Even after repair of aortic coarctation without restenosis there is a high incidence of arterial hypertension. This study was performed to assess the contribution of several inherited gene polymorphisms, which are known to be related to essential hypertension. 122 patients aged 17-72 years, 46 women, and 2-27 years after repair of isolated aortic coarctation without restenosis were investigated. Genomic polymorphism of angiotensin converting enzyme (ACE I/D), angiotensinogen (AGT, c.704C>T), angiotensin II receptor type 1 (AGTR1, c.1166A>C), aldosterone synthase (CYP11B2, c.-344C>T), endothelin 1 (EDN1, EDN1/ex5-c.5665G>T), G protein (GNB3, c.825C>T), G protein-coupled receptor kinase 4 (GRK4, c.679C>T), fibrillin 1 (FBN1, VNTR(TAAA)) and two polymorphisms each of the β1 adrenoreceptor (ADRB1, c.145G>A and c.1165C>G), β2 adrenoreceptor (ADRB2, c.46A>G and c.79C>G), and endothelial NO synthase (NOS3, intron 4 I/D and NOS3, c.894G>T) were determined by PCR amplification and fragment length analysis. Patients were classified "normotensive", if they were not on antihypertensive drugs and showed normal blood pressure both on ambulatory measurement and exercise test. None of the investigated genomic polymorphism could be related to hypertension. Only patients with the ACE I/I genotype had a less pronounced nocturnal dipping and patients with a ADRB1 c.1165 C/C genotype had a higher systolic and
mean blood pressure at night. Development of late hypertension after aortic coarctation repair could not be related to the investigated genomic polymorphism. The correlation of the ACE I/D and the ADRB1 c.1165C>G polymorphism to nocturnal dipping and blood pressure at nighttime needs further confirmation.

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