Hypertension

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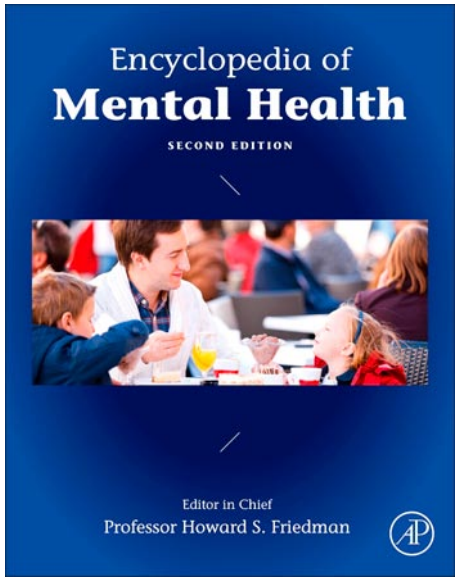

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Hypertension

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Glossary

Ambulatory blood pressure monitoring A method of obtaining regular blood pressure measurements in an individual's natural environment.
Cardiovascular reactivity The magnitude and duration of cardiovascular responses (e.g., heart rate and blood pressure) to stress.
Emotional defensiveness Attempts to minimize or avoid the experience of emotions.
Lifestyle modification Promoting healthy lifestyles through a combination of diet, physical exercise, and other health behaviors.
Masked hypertension Cases in which an individual's blood pressure is within the hypertensive range in the natural environment, but is within the normal range in clinic settings.
Negative affect Experience of subjective distress, which is typically characterized by emotions such as anger, depression, or anxiety.
Suppressed hostility Controlling or suppressing the expression of hostility or anger.
White coat hypertension Cases in which an individual's blood pressure is within the hypertensive range in clinic settings, but is within the normal range in the natural environment.

High blood pressure, clinically known as hypertension, affects more than 75 million Americans, an estimated 1 billion individuals worldwide, and is a serious, life-threatening health condition (Go *et al.*, 2013; World Health Organization, 2013). The prevalence of hypertension is expected to increase annually and already costs billions of dollars in healthcare costs and lost worker productivity. Hypertension is a primary risk factor for a number of chronic health conditions, including cardiovascular diseases (e.g., heart attack, heart failure, and stroke) and kidney diseases (Kannel, 1996; Rapsomaniki *et al.*, 2014). The economic costs, prevalence, and associated long-term health risks confirm that hypertension is a serious global health issue.

Definition and Types of Hypertension

The circulatory system is responsible for providing oxygen and other nutrients essential for life to all organs in the body. When sustained high blood pressure occurs in the circulatory system, one of two types of hypertension can be diagnosed: essential hypertension and secondary hypertension. In the 90–95% of cases in which no identifiable cause of elevated blood pressure can be discerned, the condition is called essential hypertension. In contrast, secondary hypertension results from an identifiable cause, like presence of underlying kidney disease, coarctation (narrowing) of the aorta, metabolic disorders, or specific enzyme deficiencies.

Diagnosis of hypertension depends upon levels of systolic blood pressure (SBP), the highest pressure during a typical cardiac cycle that occurs when the heart is contracting, and diastolic blood pressure (DBP), the lowest pressure during the cycle when the heart is gradually filling with blood in between contractions. The seventh report of the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure describes standard guidelines for diagnosing hypertension (National Heart, Lung, and Blood Institute, 2004). Normal blood pressure is indicated by a SBP of <120 mm Hg and a DBP of <80 mm Hg. Prehypertension is defined as blood pressure values between 120–139/80–89 mm Hg. Hypertension, defined as blood pressure $>140/90$ mm Hg, consists of two stages: stage 1 hypertension, for blood pressures between 140–159/90–99 mm Hg, and stage 2 hypertension, for blood pressures that exceed 160/100 mm Hg.

Pathophysiology of Hypertension

Multiple systems and physiological mechanisms are involved in maintaining normal blood pressure, with the two primary determinants being cardiac output and total peripheral resistance. Cardiac output refers to the amount of blood flow pumped by the heart each minute and is affected by both heart rate and stroke volume (amount of blood ejected from the heart during each contraction). Increases in either heart rate or stroke volume will result in increased cardiac output and thereby increased blood pressure. Total peripheral resistance refers to the amount of force affecting resistance to blood flow throughout the circulatory system. As blood vessels constrict, resistance to blood flow increases, but as these vessels dilate, peripheral resistance declines. Blood pressure is then affected by changes in cardiac output, total peripheral resistance, or by changes in both cardiac output and total peripheral resistance. Regulation of cardiac output and peripheral resistance is influenced by systems outside of the circulatory system, including the autonomic and central nervous systems and the renal system. For example, increases in the sympathetic branch of the autonomic nervous system result in increased blood pressure and heart rate, whereas increases in the parasympathetic branch of the autonomic nervous system typically result in the opposite effect. Sympathetic activity also leads to reabsorption of salt and water by the renal system, which increases blood pressure. Additionally, sympathetic activity is

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associated with increased secretion of renal hormones like Angiotensin II and aldosterone that lead to elevated blood pressures. Prolonged sympathetic activity also results in vascular remodeling of blood vessels that results in thickening and hardening of the vessel walls (Heilpern, 2008).

The central nervous system exerts control over other systems involved in blood pressure regulation (e.g., circulatory, autonomic nervous, and renal systems) through feedback processes. For example, baroreceptors, located in the carotid artery and aortic walls, detect pressure changes in the arteries and signal the brain to activate either the sympathetic or parasympathetic nervous system to raise or lower blood pressure, respectively. Sustained elevations of blood pressure that occur in patients with essential hypertension eventually damage various organ systems in the body. Specifically, damage to the heart (e.g., left ventricular hypertrophy (thickening), angina or myocardial infarction, coronary revascularization, and heart failure), the brain (stroke or transient ischemic attack and dementia), the kidney (e.g., chronic kidney disease and renal failure), peripheral arterial disease, and retinopathy may occur in response to sustained elevated blood pressures (Kannel, 1996; Rapsomaniki *et al.*, 2014). This damage occurs mainly as the result of changes in the elasticity of blood vessels. Through exposure to chronically elevated pressures, vessel walls lose elasticity, making them more susceptible to cellular injury. As the body attempts to repair these injuries, cholesterol and fat deposits interact with inflammatory responses to restrict blood flow or occlude it completely.

The negative effects of hypertension extend to cognitive functioning, as individuals with hypertension are more likely to develop dementia or cognitive impairment than persons with normal blood pressures (National Heart, Lung, and Blood Institute, 2004). In particular, hypertension is associated with an increased risk for developing vascular dementia (Sharp *et al.*, 2011). Milder forms of cognitive impairment are also apparent among patients with hypertension, who exhibit decreased performance on tasks measuring attention, reaction time, verbal fluency, and executive function when compared with persons with normal blood pressures (see Waldstein *et al.*, 1991).

Measuring and Diagnosing Hypertension

There are two primary methods for measuring blood pressure: the auscultatory and the oscillometric methods. The auscultatory method involves listening for the first Korotkoff sound (SBP) and the final Korotkoff sound (DBP) detected when blood flow resumes in a limb in which blood flow was temporarily occluded with a blood pressure cuff. In contrast, the oscillometric method utilizes pulse-waveform signals to measure blood pressure instead of Korotkoff sound detection. The standard protocol for measuring blood pressure is to avoid consuming caffeine, exercising, or smoking for 30 min before measuring blood pressure. In addition, blood pressure should be measured when the individual is in a seated position for 5 min with his or her arm supported at heart level (National Heart, Lung, and Blood Institute, 2004). It is recommended that clinicians obtain a minimum of two measurements during each blood pressure determination and then average the two measurements to measure blood pressure accurately.

Ambulatory blood pressure monitoring (ABPM) can provide measures of blood pressure across entire days or longer periods of time and is indicated in several clinical situations. Blood pressure waxes and wanes according to a circadian rhythm with lower blood pressure values occurring during rest and sleep and higher blood pressure values during wakeful activity. ABPM values are typically lower than blood pressure readings obtained in clinic settings and are more highly correlated with target organ damage (National Heart, Lung, and Blood Institute, 2004; O'Brien *et al.*, 2003). ABPM is indicated in situations in which clinic measures are not suspected to reflect the patient's actual daily blood pressures. White coat hypertension is one example of this and refers to situations in which individuals have hypertensive blood pressure readings during clinic measurements, but are in normal limits otherwise (Pickering, 1998). Although white coat hypertension is common, occurring in approximately 20–35% of patients, the risk of cardiovascular events among individuals with white coat hypertension is not significantly different from individuals with normal blood pressures (Fagard and Cornelissen, 2007). Masked hypertension, conversely, occurs when individuals have hypertensive blood pressure measures throughout daily life accompanied by normal blood pressure measures in clinic settings. In contrast to white coat hypertension, target organ damage and risk for cardiovascular disease among masked hypertensives is equivalent to patients with diagnosed hypertension (Fagard and Cornelissen, 2007). ABPM also enables examination of the magnitude of nocturnal blood pressure dipping. Typically during the night, blood pressure values decrease (i.e., dip) between 10% and 20%. Research indicates that risk of target organ damage, including impaired left atrial and ventricular functioning, is elevated among nondippers (Tadic *et al.*, 2013).

Demographic and Developmental Risk Factors for Hypertension

Risk of developing hypertension is strongly influenced by family history and age. A positive family history of hypertension is a risk factor for the development of hypertension, and it is well known that risk for hypertension increases as we age (Kannel, 1996; National Heart, Lung, and Blood Institute, 2004). Other factors influencing risk of hypertension include sex and race. Overall, the prevalence of hypertension in the United States is relatively similar for men and women. However, the prevalence of hypertension is higher in men than women before 45 years of age. By age 65, this trend is reversed and women have a higher prevalence of hypertension than men (Go *et al.*, 2013). As the rates of hypertension are lower among premenopausal women as compared to postmenopausal women, it is hypothesized that estrogen protects women from developing hypertension (Orshal and Khalil, 2004). Racial and ethnic differences in hypertension are also evident. Compared to non-Hispanic Whites or Caucasians, African Americans have a higher prevalence of hypertension. In addition, hypertension in African Americans

is more severe and has an earlier age of onset (Cooper and Rotimi, 1997).

Psychological and Behavioral Factors and Hypertension

In addition to lifestyle behaviors known to be associated with increased risk for hypertension (e.g., consuming a high salt diet; excessive alcohol consumption; being overweight; and low engagement in physical activity), psychological factors have long been suspected of influencing blood pressure. Foremost among these factors is evidence that chronic exposure to stress is linked with an increased risk of developing hypertension (Rutledge and Hogan, 2002; Sparrenberger *et al.*, 2009). Evidence that stress is associated with elevated blood pressures comes from numerous sources including the literature on major life events, occupational stress, and stressful living circumstances, including low socioeconomic status (SES), marital stress, and exposure to discrimination (Spruill, 2010). Research has shown that blood pressures increase, for example, following exposure to natural and manmade disasters (e.g., Baum, 1990). Studies examining occupational stress have focused on job strain, defined as the combination of high job demands and low control over work-related decisions (Karasek *et al.*, 1981). Several longitudinal studies have demonstrated a positive relation between high job strain and the development of hypertension (e.g., Guimont *et al.*, 2006; Ohlin *et al.*, 2007). In addition, as job strain increases among workers, incidence of hypertension increases (Markovitz *et al.*, 2004). Although low SES is associated with increased risk for hypertension, it is uncertain how associated circumstances (e.g., poor walking environment, limited availability of health foods, and safety issues) may explain the relation between low SES and risk of hypertension (Mujahid *et al.*, 2008). It should be noted that evidence linking exposure to chronic stress to hypertension in humans is substantiated by decades of research using various animal models (Dornas and Silva, 2011). Given the amount of research that has linked exposure to chronic stress to the development of hypertension, it is important to consider various pathways through which stress exerts its pathogenic effect on blood pressure regulation. Support for five pathways focusing on how humans respond to stress has been uncovered (see Jorgensen *et al.*, 1996). Each is considered in the sections that follow.

Negative Affect Pathway

Exposure to stress could lead to an increased incidence of hypertension because it is common to respond to stress by experiencing negative affect. Indeed, the primary components of negative affect – depression, anxiety, and anger/hostility – comprise what has been referred to as the 'hypertensive personality' based upon evidence that their frequent experience is associated with an increased risk of hypertension (Friedman *et al.*, 2001; Rutledge and Hogan, 2002). Although the evidence is mixed, multiple studies have shown that depressed affect is associated with an increased risk of hypertension (e.g., Davidson *et al.*, 2000; Nabi *et al.*, 2011). Other studies have found that a general pattern of negative affect, such as the combination of depression and anxiety, was predictive of future hypertension (e.g., Jonas and Lando, 2000). Similarly, comorbid diagnoses of major depressive disorder and generalized anxiety disorder have been reported to be associated with a greater risk of hypertension compared to either diagnosis alone (Carroll *et al.*, 2010). There is also evidence linking the experience of anger/hostility to elevated blood pressure (Schum *et al.*, 2003), but the consistency of these findings is not as strong as it is for anxiety and depression.

Suppressed Hostility Pathway

In contrast to the negative affect pathway that focuses on the experience of uncomfortable negative emotions, the suppressed hostility pathway hypothesizes that the association between stress and hypertension occurs because of individual differences in the expression of anger. Stemming from psychoanalytic theories of the early 1900s, it was hypothesized that hypertensive patients suppress negative emotions (primarily anger) and their blood pressures increased as a consequence. Early studies supported this hypothesis. For example, several studies found that hypertensive patients were less skilled in their behavioral responses to engaging with others in conflict-laden situations (e.g., Harris *et al.*, 1953; Kalis *et al.*, 1961; Keane *et al.*, 1982). More recent work (Morrison *et al.*, 1985; Larkin and Zayfert, 2004) extended these findings to show that the specific behavior deficits in assertive behaviors detected among hypertensive patients fell into two distinctive types: a nonassertive type consistent with the suppressed hostility hypothesis, and an overly aggressive type, who clearly did not suppress emotion. In support of these discoveries, Everson *et al.* (1998) found an increased incidence of hypertension among men who scored both high and low on a measure of anger expression. Based upon the premise that both submissive and aggressive forms of anger expression were associated with increased risk for hypertension, Davidson *et al.* (2000) introduced the construct, constructive expression of anger, and found it to be associated with lower blood pressures, even after controlling for a variety of standard hypertension risk factors. In sum, it appears that a curvilinear relation exists between anger expression and hypertension, with elevated blood pressures occurring both among those who suppress anger and those who express it intensely.

Emotional Defensiveness Pathway

The third pathway through which stress might increase risk for hypertension pertains to emotional defensiveness or the tendency to deny or minimize emotionally laden experiences. Potentially, due to discomfort associated with the experience of emotion, pre-hypertensive individuals become more attentive to their environments so that they can detect and avoid emotionally laden experiences; consequently, their blood pressures increase due to this heightened state of vigilance. Although multiple related constructs comprising this literature have been examined (e.g., repression, denial, blunting, and alexithymia), they share a common heritage in their function to minimize the experience of emotion. Although findings are

mixed, there is some evidence to suggest that emotional defensiveness is associated with hypertension; in fact, there is a prospective study that reveals that emotionally defensive persons are more likely to develop hypertension than persons who are more open to emotional experiences (Rutledge *et al.*, 2000). In a related area of inquiry, there are a few studies that have documented that hypertensive patients report less pain when exposed to standardized pain stimuli than patients with normal blood pressures (e.g., Bruehl *et al.*, 1992), providing further support for the finding that hypertension is associated with a tendency to minimize the self-report of pain or stimuli that induce negative affect.

Cognitive Deficit Pathway

As noted previously, hypertension is associated with a range of subtle performance deficits on neuropsychological and/or cognitive tasks. Rather than being a consequence of hypertension, it is possible that these deficits were present before the onset of hypertension, and perhaps functioned to increase risk for hypertension onset. For example, interacting with daily life stressors might be more challenging for persons exhibiting deficits in information processing, reaction time, and/or memory functioning. Studies examining this hypothesis have detected differences in performance on these sorts of cognitive tasks in fairly young adult samples (Elias *et al.*, 1990; Waldstein *et al.*, 1996) as well as in offspring of hypertensive parents (Pierce and Elias, 1993; Waldstein *et al.*, 1994). This suggests that these information processing deficits may be involved in the etiology of hypertension and risk for developing hypertension may be increased among those who struggle with tasks involving specific types of information processing.

Cardiovascular Reactivity to Stress Pathway

A final hypothesis pertaining to how stress leads to hypertension focuses on the magnitude and pattern of the autonomic response to stress, specifically the cardiovascular stress response. According to the reactivity hypothesis, prolonged or heightened cardiovascular responses to stress contributes to the development of hypertension as well as cardiovascular disease (Manuck, 1994). Exaggerated cardiovascular responses typically involve increased heart rate and blood pressure, which alters the structure and function of the heart and vasculature over time, leading to the onset of conditions like hypertension. According to the reactivity hypothesis, cardiovascular reactivity to stressors observed in a laboratory or clinic mimics cardiovascular reactivity to stressors occurring in naturalistic settings (Turner *et al.*, 1990). In general, research supports this aspect of the reactivity hypothesis, as individuals who are categorized as high reactors to laboratory tasks have been shown to demonstrate heightened blood pressures in naturalistic settings. There is also considerable evidence that the magnitude of cardiovascular reactions detected in laboratory settings predicts onset of hypertension later in life, as long as the follow-up period extended into middle adulthood when hypertension typically is detected (Carroll *et al.*, 2011; Treiber *et al.*, 2003); in sum, young adults who exhibit substantial cardiovascular reactions to mental or social stress are more likely to develop hypertension than low-reactive young adults. It is also possible that hypertension is associated with delayed recovery of the cardiovascular system following exposure to stress. Rutledge *et al.* (2000), for example, demonstrated that cardiovascular recovery predicted blood pressure levels during an ambulatory recording period better than cardiovascular reactivity.

Given the knowledge that hypertension is partly heritable, considerable research has been conducted contrasting cardiovascular reactions to stress between young adults with and without hypertensive parents. In a review of this body of literature, Fredrikson and Matthews (1990) concluded that offspring of hypertensive patients exhibited significantly greater cardiovascular reactions to a range of mental stressors than offspring of parents with normal blood pressures. Because exaggerated cardiovascular reactions are observed among these high-risk persons early in life, it appears that reactivity is more likely involved in the etiology of hypertension than being a consequence of it.

Treatment of Hypertension

Although hypertension has many dangerous medical consequences, it is easily treated, and a multitude of interventions have demonstrated efficacy in lowering blood pressures. Treatment of hypertension typically follows a step-wise approach with lifestyle modifications recommended as the first step and pharmacological treatment initiated after lifestyle modifications have failed to reduce blood pressure for most patients (James *et al.*, 2014; National Heart, Lung, and Blood Institute, 2004). For patients with stage 2 hypertension, pharmacological treatment is often initiated immediately due to the urgent need to reduce blood pressure rapidly.

Lifestyle Modification

The goal of lifestyle modification is to promote an overall healthy lifestyle, which thereby promotes a reduction in blood pressure. First, an ideal goal for both treating and preventing hypertension is the maintenance of a normal body weight. Reducing body weight by as little as 10 lbs is known to reduce blood pressure (He *et al.*, 2000; The Trials of Hypertension Prevention Research Collaborative Group, 1997), with the greatest reductions occurring in SBP (5–20 mm Hg). Second, consuming a diet that is rich in fruits, vegetables, low-fat dairy products, potassium, and calcium but low in dietary cholesterol and saturated and total fat is recommended for patients with hypertension. This diet, known as the Dietary Approaches to Stop Hypertension diet, has also been shown to result in a SBP reduction of 8–15 mm Hg (Vollmer *et al.*, 2001). Additional lifestyle recommendations include reducing dietary sodium intake, limiting alcohol intake, and increasing aerobic physical activity (Kelley and Kelley, 2000; Whelton *et al.*, 2002; Xin *et al.*, 2001). Although lifestyle modifications have been shown to lower blood pressures, many hypertensive patients do not adhere to them.

Psychological Intervention

Given the relation between exposure to stress and onset of hypertension noted above, it is not surprising that several

investigators have examined whether stress management strategies can be used to treat or prevent development of hypertension. The primary goal of these psychological interventions is to reduce blood pressure by reducing the magnitude of stress responses and physiological arousal (Linden and Moseley, 2006). There are two main approaches comprising this body of literature. The first approach involves learning relaxation techniques, meditation, or the regulation of physiology through biofeedback to reduce physiological arousal and promote autonomic balance. The second approach involves teaching effective coping skills and learning how to appraise life events in less stressful ways. The evidence suggests that greater reductions in blood pressure occur following treatment consisting of coping skills and cognitive reappraisal than with treatment comprised of relaxation techniques. These behavioral treatments generally result in significant but small reductions in blood pressure (Abbott *et al.*, 2014; Dickinson *et al.*, 2008; Linden and Chambers, 1994), but the quality of empirical work in this area is limited and it is not clear whether these psychological interventions result in reduced morbidity and mortality from cardiovascular disease comparable to pharmacological treatments (Linden and Moseley, 2006).

Pharmacological Treatment

Pharmacologic treatment is initiated after a failure to achieve blood pressure reduction using lifestyle modification alone or concurrent to lifestyle modification efforts. Thiazide-type diuretics are often recommended and are sufficient for controlling blood pressure for most patients as well as preventing cardiovascular diseases associated with hypertension (National Heart, Lung, and Blood Institute, 2004). Depending upon patient characteristics, other antihypertensive medications have been shown to work equally well, including angiotensin-converting-enzyme inhibitors, angiotensin-receptor blockers, or calcium channel blockers (James *et al.*, 2014). Like lifestyle modifications, if patients take the recommended antihypertensive medications as prescribed, positive outcomes are commonly observed.

Summary and Conclusions

Hypertension is a chronic medical condition that leads to severe damage to numerous organs in the body (e.g., heart, kidney, and retina) and eventually results in cardiovascular disease. Further, hypertension increases the risk of developing cognitive impairment, including an increased risk for vascular dementia. With good reason, understanding the causes of hypertension is an important area of scientific inquiry that will assist in the development of effective interventions by healthcare providers. Untreated hypertension is a serious global health problem.

Most people are familiar with having their blood pressure checked during visits to their healthcare providers. By adopting the recommended assessment protocol using an occluding cuff and stethoscope, healthcare providers can measure arterial pressures reliably and easily. However, in certain circumstances, blood pressure measures obtained in clinics bear little resemblance to those that occur during daily life; in these situations, ambulatory blood pressure measurements have proven to be quite valuable. Although few medical reasons for discrepant blood pressure values measured in clinic and life settings exist, there are psychological reasons why such differences occur. Foremost among these is the well-established association between exposure to stress during daily life and elevated blood pressure. Evidence from both animal and human studies has revealed that exposure to a range of chronic stressful environments is associated with hypertension. The mechanisms responsible for this association, however, are not clear. Nevertheless, several pathways have been proposed through which stress may lead to hypertension, including the propensity for hypertensive patients to differ from persons with normal blood pressures regarding: (1) the experience of negative affect (anxiety, anger, or depression); (2) the hostile expression or suppression of anger; (3) the denial or defensiveness associated with the experience of emotion or pain; (4) specific cognitive or intellectual capabilities; and (5) the magnitude and patterning of cardiovascular responses to and recovery from acute environmental stressors. As noted in this article, there is some evidence to support the existence of each of these pathways. However, it is also clear that no single pathway is responsible for explaining how exposure to stress leads to onset of all cases of hypertension. Rather, it may prove beneficial to consider each pathway as a potential route through which psychological factors are involved in the onset of hypertension, and to develop assessments of each of these pathways that could be regarded as behavioral or psychological risk factors. With additional empirical work, this knowledge could guide the development and testing of targeted behavioral programs to treat hypertension or prevent its occurrence.

Current evidence-based treatments for hypertension include lifestyle modification and hypertensive medication. A range of psychological interventions have also been examined, but in general, result in smaller blood pressure reductions than can be obtained by losing weight, consuming less salt and alcohol, and taking an antihypertensive medication. Although psychological interventions may be less effective than other interventions for lowering blood pressure, it is also possible that we have yet to uncover strategies for optimally matching specific psychological interventions with the types of patients who will respond to them. In studies on hypertensive patients using progressive muscle relaxation (e.g., Larkin *et al.*, 1990), for example, although mean blood pressure reductions were modest, several patients exhibited substantial reductions in blood pressure ($>$ 20 mm Hg). It will be up to future research examining psychological factors involved in the etiology of hypertension to determine whether current treatments for hypertensive patients can be matched to the underlying mechanisms through which exposure to stress results in hypertension in solving this global health problem.

See also: Aggression. Anger, Hostility, and Anger Management. Anxiety, Panic, and Phobias. Behavioral Medicine. Biofeedback and Neurofeedback. Coping. Depression. Ethnic Minority Mental Health Strengths in the United States. Exercise, Physical Activity, and

Mental Health. Food, Nutrition, and Mental Health. Gender Differences and Similarities in Mental Health. Medical Regimen Adherence. Poverty and Mental Health. Psychophysiology of Mental Health. Racism and Mental Health. Responses to Natural Disasters. Socioeconomic Status. Stress. Workplace Health

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