anaemia or/and worse glycaemic profile ($p<0.01$ for all comparisons). In NGP patients LVMI was 119.0±15.0 in anaemics vs 118.7±15.3 in non-anaemics, in IGT 131.4±14.4 vs 127.3±16.9, in diabetics 144.2±17.3 vs 131.9±14.9 and in total 126.7±18.7 vs 121.9±16.3. In NGP patients LVH prevalence was 47.0% in anaemics vs 41.0% in non-anaemics, in IGT 82.6% vs 68.5%, in diabetics 95.1% vs 83.1% and in total 63.7% vs 51.2%. In NGP patients ACR was 35.0±24.0 in anaemics vs 32.5±28.7 in non-anaemics, in IGT 62.5±37.7 vs 52.6±36.1, in diabetics 126.0±136.9 vs 85.9±47.9 and in total 60.1±78.7 vs 43.1±38.2. In NGP patients micoalbuminuria prevalence was 43.7% in anaemics vs 35.4% in non-anaemics, in IGT 77.9% vs 67.3%, in diabetics 95.4% vs 89.5% and in total 61.1% vs 48.0%. In NGP patients eGFR was 74.2±17.3 in anaemics vs 74.7±17.6 in non-anaemics, in IGT 61.2±15.6 vs 66.4±15.4, in diabetics 50.5±9.5 vs 60.6±13.4 and in total 66.7±18.5 vs 71.4±17.6. Percentage differences between anaemics and non-anaemics were 0.3, 3.2, 9.3% for LVMI, 7.7, 18.8, 46.7% for ACR and -0.7, -7.7, -16.5% for eGFR in NGP, IGT, diabetic hypertensives, respectively.
Conclusion(s): The prevalence of TODs in hypertensives is higher in the presence of anaemia especially in those with impaired glycaemic profile. Underlying mechanisms need to be explored.

P4779
Comparison of ankle-brachial index and upstroke time per cardiac cycle in association with target organ damage in elderly Chinese: the Northern Shanghai Study

S.K. Yu[1], Y.W. Xu[1], Y. Zhang[1], J. Blacher[2], Y.Y. Lu[1], J. Xiong[1], C. Chi[1], J. Teliewubai[1]. *[1]Shanghai Tenth People's Hospital, Tongji University School of Medicine, Cardiology, Shanghai, China People's Republic of; [2]Hospital Hotel-Dieu, Diagnosis and Therapeutic Center, Paris, France*. On behalf of the Northern Shanghai Study

Background: Recent studies indicated that upstroke time per cardiac cycle (UTCC) in lower extremities is equivalent to ankle-brachial index (ABI) in diagnosing peripheral arterial disease and in predicting cardiovascular mortality.
Purpose: This study aims to compare ABI and UTCC on their associations with target organ damage (TOD).
Methods: 1841 elderly participants from the Northern Shanghai Study were included. ABI and UTCC were measured using VP-1000 device. TOD including left ventricular hypertrophy and diastolic dysfunction, carotid intima-media thickness and plaque, carotid-femoral pulse wave velocity (CF-PWV) and renal damage, were all evaluated.
Results: When ABI and UTCC separately put into same multivariate full-mode logistic regression models, both ABI (OR: 2.273; 95% CI: 1.632–3.165) and UTCC (OR: 1.627; 95% CI: 1.182–2.240) significantly associated with carotid plaque, but only UTCC significantly associated with increased CF-PWV (OR: 1.664; 95% CI: 1.147–2.416) and renal damage (OR: 1.625; 95% CI: 1.068–2.472). When ABI and UTCC both put into same multivariate stepwise logistic regression models, consistent results were observed. In ROC curve analysis, UTCC was better than ABI in discriminating increased CF-PWV (AUC: 0.68 vs. 0.57; $P<0.001$) and renal damage (AUC: 0.67 vs. 0.60; P=0.012). Variables staying in the final model are presented in the table.
Conclusion: Compared with ABI, UTCC showed stronger association with vascular and renal damage in this elderly cohort, which suggested that UTCC may be a useful tool for diagnosing PAD and stratifying cardiovascular risk.
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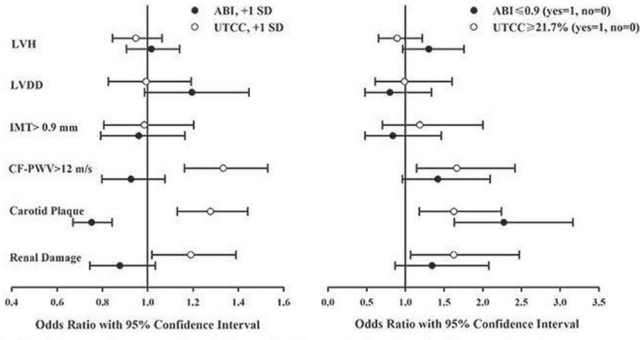
Figure 2. Association of TOD with ABI and UTCC analyzed by multivariate regression when ABI and UTCC separately put into the same model



Multivariate regression was conducted to investigate the association of each TOD with ABI and UTCC. Age, gender, body mass index, current smoking, hypertension, diabetes mellitus, total cholesterol, and high-density lipoprotein were forced into all models. ABI and UTCC were put into same models separately. The left, ABI and UTCC were in continuous form. The right, ABI and UTCC were in categorical form. ABI, ankle brachial index; UTCC, upstroke time per cardiac cycle; LVH, left ventricular hypertrophy; LVDD, left ventricular diastolic dysfunction; IMT, carotid intima-media thickness; CF-PWV, carotid-femoral pulse wave velocity.

P4780
Activation of astrocytes is requred for the persistence of post-stress blood pressure elevation

Y. Hasebe[1], S. Yokota[2], K. Takeda[3], S. Sugama[4], Y. Kono[1], K. Koizumi[1], I. Fukushi[5], M. Hoshiai[1], Y. Kakinuma[4], M. Pokorski[6], J. Horiuchi[7], K. Sugita[1], Y. Okada[5]. *[1]University of Yamanashi, Department of Pediatrics, Graduate School of Medicine, Yamanashi, Japan; [2]Shimane University, Anatomy and Morphological Neuroscience, Izumo, Japan; [3]Fujita Health University, Faculty of Rehabilitation, School of Health Sciences, Mie, Japan; [4]Nippon Medical School, Department of Physiology, Tokyo, Japan; [5]National Hospital Organization Murayama Medical Center, Clinical Research Center, Musashimurayama, Tokyo, Japan; [6]Opole Medical School, Faculty of Physiotherapy, Opole, Poland; [7]Toyo University, Department of Biomedical Engineering, Graduate School of Science & Engineering, Saitama, Japan*

Background: When psychological stress is loaded to a subject, the sympathetic nervous system is excited, which reflexively evokes blood pressure (BP) elevation. Even after the stress is relieved, BP elevation persists. However, cellular mechanisms of stress induced persistent BP elevation have not been fully clarified. Because sustained BP elevation could be a consequence of neural plasticity of the sympathetic nervous activity and it has been recently revealed that not only neurons but astrocytes play active roles in induction of neural plasticity in various brain functions, we hypothesized that astrocytes are involved in post-stress persistent BP elevation.
Purpose: We aimed to test this hypothesis by analyzing the effects of an inhibitory modulator of astrocytic function on responses of BP and heart rate (HR) to stress loading.
Methods: Responses of BP and HR to air-jet stress were analyzed in unanaesthetized rats before and 60 min after injection of arundic acid which selectively suppresses activation of astrocytes. Further, the effects of arundic acid on air-jet stress induced activation of neurons were examined by c-Fos immunohistochemistry in brain cardiovascular regions (PVN, DMH and RVLM) under four difference conditions (air-jet stress/arundic acid; 1: (–)/(–), 2: (+)/(–), 3: (–)/(+), 4: (+)/(+)).
Results: The mean arterial blood pressure (MAP) and HR before stress loading were not different among the conditions before and after administration of arundic acid. Air-jet stress evoked immediate elevation of MAP, and the elevation of MAP persisted during stress loading in the control and all drug conditions. However, the elevation in MAP, assessed as a difference before and during stress loading, was significantly smaller in each dose-group of arundic acid than that in the control group. The mitigating effect of arundic acid on the BP elevation during stress loading was dose-dependent. After the stress loading, the persistent elevation of MAP was observed in control group. However, such post-stress sustained BP elevation was not observed in any arundic acid groups. Air-jet stress evoked immediate increases of HR in control and all drug conditions. Increases of HR during stress loading as compared to the pre-stress condition were less in the arundic acid groups, but the differences were not significant. Histochemically, before arundic acid but after loading of air-jet stress, the numbers of c-Fos positive cells were markedly increased in brain cardiovascular regions. However, in the groups pretreated with arundic acid, the numbers of c-Fos positive cells were not different between the conditions without and with air-jet stress.
Conclusion: We demonstrated that astrocytes, beside neurons, are actively involved in the post-stress sustainment of BP elevation. These findings suggest the causative relationship between astrocytes and hypertension. Astrocytes could be a target in the development of anti-hypertensive drugs.

Abstract P4779 – Table 4. Association of TOD with ABI and UTCC analyzed by multivariate stepwise regression when ABI and UTCC both put into the same model

	LVH OR (95% CI)	LVDD OR (95% CI)	IMT>0.9mm OR (95% CI)	CF-PWV>12m/s OR (95% CI)	Carotid Plaque OR (95% CI)	Renal Damage OR (95% CI)
Age, year	1.027 (1.009, 1.045)	–	1.069 (1.037, 1.102)	1.109 (1.082, 1.137)	1.062 (1.042, 1.082)	1.174 (1.141, 1.209)
Gender (male=1, female=0)	0.280 (0.221, 0.355)	0.467 (0.323, 0.677)	1.568 (1.003, 2.450)	–	1.279 (1.019, 1.606)	–
Body mass index, kg/m^2	1.075 (1.041, 1.110)	1.088 (1.035, 1.144)	–	–	–	–
Current smoking (yes=1, no=0)	–	–	1.748 (1.014, 3.015)	–	–	–
Hypertension (yes=1, no=0)	1.961 (1.546, 2.487)	1.996 (1.323, 3.011)	–	2.534 (1.691, 3.798)	–	1.554 (1.024, 2.359)
Diabetes (yes=1, no=0)	–	–	–	2.276 (1.618, 3.201)	1.422 (1.101, 1.838)	–
Total Cholesterol, mmol/L	–	–	–	1.277 (1.104, 1.477)	1.154 (1.041, 1.279)	1.176 (0.986, 1.403)
High density lipoprotein, mmol/L	–	–	–	–	–	0.343 (0.188, 0.626)
ABI<0.9 (yes=1, no=0)	–	–	–	–	2.085 (1.490, 2.919)	–
UTCC≥21.7% (yes=1, no=0)	–	–	–	1.718 (1.187, 2.486)	1.387 (1.000, 1.925)	1.673 (1.110, 2.520)