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C. Strandhave, K.N. Hvidt, L. Boesby, H.S. Bosselmann, C.D. Peters, D. Khatir, E. Laugesen, S.W. Greve, N. Wiinberg

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may warrant more intensive strategies in preventing further deterioration of their physiology.

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A MEDICAL CONFERENCE DINNER'S IMPACT ON CENTRAL BLOOD PRESSURE AND VASCULAR AGE

C. Strandhave¹, K. N. Hvidt², L. Boesby³, H. S. Bosselmann³, C. D. Peters⁴, D. Khatir⁴, E. Laugesen⁵, S. W. Greve⁶, N. Wiinberg⁷

¹Dept. of Nephrology, Aalborg University Hospital, Aalborg, Denmark

²Division of Cardiology, Dept. of Medicine, Copenhagen University Hospital, Holbaek, Denmark

³Dept. of Cardiology Nephrology Endocrinology, Nordsjaelland Hospital, Hilleroed, Denmark

⁴Dept. of Renal Medicine, Aarhus University Hospital, Aarhus, Denmark

⁵Dept. of Endocrinology and Internal Medicine, Aarhus University Hospital, Aarhus, Denmark

⁶Dept. of Endocrinology, Odense University Hospital, Odense, Denmark

⁷Dept. of Clinical Physiology, Copenhagen University Hospital, Frederiksberg, Denmark

Central blood pressure (BP) is recognised as a predictor of cardiovascular disease (CVD). Moderate alcohol consumption has been shown to have a beneficial effect on CVD. However, consumption of a lipid-rich meal may exert the opposite effect. Thus, the aim of the present study was to examine whether the immediate effect of a medical conference dinner was associated with reduced central BP and hence improved vascular age.

We examined attendees at a medical conference before and after the conference dinner which included a three course meal and wine menu. Participants had brachial and central BP measured. Central BP was measured in duplicate over the right radial artery using the Sphygmocor device (Atcor Medical, Sydney, Australia).

The cohort consisted of 60 attendees (43% women) with a median age of 40 years (IQR 35 – 54) and a mean follow-up period of 4 ± 1 hour. Only one attendee smoked, whereas six took antihypertensive medication. While heart rate increased, all measurements of brachial and central BP were reduced after the dinner (Table 1). Multiple regression showed that central systolic BP and augmentation index (Alx) was reduced after the dinner independently of age, gender, height, and baseline heart rate ($p = 0.008$ and $p = 0.01$). Furthermore, calculations of the slope of the regression lines between $Alx@HR75$ and age before and after the dinner revealed a reduction of 5.5 years in the vascular age.

In conclusion, central BP was reduced and vascular age improved by 5.5 years after intake of a medical conference dinner.

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EFFECTS OF IVABRADINE AND ATENOLOL ON CENTRAL AORTIC PRESSURE IN HYPERTENSIVE PATIENTS WITH STABLE ANGINA

A. Shavarov, G. Kiyakbaev, V. Moiseyev, Z. Kobalava
PFUR, Moscow, Russian Federation

The aim of the study was to assess the effects of ivabradine (I) and atenolol (A) on central aortic pressure in patients with stable angina and arterial hypertension (AH).

Methods: The study was conducted on 31 hypertensive patients (57.1% male), mean age 61.9 ± 8.4 years, with angina pectoris of II-III functional class and without history of myocardial infarction and chronic heart failure. Two weeks before randomization all patients received nifedipine SR (30 mg o.d.) additionally to antihypertensive treatment. Then patients were randomly assigned to I ($n=15$) and A ($n=16$) dose up-titration for 2 weeks. Next 4 weeks doses of I and A were consistent. Mean doses were 14,4 mg for I and 137,5 mg for A. Heart rate, peripheral systolic (SBP) and diastolic blood pressure (DBP), central aortic systolic (CSP) and diastolic pressure (CDP), aortic pulse pressure (PP) were measured at baseline and at the end of the study.

Results: Heart rate (HR) decreased from 74.0 to 54.0 bpm with I and from 74.5 to 54.5 bpm with A (both $p=0.001$). SBP decreased from 132.0 to 129.5 mm Hg with I ($p=0.55$) and from 132.0 to 122.0 with A ($p=0.01$). DBP decreased from 80.0 to 79.5 mm Hg with I ($p=0.96$) and from 80.0 to 76.0 mm Hg with A ($p=0.001$). CSP decreased by 6.9 mm Hg with I ($p=0.01$) and 8.0 mm Hg with A ($p=0.002$). DSP decreased by 3.0 mm Hg with I ($p=0.01$) and 4.0 mm Hg with A ($p=0.004$). PP decreased by 4.0 mm Hg after I ($p=0.64$), while PP increased by 5.0 mm Hg after A ($p=0.76$).

Conclusion: After achieving of target HR ivabradine without influence on peripheral BP decreased CSP compared with atenolol.

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DIURNAL VARIATION OF CARDIO-ANKLE VASCULAR INDEX IN INDIVIDUALS WITH AND WITHOUT HEART DISEASE

Yanlei Li, Cordes Mareike, Hanssen Henner, Schmidt-Trucksäss Arno
Division Sports Medicine, Institute of Exercise and Health Sciences, University of Basel, Basel, Switzerland

Background: Clinical studies revealed age and pathological-related arterial stiffening. Arterial stiffening is associated with a higher risk of cardiovascular disease. Cardio-ankle vascular index (CAVI), which reflects both central elastic and peripheral muscular arterial stiffness, has been applied as a simple noninvasive method to evaluate the risk for cardiovascular events. However, whether it is necessary to standardize the time of the day when performing this measurement is unknown. We aim to examine the effect of daytime on CAVI in individuals with and without heart disease.

Methods: We investigated the daytime variation of CAVI using Vasera VS-1500N (Fukuda Denshi; Japan) in 23 healthy young individuals (28.3 ± 4.7 yr, HY), 22 healthy elderly individuals (61.1 ± 9.0 yr, HE) and 25 patients with heart disease (63.9 ± 11.5 yr, HD).

Results: The effect of time on CAVI was shown to be significant in both univariate and multivariate analysis. Age was found as a significant determinant of CAVI ($p < 0.001$). After adjustment for age, sex and MAP, CAVI was shown to be 4% (09:00 versus 13:00, $p=0.022$) and 5% higher (09:00 versus 17:00, $p=0.002$) in the morning than the following time points. Furthermore, the patterns of variation over the day showed no significant differences among groups in CAVI.

Conclusion: CAVI showed a significantly higher value in the morning, which provides further support to standardize the time for measurements of arterial stiffness using CAVI in routine clinical practice and longitudinal studies.

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24 HOURS PULSATILE HEMODYNAMICS IN BORDERLINE VERSUS RESISTANT HYPERTENSIVES

T. Weber¹, B. Hametner², B. Eber¹, S. Wassertheurer²

¹Cardiology Department, Klinikum Wels-Grieskirchen, Wels, Austria

²Health & Environment Department, AIT Austrian Institute of Technology, Vienna, Austria

Background: Diurnal variations of brachial blood pressure have important prognostic implications. The aim of this study was to investigate differences in day- and night-time values of brachial and central hemodynamic parameters in two groups of borderline and resistant hypertensives.

Methods: We performed 24 hour pulse wave analysis, using a brachial cuff and validated ARCSolver algorithms. Central pressures were derived with a generalized transfer function using measured mean and diastolic pressure for calibration. 50 borderline hypertensives (BH; mean age: 52 years, mean 24h brachial blood pressure (bBP): 123/81 mmHg) and 25 resistant hypertensives (RH; mean age: 58 years, mean 24h bBP: 138/85 mmHg) were included in the study. Day-time was specified as 09:00-21:00 and night-time as 01:00-06:00.

		BH	RH
Brachial systolic BP (mmHg)	Day	127*	139 ⁵
	Night	113	135 ⁵
Central systolic BP (mmHg)	Day	128	144 ⁵
	Night	123	147 ⁵
Brachial pulse pressure (mmHg)	Day	41	51 ⁵
	Night	40	54 ⁵
Central pulse pressure (mmHg)	Day	41*	54 ⁵
	Night	49	64 ⁵
Heart rate (bpm)	Day	72*	69*
	Night	59	60
Alx	Day	23*	29* ⁵
	Night	29	38 ⁵
Alx@75	Day	22	25*
	Night	20	30 ⁵
Amplitude backward wave (mmHg)	Day	16*	22* ⁵
	Night	21	28 ⁵
Reflection magnitude	Day	61*	64*
	Night	70	71

* $p < 0.05$ day vs night; ⁵ $p < 0.05$ BH vs RH.