were presented on 35mm slides, and the subject was instructed to call out the color she saw to an experimenter in another room. The stimulus was modified in this manner to enable assessment of the differences in reactivity produced by different feedback conditions.

Three different stressful conditions were developed: (1) no feedback, (2) feedback without observer present, and (3) feedback with observer present. It was hypothesized that error feedback by means of a buzzer would create performance anxiety, thereby increasing the stressfulness of the task. Thus, greater reactivity was expected in the feedback without observation condition, than in the no feedback condition for at least some groups, and physiological Cardiovascular Reactivity 35
reactivity in each stress condition was expected to be reliably elevated from baseline. Since women have been shown to perceive interpersonal situations as more stressful (e.g., Harris et al., 1953), especially those of an evaluative nature (e.g., MacDougall et al., 1981) a third condition, error feedback with observer present was added. This enabled the study of the differential effects of interpersonal evaluation, apart from the stress engendered by the task itself.

**Physiological Measures**

As physiological indicators of arousal (Lacey & Lacey, 1970) due to the differentially stressful conditions, measures of systolic and diastolic blood pressure and heart rate were taken during the baseline and experimental conditions. These measures have been used frequently in other studies, some of which suggest that they do not always react in concert with each other. The reasons for this are not completely clear, due to the complex interactions which characterize cardiovascular physiology.

Ax (1953) found that a laboratory paradigm which induced anger produced differential patterns of reactivity from one which produced fear, in terms of diastolic blood pressure, heart rate, skin conductance and muscle tension. He proposed that fear was an epinepherine-like response, and that anger was a mixed epinepherine-norepinepherine
response. Most autonomic responses are dually innervated by the sympathetic and parasympathetic branches of the autonomic nervous system, as well as by hormones and a typical response can be mediated by a number of different variables (Schwartz, 1982). For example, an increase in heart rate could be related to "an increase in local sympathetic activity, a decrease in local parasympathetic activity, an increase in circulating epinephrine (or any other heart-stimulating hormone), or any combination or pattern of these mechanisms" (Schwartz 1982, p.82).

Prior studies (e.g. Bonvallet & Allen, 1983) have found that one role of the central nervous system is that of an inhibitory mechanism, delimiting the duration and course of cortical, autonomic and muscular responses to internal and external stimulation through the bulbar inhibitory area. These investigators demonstrated that if the vagus and glossopharangeal nerves were cut, preventing afferent input from reaching this area of the brain, an irregular pattern of poststimulatory cortical activation was prolonged.

Because all of these variables are highly interactive and interdependent on cross-feedback, differential reactivity based on individual differences in perception might be expected. If this is the case, similarities or differences in patterns of response between measures could provide information about the differing physiological Cardiovascular Reactivity
effects of specific types of stress on specific types of individuals. Unfortunately, few studies have explored this possibility. In one such study, Schwartz Weinberger and Singer (1981) noted significantly greater diastolic blood pressure during anger imagery than during fear imagery. When exercise was combined with imagery, however, large differences in systolic blood pressure and heart rate, but not diastolic blood pressure were found between the two emotions.

Not only do these studies suggest that specific physiological variables do show differences in reactivity which are situationally dependent, but also that the emotional components of situations may not be unitary in terms of the feelings generated. In a study of emotional constructs, Schwartz, Weinberger and Brown (1980) demonstrated that a given emotion did not represent a singular quality (e.g. "sad" or "angry") for a given individual. Subjects were asked to imagine different situations such as "your dog dies" or "your boyfriend leaves you for another," and to self-rate the emotions generated from an array of possible feelings (e.g. happy, sad, depressed, angry, fearful, and anxious). While the "dog" situation engendered high ratings in feelings of sadness and depression, the "boyfriend" situation engendered high ratings in anger and anxiety as well as sadness and cardiovascular reactivity.
depression. Thus given situations generated highly complex blends of emotion, rather than a single quality. Although not specifically addressed by these investigators, it might also be hypothesized that this effect would be magnified by differences in individual perception of situations, and the meanings attributed thereto. A number of studies have addressed different aspects of this variability.

Funkenstein, King and Drolette (1954), for example, studied the relationship between direction of anger expression and blood pressure and heart rate reactivity. While groups classified as high in anxiety or internalized anger manifested systolic blood pressure and heart rate increases, the group which expressed anger outwardly was divergent, exhibiting a large increase in diastolic blood pressure, associated with markedly elevated peripheral resistance and little heart rate change. Other research has found these variables to form different patterns of relationship to each other in response to varying types of stressors. Lacey and Lacey (1970) first noted that cardiac deceleration (the "orienting response") was associated with situations which demanded the subject's attention, such as a reaction time task which was cued by prior signal. An interesting related study (Israel, 1968, 1969) found that the magnitude of this deceleration was seemingly related to the cognitive style of the subject. For individuals whose Cardiovascular Reactivity
natural style was to be acutely aware of and attentive to the details of their external environments, decelerations were of similar magnitude regardless of the stimulus—whether low preference, high preference, or anxiety producing. Subjects who tended to be less attentive to environmental input exhibited less deceleration overall, and the magnitude of the deceleration varied, depending on the character of the stimulus. Likewise, Craig and Woods (1969) reported differences in the direction of cardiac response between directly-experienced stress on a cold-pressor task, and the vicarious experience of watching others perform the task.

The limited evidence available at this time seems remarkably consistent in suggesting that neither emotional perception nor physiological reactivity to such perception is as simple as was once thought. While the idea of physiological patterning is intriguing, the complex interactions among variables, and the fact that the reactivity of one variable in a given direction may be caused by a number of different contributory factors highlight the problems inherent in such an approach. It is clear that much additional study is necessary before it can be determined whether specific situations and the perceptions they engender are consistently associated with particular patterns of physiological reactivity. However, it
is also clear that if such patterns can be interpreted, a wealth of new information might be made available with regard to similarities and differences between self-described emotional state and apparent physiological state. For this reason, both heart rate and blood pressure data was collected for this study.

**Hypotheses**

1. Women who scored in the upper quartile of the normative distribution on a pretest measure of denial (Marlowe Crowne Social Desirability Scale; MCSD) and in the lower third of the normative distribution on the Trait Anger scale of the State Trait Anger Expression Inventory (STAXI; Spielberger, 1988), were expected to show reliably higher levels of physiological reactivity to stressful cognitive laboratory tasks (no feedback, feedback without observer, feedback with observer) than women who scored in the lower third of the normative distribution on both anger and denial.

2. All subjects were expected to exhibit reliably greater physiological reactivity to the three different feedback conditions of the Stroop Cognitive Conflict Test than was exhibited during the baseline periods (without Stroop) which occurred before and after the experimental stress.

3. Differential reactivity by groups to the three Cardiovascular Reactivity
separate conditions was anticipated. Specifically, error feedback delivered in a situation where an observer was present was hypothesized to create significantly higher physiological reactivity than the other two feedback conditions.

4. No hypotheses were developed for possible differential performance on the Stroop Color and Word Test across groups or conditions, although it was investigated.
CHAPTER 2

Method

Preliminary Screening Phase

Instruments

Scores on two self-report measures were used to identify subject characteristics representative of three subject groups. The first grouping measure used was the Trait Anger Scale, a 10-item subscale which forms a part of the State-Trait Anger Expression Inventory (STAXI; Spielberger 1988; see Appendix A). Trait anger represents a measure of individual differences in both frequency and intensity of anger experienced across situations. Internal consistency for the fifteen-item version of the Trait Anger Scale is 0.87 for both males and females. The ten-item version which was used in the present study correlates with the larger version at 0.97. (for a more complete definition and description of this scale see p. 40).

The second grouping measure used was the Marlowe-Crowne Social Desirability Scale (MCSD; Crowne & Marlowe, 1964; see Appendix B). This scale consists of 33 items and has been represented by the authors to measure defensiveness, protection of self-esteem and affect inhibition. Internal consistency is reported to be 0.88. (For a more complete description of this scale see p. 45-46). The MCSD was utilized as a measure of denial (Cunes-Rayner & Price, 1988; Cardiovascular Reactivity 43

**Prescreening Process**

Both pretest measures were administered to all subjects within a four-day time period, within two weeks of the administration of the experimental phase of the study. Based on scores on the pretest measures, forty-five women subjects were assigned to one of three groups of fifteen subjects each. The first group, low anger/low denial was composed of subjects who scored in the lower third of the normative distribution on the anger inventory and the lower third of the sample distribution on the denial inventory. This group obtained a mean score of 16.00 on the trait-anger measure (range 13-17) and a mean score of 11.80 on the denial measure (range 3-17). The second group, high anger/low denial was composed of subjects who scored in the upper third of the normative distribution on the anger inventory and the lower third of the sample distribution on the denial inventory. These subjects obtained a mean score of 24.00 on the trait anger measure (range 22-29) and a mean score of 10.73 on the denial measure (range 2-15). The third group, low anger/high denial, was made up of subjects who scored in the lower third of the normative distribution on the anger inventory and the upper quartile of the sample distribution (as suggested by Weinberger et al., 1979) on the denial Cardiovascular Reactivity
inventory. These subjects obtained a mean score of 13.73 on the trait anger measure (range 11-17) and a mean score of 23.00 on the denial measure (range 21-29).

Selection of Cut-off Points

When initially published in 1964, the normative mean on the MCSD for women students was 16.82 with a standard deviation of 5.50. The mean for the present sample was 18.59 with a standard deviation of 2.74. In addition to testing a sample of women for the present study, a sample of 154 men was measured by the same instrument at the same time. The mean for males in this sample was 18.82 with a standard deviation of 2.74, while the normative mean for males published in 1964, of 15.08 with a standard deviation of 5.58. To allow for these differences in the present study, the cutoffs for both "high denial" and "low denial" were adjusted by 3 raw score points above that suggested by Weinberger, et. al. (1979), making the upper cutoff equal to or greater than a raw score of 22 and the lower cutoff equal to or less than a raw score of 17.

Experimental Phase

Subjects

Forty-five women, ranging in age from 19 to 21 years (with a mean of 19.36 years) were recruited from students enrolled in undergraduate psychology courses at Virginia Polytechnic Institute and State University. The subjects Cardiovascular Reactivity
were selected (see above) from a larger sample of 135 women students based on their pretest scores on two instruments, (1) The T-Anger subscale of the State-Trait Anger Expression Scale (Spielberger, 1988); (2) the Marlowe-Crowne Social Desirability Scale, used as a measure of denial (Crowne & Marlowe, 1964). Subjects received extra credit in their psychology courses for participation.

Apparatus

Systolic (Phase 1) and diastolic (Phase 5) blood pressure and heart rate (in beats per minute; BPM), were measured using an Axon Medical Systems automated oscillometric blood pressure monitor (Model 820). This device automatically rejects readings contaminated by movement artifacts, and is accurate to within ± 3 mm Hg. A standard sphygmomanometer cuff was used (W. A. Baum Inc., Copiague, N.Y.), for arm circumferences ranging from 23 to 27 cm. Alternative cuffs were used for arm sizes which were outside this range.

A constant-illumination tachistoscope (Lafayette Instruments, Model 42011A), was used to project the stimuli, for viewing by the subject. Error feedback was presented by means of an electronic buzzer, using a duration of 0.1 second.

The stimuli consisted of a set of word matrices patterned like those used in the Stroop Color and Word Test Cardiovascular Reactivity 48
(Golden, 1978). Words which name colors (i.e., red, blue, green) were printed in noncongruent colors, arranged in three columns of five words each, and presented to the viewer on 35mm slides.

Slides were projected on a white screen located at eye level, at a distance of approximately 2.5 meters from the subject. The size of the projected words was calculated to create proportionately the same size image as that given to subjects on standard Stroop stimulus sheets.

**Questionnaire Measures: Experimental Assessment**

When subjects were contacted to schedule appointments for the experimental phase of the study, they were requested to pick up a Health Information Questionnaire, and to bring it with them, completed, to the experimental session (see Appendix C). This included a personal medical history, information on height and weight, familial history of hypertension and/or clinical coronary heart disease, habits of smoking, consumption of alcohol and caffeine, ingestion of prescription and/or street drugs, dietary restrictions, and catamenia.

Subjects were asked to avoid the use of substances which have been shown in previous research to alter physiological reactivity, including caffeine, nicotine and central nervous system stimulant or depressive drugs, for a period of four hours prior to the experimental session.
Immediately prior to the beginning of the experimental stimuli, the complete STAXI, including S-Anger (see Appendix A-2), and the Anger-Expression Scale (Anger-in, Anger-out, and Anger Control, see Appendix A-3) was administered to collect data which might allow for a clearer picture of anger processing in the three subject groups. Factor loadings and alpha coefficients for the AX/In scale, the AX/Out scale, and the AX/Con scale are given in Appendix D. The S-Anger scale (Spielberger, 1988, see Appendix A-4) was given both before and after the experimental conditions to assess changes in anger affect resulted from exposure to the experimental conditions.

The Cook-Medley Hostility Inventory (CMHI; see Appendix E), a 50-item hostility measure, was also administered. Hostility, as measured by this instrument, assesses self-reported prevalence of attitudes which motivate aggressive behavior directed toward injuring other people or destroying property. The reported reliability coefficient is 0.86. (For a more complete description of this scale, see p. 43-44). Each subject signed an informed consent form (Appendix F).

**Procedures**

Each subject was taken to a quiet room and seated at a desk, in front of a microphone which amplified the subject's verbal responses into an adjacent equipment control room. The subject was positioned with limbs partially extended and Cardiovascular Reactivity

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uncrossed. Both arms were supported and positioned at about the level of the fourth intercostal space. Circumference of the subject's left arm was measured, and an appropriately-sized cuff was positioned on the arm with the cuff plug located about 2.5 cm above the antecubital space. Brachial artery location was determined by palpation.

Following placement of the cuff on the subject's arm, a brief training period followed. This was designed to acquaint the subject with the basic procedures to be utilized in the task such as familiarization with the color/word stimuli of the Stroop test, the sound of the feedback buzzer, and testing of the microphone apparatus (see Appendix G). The subject was told that she was taking part in a study of the effects of cognitive processing on her body (see Appendix H). The experimental stimulus, the Stroop Color Word Test (Golden, 1978) was explained to each subject. The subject was told that she would be viewing a series of slides which presented words naming colors in non-congruent colors. She was asked to ignore the words she saw projected on the screen and to name the color in which the words were printed as quickly as possible after the slide presentation. The importance of concentration and accuracy of performance were stressed. To eliminate order and carryover effects, the slides were projected one at a time in a predetermined random order of presentation. The Cardiovascular Reactivity 49
presentation was 0.5 second with a constant interstimulus interval of 2.0 second. To control for potential subject differences in error on the task, error feedback was delivered to subjects randomly, at a ratio of 20% of the total stimulus presentations. In order to alleviate possible suspicion that the feedback was bogus, each subject was told that error messages were delivered by a computer, and were slightly delayed from the occurrence of errors. This served to somewhat confuse the subject, so she was never certain which response was "in error."

At the end of the training period, the subject was asked to sit quietly for three minutes to allow for the obtaining of baseline measures. For each of the three conditions there was an initial three-minute baseline period (Trial 1) followed by three minutes of stimulus presentation (Trial 2). The recovery trial (Trial 3) for the condition presented first functioned as the baseline for the condition presented next, with the order for all three conditions being counterbalanced.

For analysis, the recovery trial for the condition presented first also served as baseline for the condition presented next. This pattern was followed across all conditions. This procedure was followed to minimize the possibility that extraneous thoughts would affect the baseline measures. Pilot data showed that elevations in Cardiovascular Reactivity
reactivity for this sample of young women quickly returned to baseline levels. With recovery periods which exceeded three minutes, spurious spiking effects were noted in blood pressure levels after subjects had returned to baseline. By interviewing women in this pilot sample, it was determined that after a relatively short period of time they started thinking about other things, and that these diversions in thinking produced emotional and physiological reactivity. Since the stimulus conditions were counterbalanced, non-significant variations between levels of baseline and recovery measures were expected to vary out.

The blood pressure monitor was controlled by an experimenter to provide systolic and diastolic blood pressure and heart rate readings at 60-second intervals, beginning with the baseline period, and continuing throughout the experiment.

Subjects participated in three separate conditions. In Condition 1 (no feedback), the subject received 90 stimulus presentations for each condition. The subject received no feedback as to whether she had responded correctly. In Condition 2 (buzzer feedback — no observation), the subject received 90 stimulus presentations, as described above, with "error" feedback on 20% of the total presentations, regardless of the correctness of the response. In Condition 3 (buzzer feedback with observer present in the room with
The subject received 90 stimulus presentations. In this condition, a confederate dressed in a lab coat entered the room with the subject at the beginning of the trial, explaining that she would be scoring the trial manually to "verify calibration of the computerized recording equipment." The observer sat in a chair to the left and in front of the subject and marked a clipboard when an "error" occurred. Following the last condition, the subject was instructed to remain seated and to relax during a recovery period of 3 minutes. Physiological monitoring was continued as previously described during this time.

Following the recovery period, the physiological monitoring equipment was removed from the subject. Each subject once-again completed the S-Anger Subscale of the STAXI. The subject was debriefed and allowed to leave.
CHAPTER 3

Results

A three factor mixed design analysis of variance (ANOVA) was used for this study, with the fixed factor of Group (3) and with the repeated measures of Condition (3) and Trial (3). Three physiological dependent variables, systolic blood pressure, diastolic blood pressure and heart rate, and one performance measure (number of correct responses on the Stroop stimuli per 80-second period) were analyzed using separate ANOVAs. Because one purpose of this study was to assess proportionate changes in physiological reactivity as a function of the experimental task, the raw data for the dependent variables were transformed to percent-change scores according to the following formula:

\[
\text{percent-change score} = \frac{(\text{Trial 2} - \text{Trial 1})}{\text{Trial 1}} \times 100
\]

Trial 1 was equal to the level of the variable of interest during the baseline for each condition and Trial 2 was equal to the level of the variable of interest during the presentation of the experimental stimuli for each condition.

Physiological Data

Systolic Blood Pressure

A two-way (ANOVA) on systolic percent-change scores revealed no reliable main effect, but a reliable Group x Condition interaction, \( F(4, 84) = 3.02, p < .02 \), (see Cardiovascular Reactivity 53
Figure 1). Separate ANOVAs performed by group for each level of condition revealed a main effect of Group, $F(2, 42) = 3.93, p < .02$ for Condition 1. There were no reliable effects for the other two conditions. Duncan's Most Reliable Difference Test (Duncan's MRT), a post hoc comparison of means, was performed on the means for Condition 1, and revealed that both the low anger/high denial group and the high anger/low denial group showed reliably greater reactivity than the low anger/low denial group at $p < .05$ (see Figure 1). No other reliable effects were found, although the low anger/high denial group showed marginally greater reactivity than the high anger/low denial group, with a difference in means of .030 against a criterion value of .039.

Since percent-change scores statistically equate baseline differences between observations, meaningful differences in response rate which occur as a function of trial may be masked. To further assess the effects of the experimental stimuli for each condition, as well as differences in the baseline and recovery values among groups, a separate ANOVA was performed on the raw data for each dependent variable. For systolic blood pressure, there was a reliable main effect for trial, $F(2, 188) = 78.85, p < .0001$. Duncan's MRT showed that the values for systolic blood pressure were reliably greater in Trial 2.
Figure Caption

Figure 1. Systolic blood pressure: Percent-change scores by condition. C1 = no feedback; C2 = feedback, no observer present; C3 = feedback, observer present.
Cardiovascular Reactivity
(experimental stimulus) than in Trial 1 (baseline) or Trial 3 (recovery; see Figure 2). A comparison of baseline values by groups for each condition, using Duncan's MRT at $p < .05$, found that the high anger/low denial group showed reliably higher systolic blood pressure than the low anger/high denial group during the baseline trial for Condition 1. For Condition 3, the high anger/low denial group displayed reliably higher reactivity than both the low anger/high denial group and the low anger/low denial group. No other reliable differences were found. For the recovery trial (Trial 3), the high anger/low denial group evidenced reliably greater systolic blood pressure than the low anger/low denial group in Condition 1 and Condition 2, and was reliably greater than both the low anger/low denial group and the low anger/high denial group during Condition 3.

**Diastolic Blood Pressure**

An ANOVA done on the percent-change score data for diastolic blood pressure revealed no significant main effects for group, $F (2, 42) = 1.69$, $p < .19$. The mean for the low anger/low denial group was 0.060; the mean for the high anger/low denial group was 0.078; the mean for the low anger/high denial group was 0.097. Additionally, no reliable effects were found for condition, $F (2, 84) = 0.51$, $p > .60$. The means for conditions 1, 2 and 3 were 0.077, 0.088,
Figure Caption

Figure 2. Systolic blood pressure: Trial by condition effects. C1 = no feedback; C2 = feedback, no observer present; C3 = feedback, observer present
and 0.073 respectively. Additionally, no interactions were found at a $p < .05$ level of reliability. Analysis of the raw data for diastolic blood pressure yielded a reliable main effect of trial, $F(2, 84) = 68.26, p < .0001$. Duncan's MRT test revealed reliably heightened diastolic blood pressure in Trial 2, as compared with Trial 1 or Trial 3. There were no reliable differences among groups for baseline and recovery values irrespective of condition.

**Heart Rate**

An ANOVA done on the heart rate percent-change scores showed a main effect of condition, $F(2, 84) = 4.98, p < .009$. Duncan's MRT, performed on the condition means, irrespective of group, showed reliably heightened reactivity in Condition 1 (mean = 0.095) and Condition 2 (mean = 0.0986), as compared with Condition 3 (mean = 0.0580), $p > .05$. There were no reliable interaction effects.

Analysis of the raw data for heart rate revealed a reliable main effect of trial, $F(2, 84) = 68.26, p < .0001$. Duncan's MRT confined this effect to Trial 2, where heart rate was consistently elevated over the values taken during Trial 1 and Trial 3. Again, as for the dependent variable DBP, there were no reliable differences among groups for baseline and recovery measures.

**Performance Data**

As a performance measure, the number of correct
responses to the Stroop stimulus presentations was recorded and totaled for each of the three 60-second periods between blood pressure readings during a given condition. This enabled comparison of performance at the beginning, middle, and end of each condition. An ANOVA revealed a reliable main effect of period, $F(2, 84) = 6.48, p < .002$. Duncan’s MRT revealed that performance means for Period 2 (mean = 28.985) and Period 3 (mean = 29.141) were reliably higher than for Period 1 (mean = 28.585), irrespective of condition. Additionally, a Condition x Period interaction was reliable, $F(4, 168) = 2.94, p < .02$. A post hoc analysis using Duncan’s MRT procedure revealed reliably better performance during Period 1 in Condition 3 (mean = 29.87) than during Period 1 in Condition 1 (mean = 28.40) and Condition 2 (mean = 28.10). There were no other reliable effects.

**Relationship of Questionnaire Data/Physiological Data Correlations**

In order to better describe the separate groups, analyses were done on the anger, denial and hostility measures, to determine both the interrelationship of these variables, and also their relationship to the dependent variables. There were observable differences in the patterns of correlation between groups. The strength and direction of the relationship between pairs of variables was determined using the Pearson Product Moment correlation coefficient.
(Pearson r). The results of these analyses are shown in Table 1. For the low anger/low denial group, denial, anger-out, and anger control were all negatively correlated with systolic blood pressure, and hostility was positively correlated with systolic blood pressure, all at $p = <.05$ or less. The high anger/low denial group showed a different pattern, with trait anger and anger-out being strongly and positively correlated with systolic blood pressure, again at the $p = <.05$ level of significance. For the low anger/high denial group, trait anger and hostility were moderately correlated with systolic blood pressure at $p = <.05$ or less.
Table 1

**Correlation Between Behavioral and Physiological Measures**

**Low Anger/Low Denial Group**

<table>
<thead>
<tr>
<th></th>
<th>MC</th>
<th>AT</th>
<th>AI</th>
<th>AO</th>
<th>AC</th>
<th>HO</th>
<th>SYS</th>
<th>DIA</th>
<th>HR</th>
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<tbody>
<tr>
<td><strong>MC</strong></td>
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<td>.43*</td>
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<td><strong>DIA</strong></td>
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<td>.44*</td>
<td>.28</td>
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</table>

* = $p < .05$ or less.

**MC = Denial**  
**SYS = Systolic BP**

**AT = Trait Anger**  
**DIA = Diastolic BP**

**AI = Anger-in**  
**HR = Heart Rate**

**AO = Anger-out**

**AC = Anger Control**

**HO = Hostility**
Table 1 (cont.)

High Anger/Low Denial Group

<table>
<thead>
<tr>
<th>MC</th>
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<th>AI</th>
<th>AO</th>
<th>AC</th>
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<td>.04</td>
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* = p < .05 or less

MC = Denial
AT = Trait Anger
AI = Anger-in
AO = Anger-out
AC = Anger Control
HO = Hostility

SYS = Systolic BP
DIA = Diastolic BP
HR = Heart Rate

Cardiovascular Reactivity
Table 1 (cont.)

Low Anger/High Denial Group

<table>
<thead>
<tr>
<th></th>
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<th>AT</th>
<th>AI</th>
<th>AO</th>
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<th>HO</th>
<th>SYS</th>
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<td>-0.58*</td>
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<td>HO</td>
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<td>0.04</td>
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* = p < 0.05 or less

MC = Denial
AT = Trait Anger
AI = Anger-in
AO = Anger-out
AC = Anger Control
HO = Hostility

SYS = Systolic BP
DIA = Diastolic BP
HR = Heart Rate

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Multiple Regression

A multiple regression analysis was also performed to determine which factors were most predictive of elevations in systolic blood pressure for the individual groups (see Table 2). This was done using a program which tests all possible combinations of variables to determine which is the one-variable model with the highest coefficient of determination. Similarly, the two-variable model is computed, and so forth (Crum, Hastings, & Luginbuhl, 1985).

For the low anger/low denial group, the most reliable predictor of increases in systolic blood pressure was "anger-out". The best coefficient of determination (greatest number of predictors reliable at $p < .05$), included anger-out, denial, anger control and hostility in that order, with a value of 0.55. Heart rate was not as strongly correlated with any combination of these variables, suggesting that variations in heart rate are less closely associated with the effects of self-described methods of coping with anger.

For the high anger/low denial group, the most reliable predictor of increased systolic blood pressure was "anger-in." The best model included anger-in and trait anger in descending order of reliability. The coefficient of determination for this model was 0.42.

For the low anger/high denial group, there was no single variable clearly associated with increases in cardiovascular reactivity.
Table 2

**Systolic Blood Pressure: r2 Prediction Models by Group**

<table>
<thead>
<tr>
<th>Group</th>
<th>Best Model</th>
<th>F</th>
<th>p&lt; F</th>
<th>Best Model</th>
<th>F</th>
<th>p&lt; F</th>
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<tbody>
<tr>
<td>Lo/Lo</td>
<td>One Var.</td>
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<td>Overall</td>
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<td></td>
<td></td>
<td>9.78</td>
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<td></td>
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<td></td>
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<td>4.11</td>
<td>.05</td>
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</tbody>
</table>

One Variable: $r^2 = .185$  
Overall: $r^2 = .55$

MC = Denial  
AO = Anger-out  
AT = Trait Anger  
AI = Anger-in   
AO = Anger-out  
HO = Hostility
Table 2 (cont).

<table>
<thead>
<tr>
<th>Group</th>
<th>Best Model</th>
<th>F</th>
<th>p&lt; F</th>
<th>Best Model</th>
<th>F</th>
<th>p&lt; F</th>
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<tr>
<td>Hi/Lo</td>
<td>One Var.</td>
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<td></td>
<td>Overall</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>AI</td>
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<td>AT</td>
<td>4.74</td>
<td>.04</td>
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<tr>
<td></td>
<td>AI</td>
<td>18.33</td>
<td>.00</td>
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</tbody>
</table>

One Variable: r2 = .35  
Overall: r2 = .42
Table 2 (cont.)

<table>
<thead>
<tr>
<th>Group</th>
<th>Best Model</th>
<th>E</th>
<th>p&lt;F</th>
<th>Best Model</th>
<th>E</th>
<th>p&lt;F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lo/Hi One Var.</td>
<td>AT</td>
<td>6.59</td>
<td>.01</td>
<td>AT</td>
<td>1.80</td>
<td>.14</td>
</tr>
<tr>
<td></td>
<td>AI</td>
<td>1.48</td>
<td>.23</td>
<td>AO</td>
<td>0.66</td>
<td>.42</td>
</tr>
<tr>
<td></td>
<td>HO</td>
<td>1.78</td>
<td>.19</td>
<td>Overall: r² = .20</td>
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</table>

Cardiovascular Reactivity
systolic blood pressure. Correlations were low and not reliable at \( p < .05 \). Trait anger, hostility, anger-in and anger-out were included in the model, and the coefficient of determination was considerably less than for the other two groups (0.20).

Separate one-way ANOVAs were performed on the mean scores, by group, for the self-reported anger measures. This was done in an attempt to more clearly define differences in self-described anger processing which may be associated with differences in reactivity among the three groups. These data are summarized in Table 3. As expected, there were reliable differences between groups on the mode of anger expression, as well as the amount of anger experienced. The high anger/low denial group reported reliably higher state anger on the posttest (mean = 12.933) than the low anger/low denial group (mean = 11.133) and the low anger/high denial group (mean = 10.667). This group also scored reliably higher on anger expression, anger-out, trait anger and hostility than the other groups. For anger control, the low anger/high denial and low anger/low denial groups scored reliably higher than the high anger/low denial group. On the denial measure, the low anger/high denial group scored reliably higher than the other two groups (see Table 3).
Table 3

Comparison of Group Means: Self-Report Measures

<table>
<thead>
<tr>
<th>Group</th>
<th>MC</th>
<th>TA</th>
<th>AI</th>
<th>AO</th>
<th>AC</th>
<th>AX</th>
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<tbody>
<tr>
<td>Lo/Lo</td>
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<td>15.9</td>
<td>14.6</td>
<td>26.2</td>
<td>20.3</td>
<td>17.3</td>
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<td>Hi/Lo</td>
<td>10.7</td>
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<td>18.8</td>
<td>18.6</td>
<td>31.2</td>
<td>24.3</td>
</tr>
<tr>
<td>Lo/Hi</td>
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<td>15.4</td>
<td>12.0</td>
<td>28.5</td>
<td>14.7</td>
<td>14.5</td>
</tr>
</tbody>
</table>

Utilizing Duncan's MRT at $p < .05$, reliable differences between means were as follows:

For MC: LH > LL and HL

TA: HL > LL > LH

AI: No reliable differences

AO: HL > LL > LH

AC: LH and LL > HL; do not differ reliably from each other.

AX: HL > LL > LH

HO: HL > LL and LH; LL and LH do not differ reliably from each other.
CHAPTER 4
Discussion

This study resulted in several major findings. One hypothesis, which was confirmed, was the existence of a group of highly-reactive women, characterized by low scores on a self-reported anger measure and high scores on a self-reported denial measure. This group (low anger/high denial), was predicted to show reliably greater physiological reactivity to distinct stressful conditions than a group of women characterized by low anger and low denial. Results supported this hypothesis in the no-feedback condition, as shown by the Group x Condition interaction for systolic blood pressure. Women who scored low in anger and high in denial were reliably more reactive than those who scored low in both anger and denial. These were also marginally more reactive than women who self-reported high amounts of anger in conjunction with low denial. The fact that the high anger/low denial group and the low anger/high denial group demonstrated the highest systolic blood pressure in the no-feedback condition suggests that performance on the Stroop task, without feedback about the quality of that performance, is more stressful for these highly reactive women than it is for women who are low reactors. Indeed, the low reactor women were least reactive in this condition, supporting the premise that women in the different groups Cardiovascular Reactivity 72
perceive and react to stressful conditions differently. Reactivity for the low anger/high denial women was not reliably greater than that of the high anger/low denial women for this age range. Nevertheless, the fact that they were somewhat more reactive than women who describe themselves as extreme in high anger suggests that denial does not serve a positive function, physiologically, for these individuals.

These findings are potentially important for a number of reasons. First, the low anger/high denial group represents a group of women which has not been identified previously in the literature. Earlier studies of hypertension have suggested that denial of angry or hostile feelings may be related to hyperreactivity (Cottington et al., 1986; Harburg, Blakelock, & Roeper, 1979; Jorgenson & Houston, 1986). However, this appears to be the first time that the denial variable has been used to target groups of women who may react differently to specific stressful conditions, and to predict correctly, high-reactivity as a correlate of high denial. The present study identified a group of individuals who, although they reported low anger, displayed elevated reactivity in response to stress. This reactivity was similar to or greater than that which has been shown previously, using the traditional measures of self-reported high anger and/or hostility alone.

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Some prior studies have suggested that denial may be an adaptive mechanism in certain situations. For example, using a sample of male cardiac patients hospitalized for acute myocardial infarction, Levine et al. (1987) found that those categorized as high deniers, according to a denial interview, spent fewer days in intensive care and had fewer signs of cardiac dysfunction during their hospitalization. In the year following discharge, however, the deniers were more non-compliant with medical recommendations, adapted more poorly to their conditions, and required more days of rehospitalization. Thus the adaptive function of denial in this study appears to have been related to stage of recovery. While initially denial may have allowed for a positive attitude about one's illness and chances for recovery, expediting improvement, later in the course of recovery denial became maladaptive. By functioning to inhibit a realistic appraisal of the situation, it also appears to have limited the development of new adaptive behaviors necessary for continued improvement.

The findings of the present study, similarly, suggest potential health vulnerability for women who do not acknowledge angry feelings. These women may be at equal or greater risk for cardiovascular illness than those who report high levels of anger. As in the study discussed above, the acknowledgement of problematic behavior is
adaptive in that it allows for the possibility of change. Individuals who operate within a denial framework cut themselves off from the possibility of obtaining help, both in terms of modifying stressful life situations, and altering behavioral responses to such situations. This pattern may have especially detrimental health consequences for increasing numbers of women who have recently taken on the stress of full time career employment and are committed to "looking good" in a work situation.

Also very significant is the fact that few previous studies have found differential physiological reactivity in young women. For example, Lawler et. al., (1983, 1984) found that while women of ages 25-55 years showed reliable variations in heart rate and systolic and diastolic blood pressure in response to mental arithmetic and visual puzzles, younger women (18-25 years) were not differentiated using these procedures. It is possible that the paradigm utilized in the present study is capable of tapping differences in reactivity which are relatively modest for young women, but which may become more pronounced with age. Previous comparative studies (e.g. Manuck et. al., 1985) have shown that female monkeys who displayed submissive behavior when subjected to social stress also evidenced greater heart rate reactivity to a laboratory stressor (threat of capture), and greater atherosclerotic occlusion.
upon sacrifice. If atherosclerosis develops similarly in humans, as is widely hypothesized (e.g. Manuck & Krantz, 1986), then this may explain the exacerbated effects of stress on blood pressure and increased peripheral resistance for certain individuals with increased age. Further studies using older women are being conducted to test this hypothesis.

Although the individual groups were not reliably differentiated in the conditions of this study which provided feedback, differences were apparent in directional changes in the reactivity of a given group from one condition to another. If reactivity does increase with age as prior studies suggest, then differences between groups which were not reliable in this study might be expected to become more pronounced with time, especially between the low and high reactor groups.

A further finding of the present study is that the Stroop Color and Word Test as utilized herein, is a useful tool for eliciting and maintaining stress responses over several trials. In general, each group maintained a similar A-B-A pattern of reactivity throughout the entire experimental phase of the study. Future studies should be conducted with different age and occupational groups of females, and also with males, to determine whether this paradigm is capable of inducing stress in broader segments.
of a population. These studies are currently in the planning stages in our laboratory.

The three groups used in this study differed in reactivity to stress, primarily systolic blood pressure. While reliable elevations from baseline were also demonstrated for both diastolic blood pressure and heart rate as a result of the experimental conditions, percentage change from baseline was not sufficient to differentiate between groups or between conditions based on these measures. The one exception was the main effect shown for heart rate, whereby a reliable decrease in reactivity was found across groups for Condition 3, the feedback with observer condition. As has been previously reviewed, other studies have found that heart rate and blood pressure do not necessarily respond similarly to stress. However, probably due to the complexity of the interactions among systems, few studies have attempted to interpret such responses. Direction and magnitude of reactivity appear to be situationally determined, not only by the stressor itself, but also by the subjective perception of the inherent threat and the individual's potential capability to cope with such threat. Lacey and Lacey (1970) found that heart rate decelerated during intake of environmental stimuli, at the same time that blood pressure increased in response to the noxious quality of white noise. It may be that in Condition Cardiovascular Reactivity
3, the observation component created a complex stimulus whereby vigilance was increased due to the subject not wishing to make errors in the presence of the observer. This may have caused an "orienting" or deceleratory heart rate response. Blood pressure, however, may have continued to reflect the relative degree to which the groups found the stimulus itself annoying. Since the actual subject perceptions of both the observer's presence and the Stroop test itself are not known, this premise must be regarded as speculative, especially in light of the differences in blood pressure responses to the three conditions by the three groups. A good deal of further study will be required before such interpretation could be made with confidence.

With the exception of the heart rate decrease in Condition 3, the variables did operate in the same direction in the present study. Systolic blood pressure, however, proved to be a more robust indicator of interaction among the independent variables for these young women. Given an older group of women, it seems possible that diastolic blood pressure might differentiate the anger reported by the high anger/low denial group from the anger not reported or denied by the low anger/high denial group. Prior studies have shown diastolic blood pressure to be a particularly accurate indicator of total peripheral resistance (TPR), and a good indicator of prolonged or chronic stress. With age, Cardiovascular Reactivity
peripheral resistance tends to increase due to some loss of compliance (the ability of the vein to expand or contract in relation to blood flow), particularly in individuals who exhibit chronic systolic activity due to stress. Situational elevations in TPR appear to be more associated with an anger response (Ax, 1953; Schacter, 1957), while elevations in cardiac output (indexed by systolic blood pressure) tend to be more associated with a fear response. If, as the present study suggests, individuals who are more reactive experience more anger than their low reactor counterparts, increased diastolic pressure might result from elevations in TPR as a measure of anger, in combination with lowered compliance. Anger which is present, but is consistently denied by individuals who are fearful of social censure, might be reflected autonomically by substantial increases in both systolic and diastolic blood pressure (reflective of a combined anger and fear response). Stress over time combined with a natural loss of compliance due to aging create increased TPR and elevated diastolic blood pressure. Therefore, the effects of this process would not be expected to occur in young women, but might become evident in an older sample of women. A study (Lawler et al., 1983) that measured the reactivity of a group of women ages 25-55 did find elevations in both systolic and diastolic blood pressure.

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One other finding of this study was of interest. Substantial differences were apparent in both the size and direction of specific correlations between the dependent variables and the self-reported anger and denial variables for the three groups. Additionally, the multiple regression analyses on the individual groups revealed that models comprised of the anger variables most closely associated with elevations in systolic blood pressure varied substantially by group. Also, analysis showed reliable differences in group means on the self-report measures. All of these findings support the hypothesis that the groups identified in this study utilize different ways of coping with anger, and suggest that the methods they use are somehow related to the amount of physiological reactivity they display under stress. However, the nature of these relationships, particularly for the high denial group, is not clear. What does seem evident is that any attempt to draw definite conclusions about the personality characteristics of these groups based on the present evidence would be premature. This information should, however, direct further studies, particularly into the anger coping mechanisms of the high denial group, and their relationship to elevated reactivity.
References


330.


Cannon, W. B., (1914). The emergency function of the adrenal


Dembroski, T. M., Macdougall, J. M., Williams, R. B. Jr., *Cardiovascular Reactivity*


reactions induced by different stressors.

Psychopharmacology, 47, 1-5.


Epidemiology, 107, 362-383.


Manuck, S. B., Kaplan, J. R., & Clarkson, T. B. (1985a). An...


Type A behavior pattern. Psychological Bulletin, 91, 293-323.


parsonspetite. New York, Plenum.


Williams, R. B., Lane, J. D., Kuhn, C. M., Melosh, W., White, A. D., & Schanberg, S. M. (1982). Type A behavior and elevated physiological and Cardiovascular Reactivity
Appendix A

State-Trait Anger Expression Inventory

Trait-Anger Subscale

A number of statements that people use to describe themselves are given below. Read each statement and fill in the circle with the number that indicates how you generally feel. Remember that there are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to best describe how you generally feel: 1) almost never; 2) sometimes; 3) often; 4) almost always.

11. I am quick-tempered.
12. I have a fiery temper.
13. I am a hotheaded person.
14. I get angry when I am slowed down by others' mistakes.
15. I feel annoyed when I am not given recognition for doing good work.
16. I fly off the handle.
17. When I get mad, I say nasty things.
18. It makes me furious when I am criticized in front of others.
19. When I get frustrated, I feel like hitting someone.
20. I feel infuriated when I do a good job and get a poor evaluation.
Appendix A-2

State-Trait Anger Expression Inventory

State Anger Subscale

Following are a number of statements. Please rate each statement according to how you feel right now based on the following scale:

1) Not at all; 2) Somewhat; 3) Moderately so; 4) Very much so.

1. I am furious.
2. I feel angry.
3. I am burned up.
4. I feel like yelling at somebody.
5. I feel irritated.
6. I feel like breaking things.
7. I feel like swearing.
8. I feel like hitting someone.
9. I feel like banging on the table.
10. I am mad.
Appendix A-3

State Trait Anger Expression Inventory

Anger Expression Subscale
Please rate how frequently you behave in the manner described below by rating each statement according to the following four point scale: 1) Almost never; 2) Sometimes; 3) Often; 4) Almost always.

Anger-in Subscale
1. I withdraw from people.
2. I pout or sulk.
3. I am angrier than I’m willing to admit.
4. I am secretly critical of others.
5. I boil inside.
6. I tend to harbor grudges that I don’t tell anyone about.
7. I keep things in.
8. I am irritated a great deal more than people are aware of.

Anger-out Subscale
9. I do things like slam doors.
10. I say nasty things.
11. I make sarcastic remarks to others.
12. I argue with others.
13. I lose my temper.

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14. I strike out at whatever infuriates me.
15. I express my anger.
16. If someone annoys me, I tell him or her how I feel.

Anger-control Subscale

17. I control my temper.
18. I keep my cool.
19. I calm down faster than most other people.
20. I try to be tolerant and understanding.
21. I control my angry feelings.
22. I can stop myself from losing my temper.
23. I am patient with others.
24. I control my behavior.
Appendix B

Marlowe Crowne Social Desirability Scale

Listed below are a number of statements concerning personal attitudes and traits. Read each item and decide whether the statement is true or false as it pertains to you personally.

1. Before voting I thoroughly investigate the qualifications of all the candidates.
2. I never hesitate to go out of my way to help someone in trouble.
3. It is sometimes hard for me to go on with my work if I am not encouraged.
4. I have never intensely disliked anyone.
5. On occasion I have had doubts about my ability to succeed in life.
6. I sometimes feel resentful when I don't get my way.
7. I am always careful about my manner of dress.
8. My table manners at home are as good as when I eat out in a restaurant.
9. If I could get into a movie without paying and be sure I was not seen, I would probably do it.
10. On a few occasions, I have given up doing something because I thought too little of my ability.
11. I like to gossip at times.
12. There have been times when I felt like rebelling.
against people in authority even though I knew they were right.

13. No matter who I'm talking to, I'm always a good listener.

14. I can remember "playing sick" to get out of something.

15. There have been occasions when I took advantage of someone.

16. I'm always willing to admit it when I make a mistake.

17. I always try to practice what I preach.

18. I don't find it particularly difficult to get along with loud-mouthed, obnoxious people.

19. I sometimes try to get even, rather than forgive and forget.

20. When I don't know something I don't at all mind admitting it.

21. I am always courteous, even to people who are disagreeable.

22. At times, I have really insisted on having things my own way.

23. There have been occasions when I felt like smashing things.

24. I would never think of letting someone else be punished for my wrongdoings.

25. I never resent being asked to return a favor.

26. I have never been irked when people expressed ideas.

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very different from my own.

27. I never make a long trip without checking the safety of my car.

28. There have been times when I was quite jealous of the good fortune of others.

29. I have almost never felt the urge to tell someone off.

30. I am sometimes irritated by people who ask favors of me.

31. I have never felt that I was punished without cause.

32. I sometimes think when people have a misfortune they only got what they deserved.

33. I have never deliberately said something that hurt someone's feelings.
Appendix C

Health History Questionnaire

Please fill in the information requested below. Please bring it with you when you come for your experimental session.

Code #: ______________________

Sex: M / F Age _______ Height _______ Weight ______

The purpose of the study in which you have volunteered to participate is to measure the effects of cognitive processing on physiological response. Since many different things may affect these measurements, we ask that you be as honest as possible in answering the following questions.

Your individual identity will be kept confidential.

Do you smoke? ______ Do you drink alcohol? ______

Approximately how many drinks per week? ______ per mo? ______

Do you do physical exercise? Yes/No If yes, approximately how many minutes a week do you exercise? ______ What type of exercise do you do (i.e., walk, jog, bike, swim, other)?

__________________________________________________________

1. Does anyone in your immediate family (mother, father, siblings) have a history of the following:

Hypertension: Yes/No Who? _________________________________

Coronary Heart Disease: Yes/No Who? _______________________

Indicate type if you know it:

Myocardial Infarction

Angina Pectoris

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Atherosclerosis

Do you personally have a history of any of the following? (please circle those that apply to you):

1) Hypertension; 2) Coronary Heart Disease; 3) Headaches; 4) Difficulty eating or sleeping;

Do you have any other medical problems? Yes/No If yes, please explain: __________________________________________
__________________________________________________________________________
__________________________________________________________________________

Do you use caffeine (coffee, cola, chocolate, Excedrine)? Yes/No How often? ______ How much? ______

Please list any medications or drugs you are currently using, and dependencies, if any: ________________________
__________________________________________________________________________
__________________________________________________________________________

Do you have any known neurological disorders? Yes/No If yes, please explain: _____________________________
__________________________________________________________________________
__________________________________________________________________________

Do you have any dietary restrictions? If so, please explain
__________________________________________________________________________
__________________________________________________________________________

For women: What is the approximate starting date of your last menstrual period?_________ How many days is your cycle? _________

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Do you have any history of PMS or other menstrual problems?
Yes/No  If yes, please describe: __________________________
______________________________
______________________________
Appendix D

State-Trait Anger Expression Scale Factor Loadings,

Eigenvalues and Alpha Coefficients

<table>
<thead>
<tr>
<th>Abbrev. Item</th>
<th>Anger-in</th>
<th>Anger-out</th>
<th>Anger</th>
</tr>
</thead>
<tbody>
<tr>
<td>Keep my cool</td>
<td></td>
<td></td>
<td>.73</td>
</tr>
<tr>
<td>Control my behav.</td>
<td></td>
<td></td>
<td>.69</td>
</tr>
<tr>
<td>Control angry feel.</td>
<td></td>
<td></td>
<td>.71</td>
</tr>
<tr>
<td>Tolerant and understanding</td>
<td></td>
<td></td>
<td>.69</td>
</tr>
<tr>
<td>Patient w/others</td>
<td></td>
<td></td>
<td>.63</td>
</tr>
<tr>
<td>Control temper</td>
<td>-.40</td>
<td></td>
<td>.63</td>
</tr>
<tr>
<td>Calm down faster</td>
<td></td>
<td></td>
<td>.52</td>
</tr>
<tr>
<td>Stop self from temper</td>
<td></td>
<td></td>
<td>.62</td>
</tr>
<tr>
<td>Slam doors</td>
<td>.49</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Make sarcastic remarks</td>
<td></td>
<td>.52</td>
<td></td>
</tr>
<tr>
<td>Say nasty things if someone annoys me</td>
<td>.57</td>
<td></td>
<td>-.36</td>
</tr>
<tr>
<td>Express my anger</td>
<td></td>
<td>.67</td>
<td></td>
</tr>
<tr>
<td>Lose my temper</td>
<td></td>
<td>.53</td>
<td></td>
</tr>
<tr>
<td>Strike out at whatever annoys me</td>
<td></td>
<td>.58</td>
<td></td>
</tr>
</tbody>
</table>

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Appendix E

Cook-Medley Hostility Inventory

If a statement is true or mostly true, as applies to you, mark it with a T. If a statement is false, or usually not true about you, mark it with an F. Please answer every statement.

1. When I take a new job, I like to be tipped off on who should be gotten next to.

2. When someone does me a wrong I feel I should pay him back if I can, just for the principle of the thing.

3. I prefer to pass by school friends, or people I know but have not seen for a long time, unless they speak to me first.

4. I have often had to take orders from someone who did not know as much as I did.

5. I think a great many people exaggerate their misfortunes in order to gain the sympathy and help of others.

6. It takes a lot of argument to convince most people of the truth.

7. I think most people would lie to get ahead.

8. Someone has it in for me.

9. Most people are honest chiefly through fear of getting caught.
10. Most people will use somewhat unfair means to gain profit or an advantage rather than to lose it.

11. I commonly wonder what hidden reason another person may have for doing something nice for me.

12. It makes me impatient to have people ask my advice or otherwise interrupt me when I am working on something important.

13. I feel that I have often been punished without cause.

14. I am against giving money to beggars.

15. Some of my family have habits that bother and annoy me.

16. My relatives are nearly all in sympathy with me.

17. My way of doing things is apt to be misunderstood by others.

18. I don't blame anyone for trying to grab everything he can get in this world.

19. No one cares much what happens to you.

20. I can be friendly with people who do things which I consider wrong.

21. It is safer to trust nobody.

22. I do not blame a person for taking advantage of someone who lays himself open to it.

23. I have often felt that strangers were looking at me critically.

24. Most people make friends because friends are likely to be useful to them.

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25. I am sure I am being talked about.

26. I am likely not to speak to people until they speak to me.

27. Most people inwardly dislike putting themselves out to help other people.

28. I tend to be on my guard with people who are somewhat more friendly than I had expected.

29. I have sometimes stayed away from another person because I feared doing or saying something that I might regret afterwards.

30. People often disappoint me.

31. I like to keep people guessing what I'm going to do next.

32. I frequently ask people for advice.

33. I am not easily angered.

34. I have often met people who were supposed to be experts who were no better than I.

35. I would certainly enjoy beating a crook at his own game.

36. It makes me feel like a failure when I hear of the success of someone else.

37. I have at times had to be rough with people who were rude or annoying.

38. People generally demand more respect for their own rights than they are willing to allow for others.
39. There are certain people whom I dislike so much that I am inwardly pleased when they are catching it for something they have done.

40. I am often inclined to go out of my way to win a point with someone who has opposed me.

41. I am quite often not in on the gossip and talk of the group I belong to.

42. The man who had most to do with me when I was a child (such as my father, step-father, etc.) was very strict with me.

43. I have often found people jealous of my good ideas just because they had not thought of them first.

44. When a man is with a woman he is usually thinking about things related to sex.

45. I do not try to cover up my poor opinion or pity of a person so that he won't know how I feel.

46. I have frequently worked under people who seem to have things arranged so that they get credit for good work but are able to pass off mistakes onto those under them.

47. I strongly defend my own opinions as a rule.

48. People can pretty easily change me even though I thought that my mind was already made up on a subject.

49. Sometimes I am sure that other people can tell what I am thinking.
50. A large number of people are guilty of bad sexual conduct.
Appendix F

Statement of Informed Consent

INFORMED CONSENT TO PARTICIPATE IN RESEARCH

Virginia Polytechnic Institute and State University,
Blacksburg, Va. 24060

You are being asked to volunteer as a participant in a research study. This form is designed to provide you with information about the study and to answer any of your questions.

Title of Research Study: Anger and denial as predictors of female cardiovascular reactivity.

Project Director: Dr. David W. Harrison
Phone: 961-4422

PURPOSE OF THE RESEARCH: To evaluate your physiological responsiveness to cognitive processing.

EXPERIMENTAL PROCEDURE: You may be asked to fill out several questionnaires. Some of the questions may be personal in nature. If they are too personal, you do not have to answer them. You may be asked to perform as quickly as possible on a task requiring you to differentiate color words from colored print and to report the information to an experimenter.

RISKS: If you wish to discuss these or any other discomforts you may experience, you may call the project director.

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Because most of the tests are mainly evaluative, there are no serious risks involved. You may cease participation in the experiment at any time. The session should last for approximately one hour.

POTENTIAL BENEFITS: By participating in this study, you may gain new knowledge of experimental design or protocol which might prove helpful in the future. If you wish to know the results of your tests, you may receive this information at the end of this study.

ALTERNATE TREATMENT OR PROCEDURE: No alternate procedures will be used.

If the data from this experiment cannot be treated in such a way as to maintain the anonymity of the subjects, then it will not be used for any purpose other than those we have described without their consent. Subjects in this experiment will not be identified by name or other identifiers, and personal data will remain confidential.

EXCEPTION: If a potential for self-injurious or life-threatening behavior is indicated by the results of this experiment, such results will be reported to an appropriate agency.

This project has been approved by the Human Subjects Research Committee and the Institutional Review Board.

Any questions you may have about this experiment should be directed to Dr. David W. Harrison, Ph. 961-4422.
I hereby agree to voluntarily participate in the research project described above and under the conditions described above.

Signature of participant: ____________________________
Date: ____________________________
Appendix G

**Experimental Protocol**

1. The subject is taken into the lab, seated, and given a letter of informed consent to read and sign. Following this, she is given the STAXI and the MCSD inventories to complete.

2. The following information is given the subject by the experimenter:

   You are taking part in a study of the effects of cognitive processing on your body. To monitor these effects, we will be measuring your heart rate and blood pressure periodically throughout the procedure. All of these measures will be taken automatically. It is not a painful procedure, and is usually not upsetting or annoying. [At this point, measure subject's left arm, locate brachial artery and fit cuff to subject.] It is important that you try to keep your body as still as possible during the measurement procedure to avoid unnecessary delays.

   I am now going to show you several training slides which are similar to the experimental task. Please respond as quickly as you can by naming what you are asked to name.

   In the first group, please read the word. In the second group, tell me the color you see.
Over the next few minutes, you will be seeing a number of slides which consist of a word printed in a color (e.g., the word green printed in "blue"). When I say "start," please name just the color that you see. Please do this as rapidly as you can, and if you miss one, keep going.

Sometimes you will be given error feedback by means of a tone which sounds like this (demonstrate buzzer). The tone is to give you information on how you are doing. Error messages are slightly delayed, therefore they do not correspond to your most recent response. Keep responding when you hear the tone. You will have time neither to identify nor correct your errors.

Now, please relax and sit quietly for the next three minutes. I will tell you when it is time to begin the next part of the experiment.
The __7__ page vita has been removed from the scanned document
The vita has been removed from the scanned document
The vita has been removed from the scanned document
The vita has been removed from the scanned document
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