20) The Effect of Hyperbaric Oxygenation on Vascular Protein of Stroke-Prone and Resistant SHR. ¹Teruhiro Nakada, ¹Takashi Katayama, ²Haruo Saito, ²Kokichi Ota, ²Toshio Saegusa, ³Yukio Yamori, and ⁴Walter Lovenberg. ¹Department of Urology, Faculty of Medicine, Toyama Medical & Pharmaceutical University, Toyama 930-01, ²Saito Rosai Kyokai Hospital, Chiba 280, ³Department of Pathology, Shimane University, School of Medicine, Shimane 693, ⁴Section on Biochemical Pharmacology, NHLBI, NIH, Maryland USA.

It seems well established that hyperbaric oxygen (OHP) is a beneficial for a variety of patients with cardiac or circulatory insufficiency. However, controversy continued as to whether OHP treatment can really influence the protein synthesis of circulatory system in mammal. This study was undertaken to clarify the effects of OHP on the metabolism of vascular non-collagenous protein in genetically hypertensive rats. Data were obtained from a comparative study of 30 WKY, 31 SHR and 31 SHRSP subjected to repeated exposure of OHP treatments for 90 min daily from 6 weeks of age to 13 weeks of age. Group I, control, was handled similarly as pressure-treated groups, but at ambient oxygen pressure. Group II, high oxygen pressure, was exposed to 2 ATA under the environment of air saturating with 30 to 35% of oxygen. Group III, pressure control, was treated with 2 ATA of air without providing oxygen. Animals fasted overnight were injected iv with ³H-lysine at a dose of 0.4 μ Ci/g body wt. They were killed 2h after this injection. Blood-free hearts, aortas and mesenteric arteries were homogenized in hot 5% TCA. The precipitate was washed with cold 5% TCA. The tissue residue remaining after the TCA extraction was washed with 0.1 N NaOH, then heated at 95°C for 50 min. The residue from this extraction was washed with distilled water. The supernatant fraction containing non-collagenous protein was extracted by a modification method of Skosey et al. (Cir Res 31:145-157, 1972). The radioactivity of this protein fraction was counted, and expressed in dpm/mg protein. In each rat strain, no significant differences were noted in blood pressures between control group (I) and pressure-treated groups (II, III) during the experimental period. Incorporation of ³H-lysine into the non-collagenous protein of heart was similar among each rat strain. Increased incorporation of ³H-lysine into non-collagenous protein in mesenteric arteries in SHR and SHRSP was reduced ($p < 0.05$ - $p < 0.01$) by OHP treatments (Groups II, III). Similar tendency was also observed in aortas of hypertensive rats in lesser extent. The free ³H-lysine radioactivity of serum was almost similar among each pressure-treated or non-treated groups. With respect to the treatment of OHP, time of exposure to oxygen or degree of compression is important. The current findings in our laboratory provide a basis that our OHP-treatment is adequate to produce effective results in mammal vasculatures. Increased incorporation of ³H-lysine into the non-collagenous protein of the mesenteric arteries in hypertensive rats was confirmed in this experiment. Although the development of blood pressure in SHR or SHRSP was not prevented by OHP treatment, the increased incorporation of this protein fraction into the mesenteric arteries was decreased by this treatment. The enhanced non-collagenous protein synthesis in small arteries is one of the initial events in young SHR and SHRSP, but additional factors contributing to maintain hypertension must be considered in adult genetically hypertensive rats especially of their severe hypertensive stage.