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Abstract

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VERTICAL FIXATION DISPARITY AND ESSENTIAL HYPERTENSION

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INTRODUCTION

In 1982, Sargent and Walls provided evidence suggesting a physiological association between the visual system and the cardiovascular system.(1) In a report based on clinical findings, they presented ten patients with essential hypertension who showed significant reductions in blood pressure upon neutralization of a vertical fixation disparity with vertical prism.

Sargent and Walls hypothesized that a vertical imbalance between the eyes causes stimulation within the lateral hypothalamus. The nerves which innervate the extraocular muscles (third, fourth, and sixth cranial nerves) have been shown to communicate with the lateral region of the hypothalamus via the reticular formation. The lateral and posterior regions of the hypothalamus are the primary subcortical centers for control of the sympathetic nervous system.(1) Stimulation of these areas cause specific physiological responses including, pupil dilation, piloerection, increases in respiration, acceleration of the heart rate, and elevation of blood pressure.(14)

In approximately 90% of the hypertensive cases, the actual cause of the hypertension is idiopathic. This condition is called essential hypertension. Often essential hypertension responds poorly to antihypertensive treatment. In the

remaining 10% of the hypertensive cases, the condition is secondary to other diseases and is usually responsive to medical treatment.(2)

Homeostasis of blood pressure is maintained by no less than eight mecahinisms.(13) Guyton identifies three rapidly acting acting control mechanisms, 1) baroreceptor feedback, 2) chemoreceptor feedback, and 3) the CNS ischemic response mechanism. All three are controlled by the nervous system and respond to abnormal changes in blood pressure within one or two minutes.

Long term control of blood pressure is principally regulated by the renal-body fluid-pressure control mechanism. If the mean arterial pressure falls, the decreased pressure causes the kidneys to retain more body fluids and salt. As the blood volume increases, there is a greater venous return to the heart. This leads to an increased cardiac output which generates a higher mean arterial pressure. An increase in the cardiac output also causes an increase in the total peripheral resistance.

A possible factor contributing to essential hypertension that has not been adequately researched is the impact of visual stress on blood pressure. In recent years, an increasing amount of research evidence has accumulated which implicates the involvement of the autonomic nervous system in the

regulation of blood pressure and hypertension. The effect of stress on the sympathetic nervous system has been well documented in the literature. Shapiro and Goldstein cite several sources of stress that are definitely associated with hypertension including, personality characteristics, culture and urbanization, prolonged illness, natural disasters, and occupational stress.(3) While the short term effects of stress appear to be reversible, the long term effects of stress on the cardiovascular system may significantly alter the system. Fixation disparity may be considered a measure of stress within the visual system that can be translated to the entire organism.

Fixation disparity has been described by Ogle as a very small deviation (measured in arc minutes) between the two eyes while under binocular conditions.(5) This deviation results in the transmission of disparate retinal images to the visual cortex. As long as the disparate cortical images remain within Panum's fusional area, single binocular vision will be maintained. If the disparity exceeds Panum's area diplopia will result. The amount of prism necessary to neutralize the fixation disparity is called the associated phoria.

Carter, in his discussion of fixation disparity parameters concurs with Ogle that the amount of disparity is a function of the strength of the muscle innervations during fusion, the amount of heterophoria, and the level of stimuli in the field

of view.(6) He also states that, "With few exceptions, significant values of fixation disparity occur only when there is a stress on the motor and/or sensory mechanisms of binocularity."

One method of causing visual stress is to induce forced vertical vergence with varying amounts of prism. Rutstein and Eskridge have shown that vertical fixation disparity is linearly related to the amount of forced vergence.(9) However, the slope of the linear relationship varies widely from subject to subject. Thus, in one subject one prism diopter of forced vergence may cause little or no change in the vertical fixation disparity, while in another subject the disparity may radically increase.

The purpose of our study, based on the work of Sargent and Walls, was to determine if there is a significant correlation between vertical fixation disparity and essential hypertension. If there is a causitive or correlational relationship between the two, one would expect to find a significant difference in the amount of vertical disparity in the hypertensive population when compared to the normotensive population.

METHOD

Ninteen essential hypertensive patients were selected at random for this study. The criteria for patient selection were that the patient: (1) be 45 years of age or less, (2) be binocular at 40 cm, (3) have at least 6/6 (20/20) correctable acuity in each eye at 40 cm, (4) be diagnosed as hypertensive by a physician and prescribed a therapy regime including medication. Each hypertensive patient was matched to a normotensive patient according to seven characteristics considered to be major risk factors in hypertension; sex, age, weight, race, use of tobacco, use of oral contraceptives, and family history of hypertension.(7) The weight of each patient was subjectively judged by the examiner to fall into one of four categories; underweight, normal, overweight, or obese. Ages of subjects were matched within +/- 2.5 years of each other.

The clinical measurements of vertical fixation disparity were performed at a distance of 40 cm using the disparometer described by Sheedy.(8) All measurements were taken with the patient's habitual near refractive correction in a trial frame. Standard room illumination was used and the illumination of the target remained constant throughout all measurements. Measurements of fixation disparity were obtained using a bracketing technique. The disparometer was

first set to a position where the right line of the vernier target appeared higher than the left line. The vertical separation between the vernier alignments was then reduced and the patient asked to respond when the left vernier line became just noticably higher than the right line. After recording this reading, the vertical distance between the vernier lines was again increased until the left line appeared much higher than the right line. Again, the vertical separation between the lines was reduced and the subject was required to respond when the right vernier line became just noticably higher than the left line. The value of the vertical disparity for a given trial was taken as the midpoint between the two measurements.

Vertical disparity measurements were taken first through the patient's habitual near prescription. Two prism diopters base-down (or less depending upon the patients ability to maintain fusion) was then placed before the right eye and the vertical disparity measurement repeated under the forced vertical vergence demand. The prism was then inverted before the same eye in a base-up orientation and the measurement repeated. Three trials were recorded at each vertical vergence demand and the readings averaged to yield a final value.

RESULTS

For each patient, the mean values for vertical disparity measurements in arc minutes, and the corresponding prism used to produced each vergence demand, were used to calculate a simple linear regression line. The average vertical disparity for a subject was taken as the y-intercept of the regression line, which corresponds to a forced vertical vergence demand of zero prism diopters. The frequency distribution of vertical fixation disparity among the hypertensive and normotensive sample populations is displayed in Figure 1.

INSERT FIG 1 ABOUT HERE

The mean values of vertical fixation disparity were determined for both populations and a repeated measures t-test was used to compare the means. The mean vertical fixation disparity for the hypertensive group was x= 1.3, SD 1.1 arc minutes, with a range of 0.0 to 4.2 arc minutes. The

normotensive group had a mean vertical fixation disparity of x=0.6, SD 0.4 arc minutes, ranging from 0.1 to 1.7 arc minutes. A statistically significant difference between the two groups was obtained ($T18=2.78$, $p<0.012$). Fifty-eight percent of the hypertensive population showed a vertical fixation disparity greater than 1.0 arc minute, while only sixteen percent of the matched normotensive group manifested a value greater than 1.0 arc minute.

DISCUSSION

The present study indicates a strong correlational relationship exists between vertical fixation disparity and essential hypertension. Sargent and Walls reported patients using vertical neutralizing prism as part of their lens therapy, experienced reductions in both systolic and diastolic blood pressures (1).Clearly, further research is indicated before a causal relationship can be established.

Roy reported relieving cases of ocular migraine by prescribing the appropriate amount of horizontal and vertical neutralizing prism found following prolonged monocular occlusion. Patients responding best to vertical prism prescription were those who manifest hyperphoria when only one eye was occluded(10).

Torticollis often results when a patient attempts to

overcome a latent vertical phoria. This habitual tilting of the head creates tension in the extraocular muscles in each orbit, severe cervical tension, and cephalalgia from supporting the head in an imbalanced posture(10). Head tilt may also produce enough tension in the neck to alter short term blood pressure homeostasis via the baroreceptors located at the bifurcation of the carotid arteries.

In order for vertical imbalance of the oculomotor muscles to affect long term blood pressure control, stimulation of the pressor system in the hypothalamus and/or an increase in ACTH releasing factor must occur(11). One neural reflex between the extraocular muscles and the heart is known to exist. The oculocardiac reflex (OCR) is a physiological response of the body to physical stimulation of the eye and/or adnexa. Typical signs and symptoms of the reflex include bradycardia, faintness, nausea, and alteration in blood pressure. The major pathway for the OCR is believed to consist of an afferent link through the ophthalmic division of the trigeminal nerve via the reticular formation to the nuclei of the vagus nerve. Vagal simulation produces the decrease in heart rate initiated by the reflex(4). However, no neural pathway has been found that would link vertical muscle imbalance to a sustained increase in blood pressure.

Since hypertension is a polygenically inherited trait(7,12), the possibility exists that vertical muscle imbalance, producing fixation disparity, is also inherited.

If is this the case, vertical muscle imbalance is a correlational and not a causitive factor in essential hypertension. Whatever the nature of the relationship, it is of intrest to the practitioner to be aware of the correlation between vertical muscle imbalance and essential hypertension. A double blind study involving the neutralization of vertical fixation disparity in a sample of essential hypertensives is indicated in order to distinguish a causal from correlational relationship.

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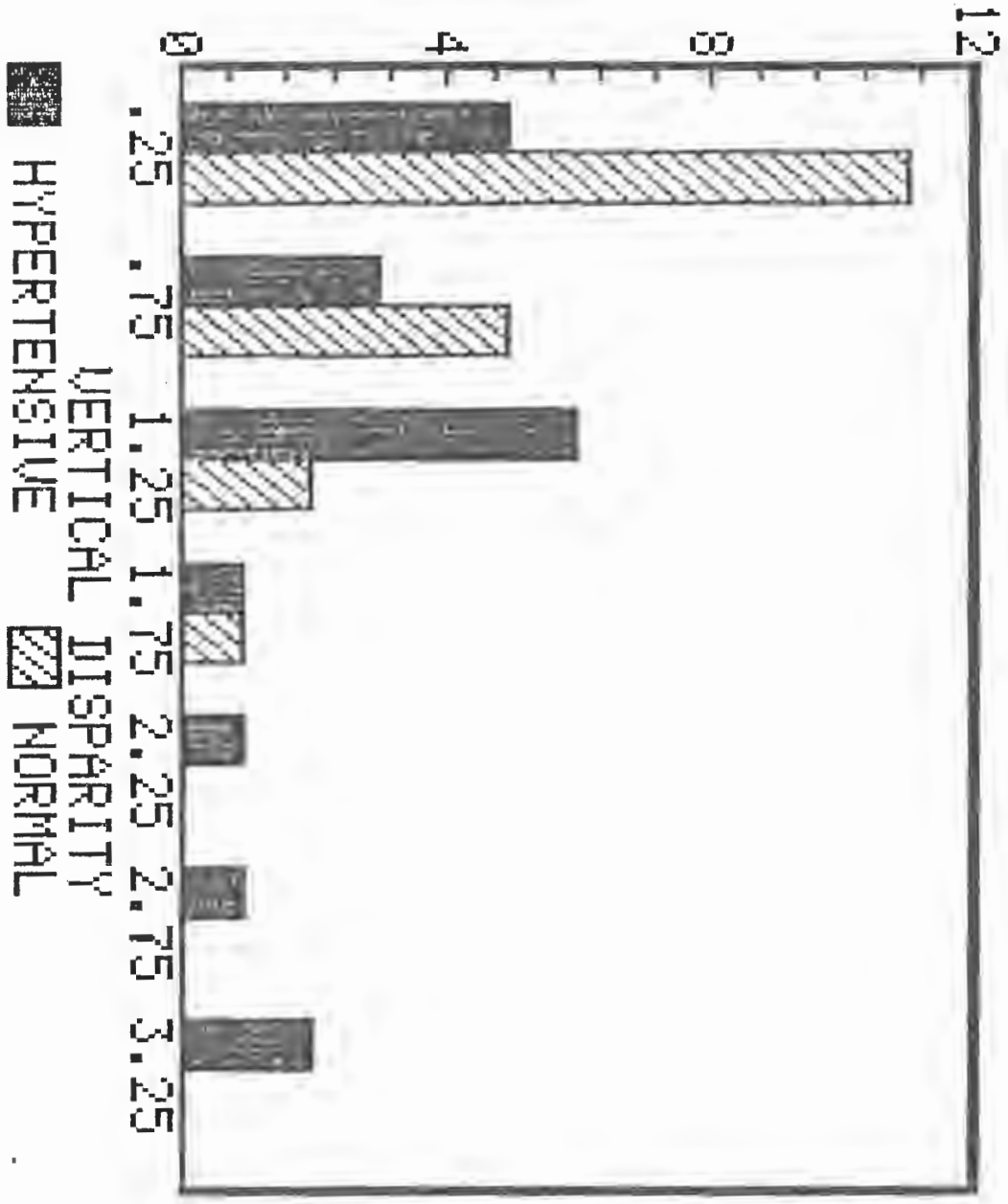
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VERTICAL DISPARITY
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NORMAL