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PROTECTIVE EFFECTS OF HYDROCEPHALUS IN POST-DOCA-NACl HYPERTENSION AND VASCULAR INJURIES IN DAHL R RATS

Jong Y. Lee. Dept of Medicine, University of Minnesota, Minneapolis, MN, United States.

The effects of hydrocephalus on the heart, kidney, and post-DOCA hypertension (HTN) were investigated. DOCA-HTN were produced in 33 Dahl R rats with 150 mg/Kg DOCA silicone and 1% NaCl drinking water for 4 weeks. After a one week recovery from the DOCA and 1% NaCl with 0.3% low NaCl chow and tap water, the rats were divided into sham and blocked groups with matching mean BP, 148 mm Hg, and weight, 215 g. Following a 4 week-postsurgery recovery period with the same low NaCl diet, BP averaged 161±3.2 mm Hg and 146±2.3 mm Hg in 16 blocked rats, p<0.0001. Then both groups entered into an 8% high NaCl diet. After 4 weeks on the 8% NaCl diet, 17 sham rats’ BP averaged a further increase, while the 16 blocked rats showed only a slight rise, 186±2.6 weeks 154±3.7 mm Hg, p<0.0001. The sham group mortality rate was much higher on the 8% NaCl diet: at 8 weeks, 7 out of 17 sham rats died vs none in blocked rats, p<0.0001; at 11 weeks, 12 sham dead vs none in blocked rats, p<0.0001 (a 71% reduction). After 11 weeks on the 8% NaCl diet, tail venous P in the sham rats was much higher than that of the blocked rats, 29±5.9 mm versus 13±0.5 mm H2O, p<0.0001, indicating the end stage of kidney and heart failure. Sham rats’ wet and dry hearts weighed much higher than those of blocked rats, 2.15 vs 1.47; 0.46 vs 0.30, both p<0.0001, as well as wet and dry kidneys, 2.83 vs 2.05, p<0.0001; 0.56 vs 0.43, p<0.005, respectively. These results indicate that the aqueduct block prevents post-DOCA HTN and vascular injuries.

Key Words: Cardiac/Renal Hypertrophy, Hydrocephalus, Hypertension

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TRANSIENT PHYSIOLOGICAL CHANGES IN DOCA-DAHL RATS IN HYDROCEPHALUS

J Y. Lee. Dept of Medicine, University of Minnesota, Minneapolis, MN, United States.

AV3V lesions prevent DOCA-NaCl hypertension (HTN). To check effects of NaCl diets and hydrocephalic brains in post-DOCA HTN, baseline, transient physiological changes and long term adjusted results were examined. DOCA HTN was induced in 36 Dahl R rats with 250 mg/kg DOCA in silicone and saline water containing 1% NaCl and 0.2% KCl. After 4 weeks, the DOCA and saline were removed and the rats were on a recovery period with tap water and a 0.3% low NaCl chow. One week later, the aqueduct of Sylvius was blocked in one group and entered into an 8% high NaCl diet. After 4 weeks on the 8% NaCl diet, tail venous P in the sham rats was much higher than that of blocked rats, 161±3 mm Hg, p<0.0001. The sham group mortality rate was much higher on the 8% NaCl diet: at 8 weeks, 7 out of 17 sham rats died vs none in blocked rats, p<0.0001; at 11 weeks, 12 sham dead vs none in blocked rats, p<0.0001 (a 71% reduction). After 11 weeks on the 8% NaCl diet, tail venous P in the sham rats was much higher than that of the blocked rats, 29±5.9 mm versus 13±0.5 mm H2O, p<0.0001, indicating the end stage of kidney and heart failure. Sham rats’ wet and dry hearts weighed much higher than those of blocked rats, 2.15 vs 1.47; 0.46 vs 0.30, both p<0.0001, as well as wet and dry kidneys, 2.83 vs 2.05, p<0.0001; 0.56 vs 0.43, p<0.005, respectively. These results indicate that the aqueduct block prevents post-DOCA HTN and vascular injuries.

Key Words: Transient Plasma NA, Sylvian Aqueduct, DOCA-Hypertension

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IMPAIRMENT OF VASCULAR FUNCTION IN COLD STRESS INDUCED HYPERTENSION

Zhiming Zhu, Changqing Yu, Lijuan Wang, Haiyan Wang, Shanjian Zhu. Department of Hypertension and Endocrinology, Chongqing Hypertension Institute, Daping Hospital, Third Military Medical University, Chongqing, China.

There are a number of epidemiological studies reporting seasonal variability of blood pressure. Colder seasons causing high blood pressure are found in both hypertensive and normotensive subjects. The mechanisms for cold-induced hypertension (CIH) are poorly understood. To investigate the role of cold stress in the development and maintenance of hypertension, three month old Wistar rats were randomly divided into two groups: (1) Cold-stress rats (n=20): rats were exposed to cold (-2 centigrade) for 4 hours per day for 8 days; (2) Control rats (n=20): rats were housed at room temperature. Systolic blood pressure (SBP) and heart rate (HR) were measured by tail-cuff technique weekly. Isometric force of aortic ring was measured using a force transducer. SBP and HR were time-dependent increased in cold stress rats. SBP and HR were not significantly changed through eight weeks in control rats. SBP was significantly higher in cold stress rats than in control rats (136.1±6 mmHg vs 105.5±5 mmHg, p<0.01). Reactivity of aortic ring to norepinephrin and angiotensin II stimulation were not different in cold stress and control rats. However, the contraction of aortic ring to Bayk8644, a L-type calcium channel activator, stimulation significantly increased in cold stress rats compared with control rats (40.05±6.15 vs 31.30±5.22, p<0.01). The endothelium dependent relaxation of aortic ring to acetylscholine stimulation significantly decreased in cold stress rats compared with control rats (9.40±2.55 vs 16.23±5.10, p<0.01). The endothelium-independent relaxation of aortic ring to nitroglycerin stimulation was not different between cold stress and control rats (8.60±2.70 vs 9.25±2.35, p>0.05). It concluded that cold stress plays an important role in the development of high blood pressure and vascular dysfunction. The mechanisms may be involved in activation of vascular calcium channel and reduced endothelium-dependent vasorelaxation in CIH.

(Supported by NSFC grant No: 30070315, 39725013).

Key Words: Vascular Reactivity, Cold Stress, Hypertensive Rat

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VASCULAR SMOOTH MUSCLE CELL-DIRECTED OVEREXPRESSION OF HEME OXYGENASE-1 ATTENUATES NITRIC OXIDE-ELICITED CGMP INCREASE, LEADING TO ELEVATION OF BLOOD PRESSURE IN MICE

Toshihiko Morita, Tomohiko Imai, Takayuki Shindo, Ryozo Nagai, Hiroki Karihara, Yoshio Yazaki, Shigehiro Katayama. Laboratory Medicine, School of Medicine, Toho University, Tokyo, Japan; Fourth Department of Internal Medicine, Saitama Medical School, Saitama, Japan; Department of Cardiovascular Medicine, University of Tokyo, Tokyo, Japan; International Medical Center of Japan, Tokyo, Japan.

To elucidate the pathophysiological role of heme oxygenase (HO)-1 in regulation of vascular tone in vivo, we have developed and characterized transgenic (Tg) mice that overexpress HO-1 site-specifically in vascular smooth muscle cells (VSMCs). The Tg mice were generated by use of human HO-1 cDNA under the control of SM22-alpha promoter. The