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**SPIDYL-PEPTIDASE-IV ACTIVITY
DECREASE LIKELY INDICATIVE OF CARDIAC
INVOLVEMENT IN ARTERIAL HYPERTENSION**

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Since hypertensive heart disease has always to be expected in hypertension, on the other hand, conventional clinical methods are rather insensitive a search for biochemical markers may be desirable. Therefore, 52 pts (19 f, 13 m) in untreated hypertension have been investigated by biochemical methods. As DP-IV is mainly localized in endothelial cells activity increase of this enzyme may be indicative of organ involvement. Though averaged DP-IV ranged within reference it was pointing to markedly elevation in some pts, esp. in those with elevated PA(LV filling) pressure and/oripoprotein (cholesterol) blood level. Conclusion: There may be biochemical markers of cardiovascular involvement in hypertension, in addition to the well-known biophysical techniques, getting obvious even under rest conditions. This confirms our experience with other biochemical alterations in arterial hypertension.

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**IN VITRO EFFECTS OF CANNABINOIDS ON CARDIAC AND VASCULAR
Na⁺-K⁺ PUMP IN THE RABBIT.**

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Cannabinoide (C), the active estabillite of spirosteroide, was shown to behave *in vitro* as partial agonist at the digitalis receptor site and to reconstitute (10⁻⁶ M) the Na⁺-K⁺ pump blocked by Ouabain (O). We assessed the pump activity in demembrated elliptical strips of rabbit renal arteries, Na⁺ loaded for 15' by means of a K⁺-free saline, and in electrically stimulated rabbit left atria. In arteries 0.1(10⁻¹⁰ M) inhibited dose dependently the cumulative K⁺-induced relaxation (from 1 to 10 μM) after activation with norepinephrine. C, (1 or 3.10⁻⁶ M) did not affect 75% of K⁺ relaxation, but it moderately reconstituted (at 3.10⁻⁶ M) the pump completely blocked by high concentration of O. However in the same range of doses it blocked contractions induced by norepinephrine, Ca⁺⁺ and high K⁺. In the atria C, (1.10⁻⁶ M) blocked both the O, (1.10⁻⁶ M) and K⁺-free saline inotropic effects. These results suggest that the reverse *in vivo* by C, of some of the effects of digitalis and its hypotensive activity could be due, not only to its interaction with the Na⁺-K⁺ pump, but also to some other non specific membrane activities in vascular smooth muscle cells.

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**Na⁺ TRANSPORT IN INFANTS OF PREGNANCY-
INDUCED HYPERTENSIVE MOTHERS**

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We examined the cellular sodium content (Na_i) and the plasma membrane Na_o-K-ATPase activity in umbilical cord erythrocytes of infants of 35 normal women and 30 mothers affected by pregnancy-induced hypertension (PIH) in order to find if this form of transient hypertension could determine alterations in cation fluxes in the offspring. The Na_o-K-ATPase activity was determined according to Kitae; Na_i was measured by an-selective analyzer. We observed an increase in Na_i and a decrease in the Na_o-K-ATPase activity in umbilical cord blood of infants of PIH mothers compared with control subjects. The data obtained from this study raise the possibility that either a genetic defect is expressed in the erythrocyte membrane of infants from PIH mothers or a circulating inhibitor of the Na pump is present in PIH.

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**AGGREGATION OF BLOOD PRESSURE IN THE FAMILIES
OF CHILDREN WITH HIGH SYSTOLIC BLOOD PRESSURE**

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We have studied 889 children 5-18 years old, their parents and offsprings. The aggregation of systolic blood pressure and diastolic blood pressure is compared among relatives of two groups of index children: children with systolic blood pressure in top quintile and bottom. Both systolic and diastolic blood pressure aggregate more strongly parents and siblings in top quintile with children in same quintile than in relatives of children in the bottom quintile. Since blood pressure aggregate so strongly in families of children with labile high systolic blood pressure, study of these children and their families may yield important information about the etiology of hypertension.