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LEFT VENTRICULAR HYPERTROPHY, LEFT VENTRICULAR FUNCTION AND BETABLOCKERS IN PATIENTS WITH ESSENTIAL HYPERTENSION

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We have attempted to evaluate the effect on the hypertensive heart of betablockers/BB analyzing the change and interrelations of arterial blood perssure (ABP), left ventricular hypertrophy (LVH) and function (LVF). A total of 190 patients with essential hypertension (EH) were treated for 12 months with different classes of BB (monoterapy). By means of M-mode echocardiography and semi-automatic analysis of the recordings performed before and at the end of the 1st, 6th and 12th month of treatment the following parameters were assessed: left ventricular muscle mass (LVMM), the ratio systolic ABP/end-systolic volume of the LV (ABP_s/ESVIV), maximal rate of augmentation of LV diameter during the fast filling period ($+dD/dt_{max\ FFP}$) and maximal rate of augmenttation of LV diameter during atrial contraction ($+dD/dt_{max\ AC}$). The ABP_s/ESVLV in hypertensive patients without LVH is not different than in normals (4,36 ± 1,04 vs 4,31 ± 0,67, $p > 0,10$) whereas in patients with LVH it is lower (4,01 ± 0,72, $p < 0,045$). There is a positive, though moderate, but still significant correlation ($r = 0,474$, $p < 0.001$, $n = 74$) between LVMM and ABP_s/ESVLV in patients without LVH and those with LVMM within the limit of 4 SD above the mean normal value. For the whole group of patients with LVH, however, the correlation is negative and significant by all means ($r = -0,653$, $p < 0,001$). The $+dD/dt_{max\ FFP}$ in patients without LVH is lower than in normals (14,6 ± 3,3 $cm.s^{-1}$ vs 16,8 ± 1,90, $p < 0,001$) and in patients with LVH there is additional reduction (12,6 ± 2,0, $p < 0,001$). The changes in $+dD/dt_{max\ AC}$ are reciprocal (4,0 ± 0,5 $cm.s^{-1}$ in normals; 5,2 ± 1,0 in patients without LVH, $p < 0,0001$; 5,6 ± 0,9 in patients with LVH, $p < 0,045$). There exist a moderate negative correlation between LVMM and $+dD/dt_{max\ FFP}$ ($r = -0,480$, $p < 0,01$) as well as a moderate positive between LVMM and $+dD/dt_{max\ AC}$ ($r = 0,449$, $p < 0,001$).

There is a significant correlation between percentage reduction of ABP_s ($r = 0,603$, $p < 0,001$) and heart rate ($r = 0,636$, $p < 0,001$) and that of LVMM; for ABP_d the correlation is moderate ($r = 0,457$, $p < 0,001$). The reduction of LVMM is greater with BB without ISMA ($p = 0,02$ for the 6th month and $p < 0,001$ for the 12th month).

Table 1. ABP_s, ABP_d, LVMM and LVF during BB therapy in hypertensive patients with LVH

Indices	*Initial*	*1st month*	*6 th month*	*12 th month*
ABP_S	156,5 ± 14,2	141,5±13,7·	134,8±11,9⁺	132,6 ±11,8
ABP_d	106,4± 8,5	90,7± 8,6·	89,4± 7,9⁺	88,8 ± 7,3
LVMM	278,9±36,6	269,5±32,3	225,5±29,0⁺	214,5±29,9
$ABP_s/ESVLV$	4,1±0,98	3,7±0,81·	3,9±0,88	4,0±1,0
$+dD/dt_{maxFFP}$	12, 6±3,8	14,1±3,9·	15,2±2,7⁺	15,7±2.7*
$+dD/dt_{maxAC}$	5,5±0,7	5,0± 0,7·	4,5± 0,5⁺	4,4± 0,6

. compares "Initial-1st month" + compares "1st month-6th month"
* compares "6th month-12th month"

Cardioselectivity has no impact on LVH-regression. Our results show that in hypertensive heart disease the adaptative increase of muscle mass is combined with the transition towards the so-called "slow type muscle" which allows managing with the increased afterload but leads to specific unfavourable consequences. There is retarded relaxation and a contractile deficit that advances with LVMM increase. The process is slow and takes place generally between the 1st and 6th month of treatment. Regression of LVH is defined by multiple factors with the leading role of ABP-control and adrenergic cardiostimulation reduction. Relying to that, LVH-regression is more pronounced when BB without ISMA are used. The end-point effect of chronic BB therapy upon systolic and diastolic function in hypertensive heart is favourable. The reduced LV afterload and LVH-regression succeed not only to compensate for the direct negative effect of BBs on myocardial contractility and relaxation but also to actually bring forth an additional improvement of ventricular function. In that respect there is no principal difference for the various clases of BBs. The quantitative differences of the negative inotropic effect of BBs with and without ISMA can be still of practical importance in the initial treatment of patients with pronounced LVH in whom there is a concealed contractile deficit. Later, however, the greater degree of regression of LVH in BBs without ISMA leads to an equal end-effect of BBs with or without ISMA upon ventricular function. Cardioselectivity has no impact on hitherto described functional changes.