52) Cellular Architecture of Hypertrophied Myocardium of Spontaneously Hypertensive Rats and its Regression with Nifedipine Treatment: A Scanning Electron Microscope Study.

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Despite numerous reports on cardiac hypertrophy, only a few studies have dealt with the structure of the heart in which hypertrophy regressed after therapeutic intervention. The purpose of this report was to study the cellular architecture of myocardium by scanning electron microscope (SEM) in adult SHR in which left ventricular (LV) hypertrophy regressed after nifedipine treatment. Nifedipine (20mg/kg/day) was given orally to male 15-week-old SHR (n=7) for 20 weeks (SHR-N). The hearts from 15- and 35-week-old SHR (n=6, SHR-15 and n=7, SHR-35, respectively) and Wistar-Kyoto rats (WKY) (n=5, WKY-15 and n=6, WKY-35, respectively) served as control. SEM study was made on the cardiocytes in the subepicardium of the LV. In each heart from each rat group, length and width of at least 50 cardiocytes and their branches were measured, and the number of branches, step to step connections, step to end connections and side to side connections per cell were evaluated in SEM photographs. The ratio of cell length to width was calculated and cell volume was estimated on the assumption of a cylindrical configuration. In SHR-N, blood pressure was significantly suppressed during 20 weeks of drug administration. In both SHR-15 and SHR-35, LV weight (LVW) and LVW/body weight ratio (LVW/BW) were significantly larger than those of age matched WKY, signifying a definite LV hypertrophy prior to nifedipine administration. In SHR-N, LVW and LVW/BW were significantly smaller than those of SHR-35 and LVW/BW was significantly smaller than that of SHR-15, signifying a regression of LV hypertrophy after nifedipine treatment. In both SHR-15 and SHR-35, cell width, cell length and cell volume were significantly larger than those in their age matched WKY. In SHR-35, both cell width and cell volume were significantly larger than those of SHR-15. In both SHR-15 and SHR-35, cell length/width ratio was not significantly different from that in age matched WKY. In SHR, from 15 to 35 weeks of age, cellular hypertrophy continued to progress with a constant cell length to width ratio comparable to that in age matched WKY. While, SHR-N, cell width and cell volume were significantly smaller than those of SHR-35 and not significantly different from those of SHR-15. There was no significant difference of cell length among SHR-15, SHR-35, and SHR-N. These microgeometry of SHR-N may indicate a prevention of progression of hypertrophy and a regression of existing hypertrophied cardiocytes. The significant decrease of LVW/BW in SHR-N was attributed to a decrease of cell volume. There was no significant difference in the number of series branches per cell among 5 rat groups. In both SHR-15 and SHR-35, there was no significant difference in the number of lateral branches per cell; lateral branches were significantly more in number than in their age matched WKY. In SHR-N, lateral branches were not significantly different in number from those in both SHR-15 and SHR-35. In SHR-35, the number of step to end connections per cell was significantly larger than that of either WKY-35 or SHR-15. In SHR-N, the number of step to end connections were significantly larger than that in WKY-35 and significantly smaller than that in SHR-35 and not significantly different from SHR-15. In SHR-15, the number of step to step connections per cell was fewer than that in WKY-15. In both SHR-15 and SHR-35, the number of side to side connections per cell were significantly fewer than those in their age matched WKY. In SHR-35, side to side connections per cell were significantly fewer than those in SHR-15. In SHR-N, the side to side connections were fewer than those of either in WKY-35 or SHR-15. The cause and time of possible dehiscence of these cell connections in SHR remains unclear. It may well be that the mode of cardiac cell connections in SHR is genetically different from that in WKY rats.