patients; HP/SBP ratio was high in waking time and low at night. HP/SBP ratios calculated from pressor and depressor phase were almost identical ($r=0.967$). Thus HP/SBP ratio is considered to be a new valuable index of BS which can be measured without any pharmacological or barometric interventions.

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GENETIC FACTOR OF HYPERTENSION, BLOOD PRESSURE RESPONSE TO EXERCISE AND ERYTHROCYTE CATION TRANSPORT IN JAPANESE NORMOTENSIVE INDIVIDUALS

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It is common knowledge that the genetic factor is very important in the pathogenesis of essential hypertension. The study of the cell membrane monovalent cation transport systems are going to throw new light on the elucidation of the genetic mechanism in essential hypertension.

Then we studied the blood pressure response to supine exercise testing by bicycle ergometer in 139 Japanese healthy sedentary men with and without the family history of essential hypertension ((+)FHH and (-)FHH).

And also several erythrocyte monovalent cation transport parameters were measured in 39 of these 139 individuals.

In the results, (1) the systolic blood pressure response to exercise testing was larger in (+)FHH than in (-)FHH. (2) the intraerythrocytic sodium contents and sodium potassium ratio were significantly higher in (+)FHH than in (-)FHH. And (3) several cell membrane monovalent cation transport systems (Li-Na countertransport, Na-K cotransport and Na-K pump activity) in human red cells were clearly accelerated in (+)FHH.

We concluded that in individuals with the family history of hypertension there were the abnormalities of the blood pressure response to exercise stress testing, intracellular sodium content and several cell membrane monovalent cation transport systems even before going up "hypertension" and these findings might be very available for detecting the genetic factor of hypertension in normotensive individuals.

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EFFECTS OF VASOPRESSIN AND THE SYMPATHETIC NERVOUS SYSTEM IN THE CARDIOVASCULAR RESPONSE TO CAROTID SINUS REFLEX

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We have previously reported that cardiopulmonary receptors with vagal afferents exert a tonic inhibitory influence on the carotid sinus reflex. In this study, we examine effects of vasopressin and the sympathetic nervous system in cardiovascular response to bilateral carotid occlusion (BCO) in the anesthetized dogs before and after vagal section. The aortic nerves and the cervical sympathetic nerves were cut in the neck. After intravenous administration of atropine and captopril, changes in mean arterial pressure (MAP),

hindlimb perfusion pressure (PP) perfused at a constant blood flow and heart rate (HR) in response to BCO were examined with and without administration of arginine-vasopressin antagonist, d(CH₂) Tyr (Me)AVP, (AVP-A), 10 µg/kg or ganglionic blockade (Hx), 12-15 mg/kg. BCO produced a significantly increase response in MAP, PP and HR after vagal section. With vagi intact, AVP-A did not alter the pressor response to BCO. Subsequent Hx eliminated both the tachycardia and the pressor response to BCO. When Hx alone was induced, the pressor response to BCO was reduced by 73%. AVP-A after Hx administration was capable of producing 27% of the control response to BCO. Plasma vasopressin concentrations increased by administration of Hx before and after vagal section.

The effects of blockade of either vasopressin or the sympathetic nervous system on the pressor response to carotid sinus reflex were significantly greater when the other system had previously been eliminated. In conclusion, carotid sinus and cardiopulmonary reflexes modulated by both the sympatho-adrenal system and vasopressin in circulatory control.

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SALT LOADING IMPAIRED CARDIOPULMONARY BAROREFLEX IN RATS

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Cardiopulmonary baroreflex contributes importantly to control of sympathetic nerve activity, and secretion of renin and ADH. The aim of this study was to examine whether salt loading alters cardiopulmonary baroreflex control of renal nerve activity in normotensive (WKY) as well as in hypertensive rat (SHR). WKY and SHR were fed either a normal (0.5% NaCl) or high (8% NaCl) salt diet for 4 weeks. High salt increased blood pressure significantly in SHR ($p < 0.01$), but not in WKY. The rats were anesthetized with α -chloralose, and sino-aortic denervation was performed. We recorded left ventricular end-diastolic pressure (LVEDP) and renal sympathetic nerve activity (RSNA) at rest and during volume loading (Dextran, 2.3 ml/min). The gain of cardiopulmonary baroreflex was assessed by obtaining stimulus (Δ LVEDP) - response (Δ RSNA) curves. The slopes of the curves were significantly attenuated by high salt intake in both SHR and WKY.

These results suggest that salt loading lowers the gain of the cardiopulmonary baroreflex in WKY as well as in SHR.

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BAROREFLEX FUNCTION IN PATIENTS WITH LONE ATRIAL FIBRILLATION

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Atrial fibrillation (AF) is associated with neurohumoral excitation. We postulated that this could result in part from impairment in the inhibitory influence of baroreflexes which could be restored by digitalis. In ten patients with lone AF and 10 normal control subjects, we studied the