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Ñ¹Á¦³¬¸ººÉĞĜÔĞÄ¼¡·Êºñ´óÊóĞÄ¼¡Ï¸°ûºË¸Æ×ªÔËµÄ¸Ä±ä

Áõ½¡[1]¡¢ºÎ×÷ÔÆ[1]¡¢ÍõÅàÓÂ[2]
1 µÚÈı¾üÒ½´óÑ§ĞÂÇÅÒ½ÔºĞÄÄÚ¿Æ
2 µÚÈı¾üÒ½´óÑ§²¡ÀíÉúÀí½ÌÑĞÊÒ

Í¨¹¸¹Ö÷¶¯ÂöËõÇ(abdominal aortic coarctation,AAC)ĞÄ¼¡·Êºñ´óÊóÄ£ĞÍÖÆ±¸¡¢²îËÙÀëĞÄÌá´¿ĞÄ¼¡Ï¸°ûºË¡¢Ã¸Ñ§·½·¨²â¶¨Ca2+-ATPase»îĞÔ¡¢45Ca2+Í¬Î»ËØ·¨²â¶¨Ë¸ÆÉãÈ¡ºÍÓ«¹â·Ö¹â¹â¶È¼Æ²â¶¨Ï¸°ûºËÄÚ×ÔÓÉ¸ÆÅ¨¶È,³õ²½½ÒÊ¾Ñ¹Á¦¬¸ººÉĞĜÄ¼¡·Êºñ´óÊóĞÄ¼¡Ï¸°ûºË¸Æ×ªµ¼Òì³£µÄ»·½Ú¡£½á¹û·¢ÏÖ:ĞÄ¼¡Ï¸°ûºËÉÏ´æÔÚ¾ßÓĞ[Ca2+]ºÍATPÒÀÀµĞÔµÄ¸ßÇ×ºÍÁ¦Ca2+-ATPase,ÒÔ[Ca2+]ÒÀÀµµÄ·½Ê½ÉãÈ¡45Ca2+,²¢³ÊÏÈÉı¸ßºó½µµÍÇ÷ÊÆ¡£AACÊõº ó4ÖÜ´óÊóĞÄ¼¡ÏÔÖø·Êºñ,°éÓĞÃ÷ÏÔµÄÑªÁ÷¶¯Á¦Ñ§Òì³£,Óë¶ÔÕÕ×é±È½Ï,AAC´óÊóĞÄ¼¡Ï¸°ûºËCa2+-ATPase»îĞÔ¼õÉÙ51.93% (p<0.001),µ«ºËCa2+ÉãÈëÁ¿(ºËÍâ[Ca2+]Å¨¶ÈÎª800-1600nmol/LÊ±)ºÍºËÄÚ[Ca2+](ºËÍâ[Ca2+]Å¨¶ÈÎª0-1000nmol/LÊ±)¾ùÃ÷ÏÔÖö¼Ó(p<0.05);Õı³£×éÀëÌåĞÄ¼¡Ï¸°ûºËCa2+ÉãÈ¡ÊÜPKA´Ì¼¤ (p<0.05),¶ø±»PKCÒÖÖÆ¼ÁºÍCaMÒÖÖÆ¼ÁÏÔÖøÒÖÖÆ(p<0.05),AAC´óÊóĞÄ¼¡Ï¸°ûºËCa2+ÉãÈ¡½öÊÜCaMÒÖÖÆ¼ÁÒÖÖÆ(p<0.01),¶øPKAºÍPKCÒÖÖÆ¼Á¶ÔäÎŞÃ÷ÏÔÓ°Ïì(p>0.05) ¡£½áÂÛÎªĞÄ¼¡·Êºñ£ĞÄ¼¡Ï¸°ûºËCa2+×ªÔËÏµÍ³¼°ÆäÁ×Ëá»¯µ÷½Ú¿ÉÄÜ·¢Éú¸Ä±ä¡£

ALTERATIONS IN CALCIUM TRANSPORT AND Ca^{2+}-ATPase ACTIVITY IN CARDIAC NUCLEI OF RAT HEART DURING OVERLOAD-INDUCED CARDIAC HYPERTROPHY

The hypertrophy rat model was established by abdominal aortic constriction, and differential centrifugation was used to fractionate the cardiac nuclei. The alterations in Ca^{2+}-ATPase activity and $^{45}Ca^{2+}$ uptake as well as intranuclear [Ca^{2+}] of the cardiac nuclei were investigated. The Ca^{2+}-ATPase activity in cardiac nuclei was decreased by 51.93% (P<0.001). $^{45}Ca^{2+}$ uptake were significantly augmented in the range of 800-1600 nmol/L of incubating free [Ca^{2+}], while intranuclear free [Ca^{2+}] elevated markedly in the range of 0-1000 nmol/L (P<0.05), as compared with those of control. PKC inhibitor (bisindolylmaleimide 5¦Ìmol/L) and CaM inhibitor (calmidazolium 1¦Ìmol/L) inhibited while PKA stimulated $^{45}Ca^{2+}$ uptake in the normalcardiac nuclei, but the $^{45}Ca^{2+}$ uptake in the hypertrophic cardiac nuclei were inhibited only by CaM inhibitor and not affected by PKA and PKC inhibitor. These results suggested that the alterations in calcium transport system in the myocardial nuclei may be partly responsible for the modified cardiac function in pressure overload-induced cardiac hypertrophy, while they also might be regulated by PKA, CaM and PKC.

¹Ø¼ü´Ê

ĞÄ¼¡·Êºñ(Cardiac hypertrophy); Ï¸°ûºË(Cell nucleus); Ca^{2+}-ATPase; ¸ÆÉãÈ¡($^{45}Ca^{2+}$ uptake)