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Autonomic Imbalance May Underlie Labile Hypertension: II. Possible Physiology

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ABSTRACT

Parasympathetic and sympathetic (P&S) assessment (ANX 3.0, Autonomic Monitoring, ANSAR, Philadelphia, PA) enables independent, simultaneous measures of P&S activity. Parasympathetic excess (PE) is an abnormal increase in P activity in response to S challenge (*e.g.*, Valsalva and stand). In a companion abstract, a study was documented comparing 388 hypertension patients (64.0 years; 244 Female). An experimental group of 328 patients (64.0 years; 208 Female), included 94 with labile hypertension (LH), the rest with frank hypertension. The remaining 60 patients (64.1 years; 36 Female) comprised the control group, including 30 LH patients. LH patients demonstrated PE with hypertension. Eighteen months additional PE therapy (low dose Carvedilol or Amitriptyline) relieved all fluctuations in BP and the PE in the experimental group and none in the control group. Therapy reduced the average experimental, LH patient's BP from 141/107 to 118/71 and from 145/109 to 144/107 in the control group. The average frank hypertension patient's BP decreased from 141/107 to 121/73 and from 146/109 to 125/89, for experimental and control, respectively. Carvedilol includes two compounds: a non-selective-beta-adrenergic-antagonist and an alpha-1-adrenergic-antagonist. Carvedilol's alpha-component is reported to change activity sites with autonomic neuropathy, indirectly reducing PE. Reducing PE is Amitriptyline 's function. PE is reported to be the primary autonomic abnormality; accompanying sympathetic excess (SE), secondary. As reported elsewhere, treating the SE as the primary imbalance exacerbates concurrent PE and SE, leading to poor outcomes. Treating the PE primarily relieves the condition, leading to improved outcomes. SE is well documented to underlie hypertension and is demonstrated in both frank and labile hypertensives. PE is demonstrated only in LH patients. Therefore, PE seems to be the cause of instability and BP fluctuation, perhaps by forcing a greater SE during a sympathetic challenge (stress or exertion), without affecting resting sympathetic levels.