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Nocturnal Blood Pressure Decrease in Hypertensive Patients and Normotensives- Association with Obstructive Sleep Apnoea and Renal Function

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SUPPORTIVE/SUPPLEMENTARY MATERIAL

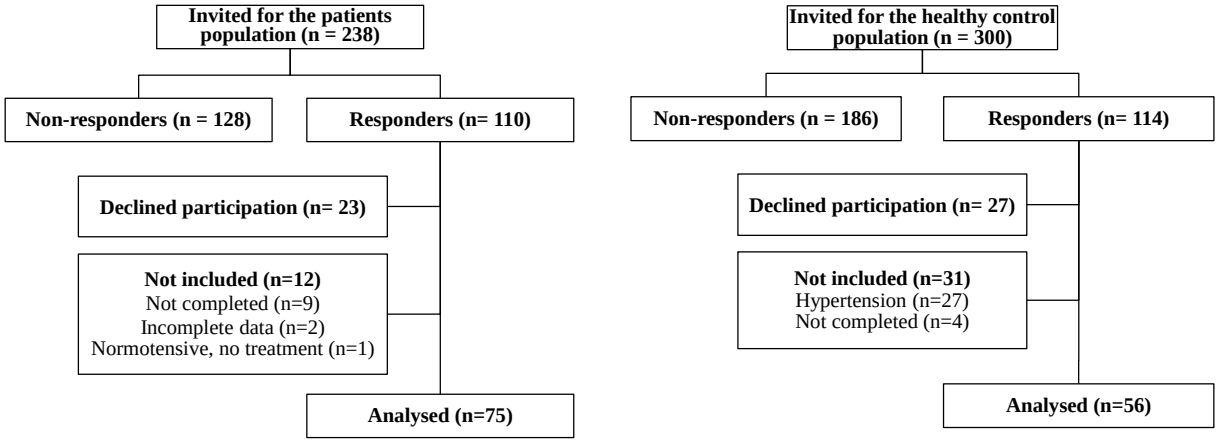

Fig. S1. Flow chart of hypertensive patients and healthy controls.

SUPPLEMENTAL MATERIAL. CARDIO RESPIRATORY MONITORING

Sleep time was set from the time where the subject fell asleep until awaken. This was done by visual inspection of sleep data. The sleep report was generated from sleep time and consisted of continuous recordings of air flow from a nasal pressure transducer, arterial oxygen saturation from a pulse oximeter, respiratory effort from thoracic and abdominal impedance belts, snoring from microphone recordings, and body position from sensors. Apnoea was defined as a cessation of nasal airflow or drop of the signal below 10% of the reference amplitude for ≥ 10 second. Hypopnoea was defined as a reduction of the signal below 70% of the reference amplitude for ≥10 second followed by a desaturation event no later than 20 seconds after the start of the hypopnoea. An oxygen desaturation event was detected when oxygen saturation fell by at least 4%. All events (apnoea, hypopnea, or desaturation) lasting longer than 120 seconds were excluded.

SUPPLEMENTAL MATERIAL. BIOCHEMICAL ANALYSES

Plasma and urinary osmolality were measured by freezing-point depression (A_2O Advanced Automated Osmometer, Advanced Instruments, Norwood, MA, USA).

P-AVP and p-Ang II were extracted from plasma with C18 Sep-Pak (Water associates, Milford, MA, USA), and subsequently determined by radioimmunoassay [1, 2]. The antibody against AVP was a gift from Professor Jacques Dürr, Miami, FL., USA. Minimal detection level was 0.5 pmol / L. The coefficients of variation were 13% (inter-assay) and 9% (intra-assay). The antibody against Ang II was obtained from the Department of Clinical Physiology, Glostrup Hospital, Denmark. Minimal detection level was 2 pmol/L. The coefficients of variation were 12% (inter-assay) and 8% (intra-assay).

PRC was determined by radioimmunoassay using a kit from CIS Bio International, Gif-Sur-Yvette Cedex, France. Minimal detection level was 1 pg/mL. The coefficients of variations were 14.5% (inter-assay) and 4.5% (intra-assay).

P-BNP was determined by chemiluminescent microparticle immunoassay (Architect BNP, Abbott Laboratories, Illinois, USA) by the Department of Clinical Biochemistry, Holstebro Hospital, Denmark. Minimal detection level was 2.89 pmol/L. The coefficients of variations were ≤12% (inter-assay) and 10% (intra-assay).

P-Aldo was determined by radioimmunoassay using a kit from Demeditec Diagnostics GmbH, Kiel, Germany. Minimal detection level was 3.99 pmol/L. The coefficients of variations were 17.2% (inter-assay) and 12.6% (intra-assay).

U-AQP2 was measured by radioimmunoassay as previously described [3, 4]. Antibodies were raised in rabbits for a synthetic peptide corresponding to the 15 COOH-terminal amino acids in human AQP2, to which was added an NH2-terminal cysteine for conjugation and affinity purification. Minimal detection level was 32 pg / tube. The coefficients of variation were 11.7% (inter-assay) and 5.9% (intra-assay).

U-ENaCγ was measured by RIA as described previously [5, 6]. ENaCγ was synthesized and purchased by Lofstrand, Gaithersburg, Maryland, USA. The ENaCγ antibody was a gift from Professor Soren Nielsen and Professor Robert Fenton, The Water and Salt Center, Aarhus University. Minimal detection level: 35 pg/tube. Coefficients of variation: 10% at a mean level of 338 pg/tube (inter-assay), 9 % at a mean level of 743 pg/tube (inter-assay), 5.0 % in the range 125-135 pg/tube (intra-assay), and 5.6 % in the range 290-380 pg/ tube (intra-assay).

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