Effect of Biofeedback-Assisted Relaxation on Blood Pressure and Cortisol Levels in Normotensives and Hypertensives

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This report examines the relationship between blood pressure and cortisol in normotensives and hypertensives. Both groups received biofeedback-assisted relaxation training. Both groups significantly decreased their muscle tension levels from baseline. Only the hypertensives decreased their blood pressure and cortisol levels after training. Implications for the role of cortisol in the relaxation response and in hypertension are discussed.

KEY WORDS: hypertension; biofeedback; cortisol; relaxation.

INTRODUCTION

The role of cortisol, a glucocorticoid, has been clearly defined in the context of the stress response (Selye, 1974). Cortisol is released from the adrenal cortex in concert with activation of the sympathetic division of the autonomic nervous system and with release of catecholamines from the adrenal medulla. In the classic stress response, an increase in systematic blood pressure is observed, along with other effects of sympathetic stimulation. Under nonstress conditions, cortisol is released according to a diurnal rhythm, with a peak of approximately 13 μg% at early morning and a nadir of about 4 μg% in the late afternoon. This episodic secretion is manifested in the

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variability observed in both the plasma cortisol and the urinary cortisol values in individuals. The functions of cortisol include widespread metabolic effects and maintenance of vascular reactivity (Guyton, 1981), with glucocorticoid receptors found in virtually every nucleated cell in the body (Munck et al., 1984).

Cortisol's role in hypertension is complex and enigmatic. Scoggins et al. (1982) studied ACTH-dependent hypertension in the sheep and suggested as possible mechanisms the redistribution of body fluid volume, sodium retention, and increased vascular responsiveness, mediated by glucocorticoids. Hausler et al. (1983), in studies with spontaneously hypertensive rats (SHR), suggested that an increased sensitivity of hypothalamic-pituitary-adrenocortical function may be linked to the development of hypertension but may not be associated with the maintenance of high blood pressure. Abnormal metabolism of cortisol was found to be a causative factor in the hypertension of some children (Ulrick et al., 1979). However, in dogs, high plasma levels of cortisol were correlated with hypotensive effects (Lohmeier and Kastner, 1982).

In order to begin identification of the mechanism of action of relaxation-based treatment, several investigators have measured physiological and/or biochemical parameters before and after relaxation treatment in normotensives and in hypertensives. Jevning et al. (1978) found significant decreases in plasma cortisol after meditation in those normotensives who had been practicing meditation regularly over a 3- to 5-year period. McGrady et al. (1981) investigated the effects of EMG (electromyograph) biofeedback-assisted relaxation on blood pressure, aldosterone, cortisol, and renin in 38 essential hypertensives who participated in a pretest, posttest, control-group experiment. Significant decreases were observed in blood pressure, forehead muscle tension, plasma aldosterone, and urinary cortisol in the experimental group. These findings suggested that the adrenal cortex may have had a significant role in the relaxation response of these hypertensives; however, the correlation between blood pressure and cortisol was not tested.

This paper reports on a pilot study to explore the relationship between the mean arterial blood pressure (MAP), the average pressure driving blood through tissues, and cortisol over the normotensive and hypertensive range of blood pressure. Clinic and home blood pressures, forehead muscle tension levels, and plasma and urinary cortisol levels were determined in normotensives and hypertensives before and after biofeedback assisted relaxation training. Our hypothesis was that both normotensives and hypertensives would decrease muscle tension levels, an index of relaxation; however, relaxation would not be associated with equal changes in blood pressure and in cortisol levels in normotensives and hypertensives.