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Cognitive Stress and Cardiovascular Reactivity: Relationship to Hypertension

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1. Overview

Cardiovascular reactivity refers to the difference (Δ) in heart rate, blood pressure or other measures of cardiovascular function observed between periods of rest and during the presentation of an external stressor. Reactivity may be conceptualized as a quantitative, physiological variable, such that certain individuals will show greater reactivity than others. The majority of reactivity studies have measured changes in heart rate and systolic blood pressure. To a lesser extent, reactivity has also been studied by measuring changes in ST segment depression of the ECG, rate pressure product, pulse pressure, pulse transit time, cardiac output, total peripheral resistance and left ventricular ejection time, as well as other measures of cardiac performance and circulating levels of catecholamines and cortisol. The paradigms for studying reactivity have involved a broad class of experimental stressors and designs.

The evaluation period used to establish resting levels of physiological activity may be a separate relaxation baseline, conducted on a different day than the stress session, or it may be a pre-stress baseline, conducted on the same day as the stress session. The physical stressors may involve the cold pressor test, isometric hand grip and exercise. Cognitive stressors may involve one or more of the following: mental maths, the Stroop Color Word Test, complex analytical or concept formation tasks, a stressful interview, i.e., a structured interview for Type A behaviour, choice reaction time, video games, or naturalistic stress, e.g., job environment.

The saliency of the situation for a particular subject may be enhanced by including such factors as pressure to respond quickly, threat of aversive consequences for errors, e.g., shock, incentives for successful completion of the task, competition and harassment. Different methodological permutations of task and measurement characteristics may elicit patterns of arousal, which accentuate reactivity in one or another measure(s). Suffice it to say, however, that the extent of a subject's reactivity to a particular measure, e.g., heart rate (HR) response to mental maths or a video game, is usually reproducible and stable over time. Correlations between different response measures on different tasks are more variable, but moderately high (Krantz and Manuck, 1985).

Unless otherwise specified, this review will concentrate on studies of cardiovascular reactivity using cognitive stressors. Cognitive stressors may represent an important laboratory analogue which can be used to assess the effects of acute psychological stress on the cardiovascular system. Laboratory stressors are easily administered and the physiological changes which accompany them may have relevance for the future development of cardiovascular disease.

The relationship between laboratory induced cognitive stress and cardiovascular reactivity has been studied in several ways. Early research examined the extent of reactivity in people with manifest cardiovascular disease. These studies typically did not involve comparisons to normal controls but were aimed at linking the presence of an abnormal cardiac event, e.g., excessive oxygen consumption or hypertension, to acute cognitive stress. Later studies of reactivity did include comparisons between normals and patients with cardiovascular disease. It soon became clear that the magnitude of stress induced changes in blood pressure or heart rate was not uniform across all subjects and that certain otherwise healthy subjects, as well as some patients, could be described as cardiovascular reactors. The central question, however, is whether the high reactors are at increased risk for the development of future cardiovascular disease.

This chapter reviews the pathophysiological and epidemiological significance of acute cardiovascular reactivity as it relates to cardiovascular disease. Long-term prospective studies bridging the gap between the initial observation of excessive reactivity to acute stress and the development of future cardiovascular disease are at present few in number. However, several hypotheses have been advanced which link the acute effects of cognitive stress, e.g., increased heart rate, catecholamines, etc., to known pathogenetic factors in cardiovascular disease. Research on subjects who do not yet have manifest cardiovascular disease, but who are excessively reactive to stress, may be particularly important since it could reveal the mechanisms which link the pathophysiology of future diseae to the episodic stress experienced during the preclinical period of a disorder. The reader is referred to the reviews of Krantz and Manuck (1984, 1985) for an in-depth discussion of reactivity research design and methodological issues.

2. Relevance of reactivity to cardiovascular disease: epidemiological, family history and experimental considerations in hypertension

Research on reactivity has the potential for making a unique contribution to the study of hypertension. Traditional epidemiological studies have focused on the association between resting levels of a suspected risk factor, such as heart rate, cholesterol or blood pressure, and the future development of cardiovascular disease. On the other hand, studies of reactivity have attended to the magnitude of change for a particular cardiac event during periods of rest and behavioural (or physical) challenge. The focus on change versus casual measures may be an important distinction. For example, recent studies of hypertension (Perloff *et al.*, 1983) have indicated that average ambulatory blood pressure (BP) readings may be more predictive of future hypertensive complications than casual blood pressure measures. This finding might be due to the inclusion

of the daily stress-related blood pressure peaks (reactivity) in the ambulatory readings. However, casual BP measurement is the most typically used method for assessing future blood pressure status.

A positive association between reactivity and future hypertension is also partially supported by research showing that BP obtained during work (Devererex *et al.*, 1983) is more predictive of future hypertension than BP obtained at rest. A most interesting prospective example of this comes from the Air Traffic Controller Study (Rose *et al.*, 1978; Jenkins *et al.*, 1984), which showed that relative to rest, the BP of some controllers rose dramatically while they were actually controlling aircraft. The magnitude of the BP rise (reactivity) was predictive of future hypertension. In the only other prospective study in this area, Falkner *et al.* (1981) noted that the degree of systolic blood pressure reactivity to mental mathematics among borderline adolescent hypertensives was predictive of future hypertension, five years later.

The presence of enhanced reactivity to psychological stressors among borderline hypertensives who later develop hypertension suggests that reactivity may act synergistically with other risk factors, or that it may act as a trait marker for subjects at preclinical levels of the disorder. Support for this argument has been received in studies which show enhanced blood pressure (or heart rate) reactivity among adult (Light and Obrist, 1980) and adolescent (Falkner *et al.*, 1979) normotensive males with a positive family history of hypertension. The degree of BP reactivity appears to increase when both parents are hypertensive (Hewitt *et al.*, 1982).

Recently, Manuck *et al.* (1981) helped clarify the relationship between reactivity and family history of hypertension by evaluating heart rate and blood pressure reactivity to a concept formation task (Feldman Visual-Verbal Test). Relative to a resting baseline, subjects with a parental history of hypertension showed a significant rise in systolic blood pressure (+20 mm Hg). Subjects showing a high heart rate reactivity (+18 bpm) and a positive family history of hypertension showed a larger SBP rise than those without a family history of hypertension. Among low heart rate reactors, with and without a parental history of hypertension, only modest and non-significant SBP changes were observed. Results for diastolic blood pressure showed a significant effect of the stress task, but no reactor/family history interaction was observed.

Thus, cardiovascular reactivity appears to be enhanced in subjects with a parental history of hypertension. However, no difference in SBP reactivity is observed among low heart rate reactors, with or without a family history of hypertension. A parental history of hypertension seems to act synergistically with heart rate reactivity when reactivity is observed. But, a history of parental hypertension does not necessarily ensure that high levels of cardiovascular reactivity will be observed. Perhaps heart rate reactivity among normotensives contributes something to the overall risk of hypertension irrespective of a hypertensive family history. While the data are far from complete on this matter, these findings suggest that low levels of HR reactivity may be associated with a suppression of the SBP reactivity that might be expected in the offspring of hypertensive parents. Low heart rate reactivity may also be associated with lower casual BPs and perhaps a lower rate for developing future hypertension in this same population.

The relationship between essential hypertension and cardiovascular reactivity during

cognitive stress has also been evaluated by assessing the blood pressure reactivity among established hypertensives. If reactivity has some bearing on the development of hypertension, then one might expect hypertensive patients to be more reactive to environmental stressors than normotensives tested under the same conditions. For example, Schulte and Neus (1983) studied 10 mild hypertensives (BP > 140/90 mm Hg), borderline hypertensives (120/80 – 140/90 mm Hg) and normotensives (120/80 mm Hg or less), during both resting and mental maths conditions. All groups significantly increased in systolic blood pressure and mean arterial pressure, from resting to stress conditions. The hypertensives showed the strongest rise in systolic and diastolic pressure (15–17 mm Hg) as well as stroke volume. In a related study, Schulte *et al.* (1984) evaluated 30 hypertensives and 13 normotensives in separate relaxation baseline and stress sessions. The stressor involved mental maths, performed under time pressure and 90 dB noise distraction. The results showed that SBP variability during the relaxation baseline correlated significantly with SBP reactivity between the stressor, and the pre-stress baseline assessment conducted on the same day. The hypertensives were also more variable at rest and more reactive during stress than the normotensives. This work extends and confirms previous findings suggesting that reactivity to cognitive stressors among high normotensives is highly correlated with variability in blood pressure measures obtained outside the laboratory (Mañuck *et al.*, 1979). It should be noted, however, that there is some question regarding the exact relationship between reactivity and resting BP variability among established hypertensives. Some studies have reported that established hypertensives may not be more variable in resting BP (Julius *et al.*, 1983) than non-hypertensives. In comparison to normotensives, we may expect a higher proportion of hypertensives to be abnormally reactive (Schulte *et al.* 1984). Resting BPs, however, may not be more labile among the hypertensives, except for those subjects in which high levels of reactivity can be demonstrated.

In a study designed to directly assess the reactivity of labile hypertnesives, Steptoe *et al.* (1984) evaluated 12 male normotensives (BP <120/80 mm Hg), 12 labile hypertensives (see below) and 12 mild hypertensives: (175/105 – 145/90 mm Hg at rest). Blood pressures were obtained in and out of the laboratory environment. After the initial screening, some subjects also self-monitored their blood pressures. The labile hypertensives were subjects whose blood pressures exceeded 145/90 mm Hg at initial screening, then dropped below this limit during subsequent self-monitoring and resting assessments. The Stroop Color Word Test (Stroop, 1935), a challenging video game (i.e., 'pong') and a stressful film were presented to all subjects. The results showed that mild hypertensives experienced significantly more SBP reactivity (+ 23 mm Hg) than normotensives (+ 17 mm Hg) to the video game and the Stroop, but not to the film. The film was considered a passive stress condition and may not have evoked sympathetic arousal sufficient enough to sustain large blood pressure changes. Labile hypertensives also showed the greatest reactivity in diastolic blood pressure, heart rate and pulse transit time: a measure which is positively correlated with SBP during β-adrenergic driving (Obrist *et al.*, 1979). Thus, in at least three separate studies, exaggerated cardiovascular reactivity has been identified among borderline (labile) and established hypertensives during the presentation of a cognitive

stressor. It would also appear that an elevated casual BP is not necessarily indicative of enhanced lability in reacting BPs. However, BP reactivity to stress may be positively correlated with resting BP lability.

3. Psychophysiological relationships of reactivity and hypertension

It is intriguing to think that essential hypertension may proceed to established hypertension though an imposition of psychological factors on the proper biological substrate. A major assumption behind this reasoning is that the episodic psychological stress can impact significantly on the pathophysiology of hypertension. As yet, this assumption is only beginning to receive support in the epidemiological literature. Prospective studies of high and low reactors, which target important features of hypertensive pathophysiology, have not yet been done. However, the endocrine and psychophysiological concomitants of reactivity may indeed influence several of the known pathogenetic mechanisms related to hypertension. For example, the pressor effects of acute stress could have implications for altering vessel morphology. The work from Folkow's laboratory (Folkow, 1977) suggests that repeated pressor episodes may lead to arteriolar hypertrophy and to changes in the wall to lumen ratio—altering geometry and rendering the vessels yet more responsive to future pressor stimuli. In time, a positive feedback loop may be established, through which vascular hypertrophy and pressor responsiveness are increased, resulting in increased peripheral resistance and blood pressure. This is an extremely important point since it helps to explain how the excessive cardiac output seen in the high reactor may be related to the enhanced peripheral resistance, characteristic of the established hypertensive. Acute reactivity in the pre-hypertensive state may directly influence the process of normalizing cardiac output in the hypertensive, while producing long-term elevations in peripheral resistance (Julius *et al.*, 1983). Reactivity tied to psychological stressors has the potential of producing frequent and pervasive elevations in cardiac performance. The reactivity appears unique to situations which have some behavioural significance and is not a ubiquitous feature of hypertension. This idea is supported by the fact that borderline and mild hypertensives are not uniformly more labile in resting BP, nor are they excessively responsive to exercise or cold pressor testing (cf. Julius *et al.*, 1983, for an extensive review). Thus, the possibility may be entertained that individuals predisposed to develop hypertension are not generally hyperresponsive, but may be more frequently exposed to pressor stimuli, which have pathognomonic consequences.

The most frequently occurring class of these pressor stimuli which may have significance for the pathophysiology of hypertension may indeed be psychological in nature. Both the magnitude and the extent (frequency) of a person's reactivity may be directly related to the risk of a hypertensive disorder (Manuck *et al.* 1978). The reactivity data of Falkner *et al.* (1979, 1981) are particularly interesting in this regard, since enhanced SBP hyperresponsiveness to a psychological stressor, e.g., mental stress, was demonstrated among normotensive and hypertensive offspring of hypertensive parents. As indicated above, subjects hyperresponsive to mental stress show a higher likelihood of being hypertensive at a 5-year follow-up than subjects low in reactivity

(Falkner *et al.*, 1981). The enhanced BP observed for the air traffic controllers (Jenkins *et al.*, 1984) during aircraft direction may possibly be related to the psychologically stressful components of this activity. It will be recalled that the risk of hypertension in the controllers was positively correlated with the magnitude of work-related reactivity. Light and Obrist (1980) also found enhanced heart rate and BP reactivity to mental stress (reaction time) among borderline hypertensives, while Hollenberg *et al.*, (1981) found normal BP but an excessive renal vasoconstriction to a cognitive stressor (maths) among similar groups.

Presumably, more frequent exposure to an event producing reactivity, e.g., adrenergically mediated psychological stress, results in a higher likelihood of cardiovascular damage and the development of hypertension. The importance of reactivity in the development of hypertension may he highlighted by the fact that only 27% of individuals in the upper quartile of all known hypertension risk factors develop hypertension 5 years into the future (Julius *et al.*, 1983). It has been suggested that factors related to psychological alertness or mental engagement could result in chronic elevations in sympathetic tone and a slow but persistent rise in BP (Julius *et al.*, 1983). Alteration in vascular smooth muscle morphology has been proposed as one consequence of elevated sympathetic activity (Folkow, 1977). However, other related hypotheses have been proposed to explain the pathophysiology of hypertension. Each hypothesis emphasizes different physiological processes. However, converging lines of evidence may link psychologically based changes in reactivity to the mechanisms normally regulating BP.

The patterns of sympathetic arousal produced during psychological (behavioural) stress have been extensively studied. Most of the stress induced elevations in cardiac performance have been related to enhanced β-adrenergic stimulation (e.g., Glass *et al.*, 1980, 1981; Obrist, 1981). For example, studies of normotensives (Obrist *et al.*, 1979) experiencing psychological stress (threat of shock) have shown an increased sympathetic influence on the myocardium, e.g., raised cardiac contractility, and heart rate. These effects like the cardiac output increases observed in some borderline hypertensives may be abolished by β-blockade (Obrist, 1981; Julius *et al.*, 1983). Thus the contention that reactivity and hypertension share a strong neurogenic component is supported.

Excessive sympathetic drive has also been noted among coronary-prone individuals (Type A) during certain types of psychological stress. Research on the salient features of the Type A–environment interaction further implicate adrenergic hyperresponsiveness in the pathophysiology of cardiovascular disease. For example, Type A's have shown enhanced reactivity during mental stress in measures of blood pressure, heart rate, forearm blood flow, catecholamine and cortisol secretion (*see* Matthews, 1982; Cinciripini, 1984). Epidemiologically, the Type A pattern has not been consistently related to hypertension (Jenkins *et al.*, 1984) but it has been related to atherosclerosis (Bloom and Herd, 1983), and electroconductive disturbances, which may be the sequelae of acute hypertensive episodes.

Psychological stress could also play an important role in the aetiology of hypertension, through its effect on the regulation of water and sodium balance. For example, Guyton and Coleman (1969) propose that, under normal circumstances,

increased blood pressure leads to diuresis, lowered blood volume and subsequently reduced blood pressure. Hypertension may result from an increased threshold for renal pressure diuresis, such that higher and higher pressure levels are required in order to initiate a volume correction. Initially, the higher threshold may be associated with volume expansion and/or increased venous return which results in elevated cardiac output. The elevations in cardiac output may set in motion a process of autoregulation, whereby vascular resistance increases in order to protect the tissues from overperfusion (Obrist, 1981) resulting from delivery of oxygen in excess of metabolic demand. The work of Obrist and his colleagues (Obrist, 1981; Light and Obrist, 1983) suggests that conditions characterized by active coping with a mental stressor, e.g., control over the delivery of aversive shocks by speedy reaction time, may be likely to raise cardiac output levels high enough to trigger autoregulation. This idea has particular appeal from the standpoint of cognitive induced reactivity since numerous studies have demonstrated that clear and, in some cases, sustained increases in cardiac output, may result from exposure to psychological stress (*see* Glass *et al.*, 1980; Bloom and Herd, 1983; Krantz and Manuck, 1985; McKinney *et al.*, 1985). Such changes in cardiac output have the potential for triggering autoregulation, subsequent vasoconstriction, and enhanced blood pressure.

It should be noted that Julius *et al.* (1983) have questioned the applicability of the concept of autoregulation to the development of essential hypertension. They note that the time constant for the development of volume expansion is variable, and among boderline hypertensives, relative, but not absolute, hypervolaemia can be demonstrated. This is contrary to the expected increase in blood volume which might be predicted if autoregulation was taking place in the borderline, or pre-hypertensive state. Julius *et al.* (1983) have also criticized the evidence supporting increased renal thresholds, suggesting that borderline and established hypertension may show increased diuresis, but the blood pressure increases expected in response to volume loading are not consistently observed.

As an alternative to the autoregulation hypothesis, Julius *et al.* (1983) have suggested that a disruption in the autonomic control of the cardiovascular system may also be important in the development of hypertension. The work of Julius *et al.* (1983) has involved studies of borderline patients in a hyperkinetic state. These hypertensive patients show increased cardiac output, and increased β-adrenergic driving. Increased sympathetic discharge is suggested as the key factor in the conversion from the stages of early to established hypertension (Julius *et al.*, 1983). As previously discussed, the early phases of hypertension may be characterized by an increased cardiac output. Established hypertension, however, involves a normokinetic state, characterized by increased peripheral vascular resistance and an absence of excessive pressor episodes. Julius *et al.* (1983) suggest that excessive sympathetic stimulation, without autoregulation or volume expansion, may result in the down-regulation of adrenergic receptors in the myocardium. Combined with structural changes in the cardiac tissue (hypertrophy), the reduced sensitivity of cardiac β-adrenergic receptors results in a return to normal cardiac output. In the arterioles, down-regulation of the α-receptors is offset by the structural changes in the vessel wall (hypertrophy) brought about by an increase in the wall to lumen ratio. Julius proposes that a positive feedback loop develops in

which less and less sympathetic stimulation is required to initiate the same level of vasoconstriction. In effect, the level of SNS tone is enhanced. Julius contends (Julius *et al.*, 1983) that excessive sympathetic drive (reactivity) to the heart and increased parasympathetic inhibition results from an abnormal central integration of cardiovascular autonomic tone, which is probably related to behavioural factors, e.g., recurrent episodic stress.

4. *Conclusion*

There are several possible avenues through which a hyperresponsiveness to mental stress can be implicated in the development of hypertension. This process may involve direct influence on the sympathetic nervous system, function and cardiovascular performance, and the renal regulation of water and sodium balance. The data suggest that reactivity may be more important that static BP measures in determining who develops hypertension, and that the frequency of pressor episodes may be related to psychologically induced stress. Highly reactive individuals may be at greater risk of developing hypertension, since they may potentially be exposed to a higher frequency of pressor episodes and the pathogenetic sequelae which follow.

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