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ARTERIAL STIFFNESS MEASURED BY PULSE WAVE VELOCITY IN PATIENTS WITH EARLY SEPSIS

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Sepsis is characterised by massive inflammatory response, which can affect vascular function. This study was designed to assess the impact of early severe sepsis and septic shock on arterial stiffness and the relationship of this impact to outcome. Twelve patients with severe sepsis and 22 with septic shock were included in the study. We measured carotid to femoral and carotid to radial pulse wave velocity (PWV), an index of aortic and brachial arterial stiffness, in patients with early severe sepsis and septic shock within 24 hours of admission to intensive care unit and repeatedly after 48 hours. No difference was observed between patients with severe sepsis and septic shock regarding carotid to femoral PWV (11.7 ± 2.2 vs. 11.3 ± 3.6 m/s) and carotid to radial PWV (12.0 ± 3.8 vs. 9.5 ± 2.2 m/s). On 48 hour follow-up, PWV did not significantly differ between survivors and non-survivors. A positive, similar correlation occurred between PWV and pulse pressure in all patients (r = 0.35, p *= 0.05), and there was a negative correlation between PWV and C-reactive protein levels (r = -0.43,* p *= 0.04). In conclusion, PWV is not affected by disease severity or prognosis.*

Key words: *arterial stiffness, sepsis, endothelium, inflammation.*

INTRODUCTION

Among the critically ill patients with sepsis, multiple organ failure and high mortality occurs in a significant proportion. The development of sepsis occurs as a result of systemic inflammatory response syndrome to infection (Nystrom, 1998). However, the outcome of sepsis is not determined by the infection itself, but mostly by the response of the host (Cohen, 2002). A central part of host response is endothelial cell dysfunction, which leads to vascular dysfunction and/or injury. Sepsis induces a cascade of inflammatory cytokines and mediators with vasoactive properties, such as nitric oxyde (NO). Inducible nitric oxide appears to play a central role in septic vascular dysfunction (Bone, 1991). Large arteries can be affected as part of vascular dysfunction. Data obtained in experimental studies show that acute systemic inflammation leads to a temporary increase of large artery stiffness (Vlachopoulos *et al*., 2005) but effect of massive inflammatory response in early sepsis on large artery stiffness has not been previously investigated.

Systemic arteries can be subdivided into two types — muscular and elastic. Aorta is the largest elastic artery in the human body. The measurement of carotid to femoral pulse wave velocity (PWV) is a recognised index of aortic distensibility and stiffness, it can be measured noninvasively and has been shown to be an important predictor of mortality in various chronic diseases (Blacher *et al*., 1999, Blacher *et al*., 1999, Stefanadis *et al*., 2000). The brachial artery is an easily accessible muscular artery. Carotid to radial PWV, which represents mainly brachial artery stiffness, is more likely to be influenced by endothelial nitric oxide (eNO) production than carotid to femoral PWV. Also, as vascular dysfunction in sepsis is functionally linked with basal eNO production, it is likely to be more influenced by sepsis. The objective of the study was to assess aortic and brachial stiffness in patients with early severe sepsis and septic shock. We investigated carotid to femoral and carotid to radial PWV as an index of sepsis-induced changes in elastic and muscular arteries, and the clinical, inflammatory, and

haemodynamic parameters correlated with PWV in such patients and their relationship to outcome.

MATERIALS AND METHODS

The investigation was conducted according to the principles outlined in the Declaration of Helsinki. The study protocol was approved by Rīga Stradiņš University Ethics Committee and informed consent was given by each patient or their next of kin.

Study population. Consecutive patients hospitalised to the 12-bed Toxicology and Sepsis Clinic of Rīga East Clinical University Hospital "Gaiļezers" with a diagnosis of severe sepsis or septic shock were enrolled (Levy *et al*., 2003). Patients were excluded if they were pregnant, aged <18 years, were requiring nitroglycerin or had aortic surgery. Septic shock was defined as severe sepsis with sepsis-induced hypotension persisting despite adequate fluid resuscitation, and requiring the administration of vasopressor agents. The clinical management of each patient was determined by clinical staff in accordance with local guidelines.

Pulse wave velocity measurement. Pulse wave velocity (PWV) was measured using an originally designed two-channel photoplethysmograph (PPG) (LU ASI, Latvia) with the patient in supine decubitus position. Two reflecting sensor probes were used to measure the arterial waveforms, first at carotid and femoral arteries, then at carotid and radial arteries. The analogue signals from PPG contact probes were digitised by an analogue-to-digital converter (16-bit accuracy, sampling rate 300 Hz) and transferred to the computer (Erts, 2005). The time delay between upstrokes of the two simultaneous arterial waveforms was measured using the 'foot-to-foot' method to obtain transit time of the arterial pulse wave along the artery, ΔT. The measured surface body distance in metres (m) between both measurement points (carotid and femoral arteries, and carotid and radial, respectively), L, was multiplied by 0.8, as this yields the best agreement with the reference aortic path length (Huybrechts *et al*., 2011). PWV in m/s was calculated using the equation: $PWV = L*0.8/\Delta T$.

Study protocol. Patients were enrolled within 24 h after admission to the Intensive Care Unit. Information collected at enrolment included demographic characteristics on age, sex, weight, and height (to calculate body mass index, BMI); Acute Physiology and Chronic Health Evaluation (APACHE) II score (Knaus 1985); Sequential Organ Failure Assessment (SOFA) score (Vincent *et al*., 1996); and primary site and type of infection.

When the patient was haemodynamically stable (mean arterial pressure (MAP) > 65 mmHg and no change in vasopressor infusion rate for 1 h), the PPG probes were placed on the skin over maximal pulsation of carotid artery on the neck and femoral artery in the groin for recording of carotid to femoral pulse wave delay, and on the skin over maximal pulsation of carotid artery on the neck and radial artery on the wrist for recording of carotid to radial pulse wave time delay. The PPG signal was recorded for 3 minutes on both sites. This process was repeated in 48 hours.

The following measurements were also obtained: arterial pressure; doses of vasopressor agents.

Statistical analysis. Discrete variables are expressed as counts (percentage) and continuous variables as mean ± SD or median (25th–75th percentiles). For demographic and clinical characteristics, differences between groups were assessed using a chi-square, Fisher's exact test, Student's t-test, or Mann–Whitney U-test. For the PWV variables in the different study groups, analysis of variance followed by a Tukey (HSD) test with adjustment for multiple comparisons or a Kruskal–Wallis test were used. Linear regression analysis (Pearson's analysis) was used to assess correlations of continuous variables. We considered $p < 0.05$ to be significant.

RESULTS

We studied 34 patients with severe sepsis (median age 62 years, interquartile range 50–76), including 12 patients without and 22 patients with septic shock (Table 1). Characteristics of the study groups are shown in Table 2.

Pulse wave velocity. Carotid to femoral pulse wave velocity was not recordable in one male patient from the septic shock group due to bilateral above knee amputation secondary to peripheral vascular disease. Carotid to femoral and carotid to radial PWV values for patient groups with severe sepsis and septic shock are shown in Figure 1. No difference was observed between the groups ($p = 0.88$). Median values for carotid to femoral PWV were 11.7 ± 2.2 m/s in the severe sepsis group (range 8.4 to 16.6), 11.3 ± 3.6 m/s in the septic shock group (range 7.1 to 18.0), median values for carotid to radial PWV were 12.0 ± 3.8 m/s in the severe

Table 1

CLINICAL DATA OF PATIENTS (n = 34)

Primary site of infection (n, %)	
Respiratory tract	11 (32)
Abdomen	10 (29)
Urinary tract	3 (9)
Skin/soft tissue	6 (18)
Other	2 (6)
Unknown	2 (6)
Type of organism (n,%)	
Gram positive	15 (44)
Gram negative	3 (9)
Mixed	5 (15)
Unknown	10 (29)
Vasoactive agents (n; dose in mcg/kg·min^{-1})	
Noradrenaline	19; 0.09 (0.06–0.12)
Non-survivors (n, %)	9 (26)

Table 2

DEMOGRAPHICS AND CLINICAL CHARACTERISTICS OF THE STUDY GROUPS

	All sepsis (n = 34)	Survivors (n = 25)	Non-survivors (n = 9)
Age (years)	62 (50.25–75.75)	60 (44.5–72.5)	76 (57–81)
Male (n, %)	19 (56)	16 (64)	3 (33)
BMI	26.25 (24.5–28.2)	25.7 (24.5–27.8)	25.7 (24.4–27)
MAP (mm Hg)	75 (70–81)	78.5 (70–85.5)	74 (69.5–77)
APACHE II	18.5 (14–24)	15.5 (13.5–20.25)*	27 (24–30)*
Initial SOFA	6.5 (4–9)	5 (4–7.5)*	11(9–12)*
Delta SOFA	3 (1–4.25)	3 (2–4)	–1(–1–5)

BMI, body mass index; MAP, mean arterial pressure.

*Significant difference at p = 5%

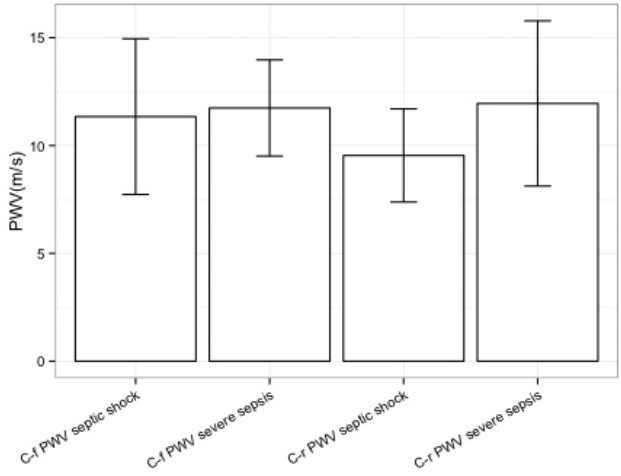

Fig. 1. Carotid-femoral and carotid-radial pulse wave velocity values for patients with severe sepsis and septic shock (p = 0.88).

sepsis group (range 7.6 to 24.9), 9.5 ± 2.2 m/s in the septic shock group (range 6.0 to 14.9).

Determinants of PWV in septic patients. We observed a significant correlation between carotid to femoral PWV and pulse pressure (r^2 = 0.35, p = 0.05) (Fig. 2). Carotid to femoral pulse wave velocity negatively correlated with C-reactive protein levels (r^2 = –0.43, p = 0.04) (Fig. 3). There was no correlation between carotid to femoral PWV and age (r^2 = 0.02).

No correlation between carotid to radial PWV and pulse pressure (r^2 = 0.05), C-reactive protein concentration (r^2 = 0.07) or noradrenaline dose (r^2 = 0.02) was found.

Follow-up. The 28-day mortality in the 34 septic patients was 26%. Two patients from the septic shock group died within 48 hours of admission, so no follow-up recording was possible. Median change in carotid-femoral PWV was –2.9 ± 4.6 m/s in survivors and 4.3 ± 7.3 m/s in non-survivors, and median change in carotid-radial PWV was –1.8 ± 1.4 m/s in survivors and 2.4 ± 4.3 in non-survivors. The change in carotid to femoral and carotid to radial PWV

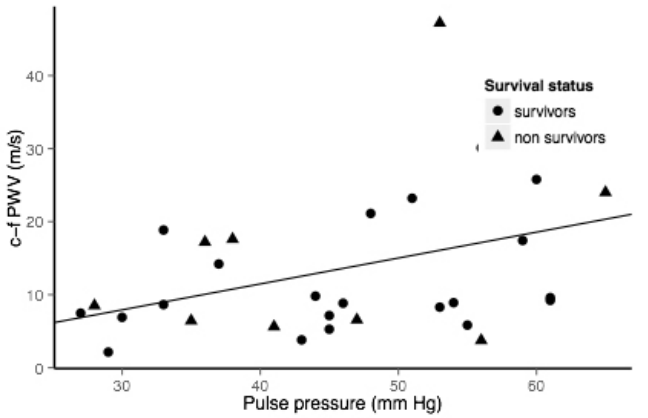

Fig. 2. Relationship between carotid to femoral pulse wave velocity and pulse pressure in patients with early sepsis (r = 0.35, p = 0.05).

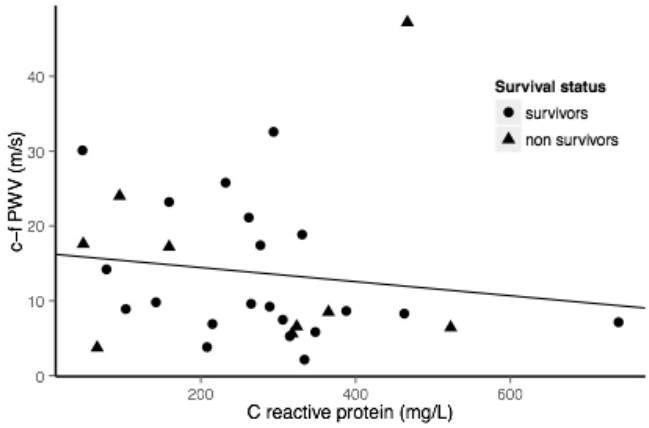

Fig. 3. Relationship between carotid to femoral pulse wave velocity and C-reactive protein concentration in patients with early sepsis (r = –0.43, p = 0.04).

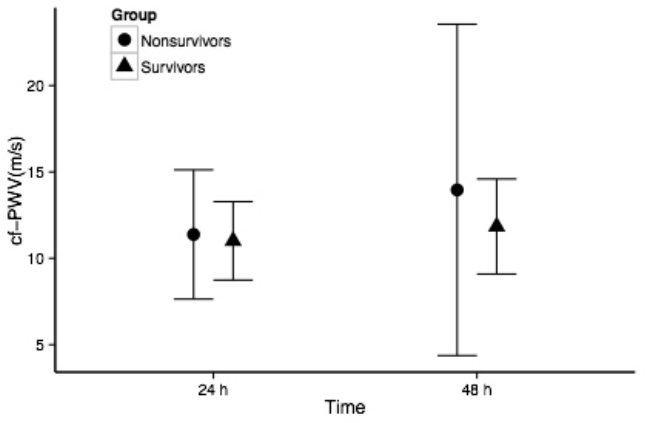

Fig. 4. Time course of carotid to femoral PWV (median ± SE) in survivors (n = 25; circles) and non-survivors (n = 9; triangles) at baseline (24 h), and after 48 h in 34 septic patients.

did not significantly different between survivor and non-survivor groups (p = 0.6) (Fig. 4).

DISCUSSION

This prospective, observational study characterises and quantifies sepsis-induced alterations in arterial stiffness in

elastic and muscular arteries. We demonstrated that PWV, a recognised index of arterial stiffness, is not modified in patients with severe sepsis compared with patients with septic shock. Likewise, PWV and its change over 48 hours of early sepsis was not related to patient outcome. Carotid to femoral and carotid to radial PWV was correlated with pulse pressure, and carotid to femoral PWV was negatively correlated with inflammatory status.

Although PWV is not a direct measure of arterial distensibility, several reports have considered it an important index of arterial stiffness and consequently, arterial distensibility. There are some cardiovascular risk factors that can modify arterial distensibility and PWV measurement differently. However, important factors like age and blood pressure interfere similarly with both arterial distensibility and PWV measurement.

In our cohort of patients with severe sepsis and septic shock, median carotid-to-femoral PWV was 11.2 m/s at baseline and 9.3 m/s at follow up (after 48 hours). The values are very similar to results of a large recent study establishing normal and reference values for carotid to femoral PWV based on a data set obtained from 13 centres distributed across Europe (Anonymous, 2010). No large population studies have been done examining reference values for carotid to radial PWV. However, as the carotid to radial PWV values do not significantly differ from carotid to femoral PWV values in this study, it can be assumed they are in the normal range for the general population. A subgroup of patients in this study had lower than normal blood pressure, and it is difficult to interpret PWV as no reference values currently exist.

Numerous publications and several reviews (Oliver and Webb, 2003; Zieman *et al*., 2005) reported various pathophysiological conditions associated with changed, mainly increased, arterial stiffness associated with structural artery wall changes. Most of them describe the effect of aging, but also (Boutouyrie and Vermeersch, 2010) include the following: physiological conditions, such as menopausal status (Tanaka *et al*., 1998), genetic background, such as family history of myocardial infarction (Riley *et al*.,1986) and genetic polymorphisms (Benetos *et al*., 1995); cardiovascular risk factors, such as smoking (Kool *et al*., 1993) and/or high C- reactive protein (CRP) level (Yasmin *et al*., 2004); and cardiovascular diseases, for example, coronary heart disease (Boutouyrie *et al*., 2002). Acute systemic inflammation, which characterises severe sepsis and septic shock, is unlikely to induce structural artery wall changes because of the short timeframe.

Another group of primarily non-cardiovascular diseases, such as rheumatoid arthritis (Klocke *et al*., 2003, Turesson *et al*., 2005), systemic vasculitides (Booth *et al*., 2004) and systemic lupus erythematosus (Selzer *et al*., 2001), have been described in studies as associated with changes in aortic rigidity, showing the role of inflammation in the stiffening of large arteries. The inflammation process, either acute during Salmonella typhi vaccination (Vlachopoulos *et al*., 2005) or chronic during rheumatoid arthritis or systemic lupus erythematosus, can cause stiffening through various mechanisms, including endothelial dysfunction, cell release of any number of inducible matrix metalloproteinases (including MMP-9), medial calcifications, modified proteoglycan composition and hydration state, and/or cell infiltration around the vasa vasorum leading to vessel ischemia (Roman *et al*., 2005, Maki-Petaja *et al*., 2006). The primary proinflammatory cytokines, TNFa, and interleukin-6, are the main inducers of hepatic CRP synthesis. In untreated patients with essential hypertension, aortic stiffness, assessed with carotid-to-femoral PWV, was significantly associated with CRP and interleukin-6 (Mahmud and Feely, 2005). In contrast to the previously published data, in this study high CRP concentrations were associated with lower carotid-to-femoral PWV. The studies describing association of CRP and PWV have enrolled patients with low grade inflammation, and in cases of a massive inflammatory response, the impact of CRP concentration on PWV could be different.

Because sepsis is a dynamic state and haemodynamic parameters can change quickly, functional determinants of PWV may play a significant role. Functional factors that can cause alterations in arterial distensibility include mean and pulse arterial pressure values and smooth muscle cell tone of the arterial wall, in relation to adrenergic activity. In our study, the correlation of PWV and pulse pressure was statistically significant in all sepsis patients, supporting the data indicating that the influence of blood pressure on vascular properties is not modified in septic patients. There was no clear effect of added adrenergic activity on PWV measurement of patients requiring vasopressors and those without vasopressor support.

This study was the first to assess longitudinal changes in arterial stiffness in a cohort of patients with severe sepsis and septic shock and examine the influence of haemodynamic parameters and inflammation on arterial stiffness. However, it has some limitations. The patient population was heterogeneous concerning disease severity and treatment and the sample size was relatively small. There are no reliable reference values for PWV when measured at lower than normal blood pressures. Functional factors have marked influence on PWV, which could make the reproducibility of isolated measurements questionable, raising the importance of longitudinal assessment. Possible confounders that were not measured include different levels of cardiovascular risk factors. The measurement of endothelial function could have helped to explain changes in arterial stiffness in relation to C-reactive protein concentration.

This study does not support the role of arterial stiffness in early sepsis as a potential prognostic factor. The new finding, which has not been described in previous literature, was the negative correlation between PWV and C-reactive protein concentration under conditions of massive inflammatory response.

REFERENCES

Anonymous (2010). The reference values for arterial stiffness' collaboration. Determinants of pulse wave velocity in healthy people and in the presence of cardiovascular risk factors: "Establishing normal and reference values". *Eur. Heart J*., **31** (19), 2338–2350.

Benetos, A., Gautier, S., Ricard, S., Topouchian, J., Asmar, R., Poirier, O., Cambien, F. (1996). Influence of angiotensin-converting enzyme and angiotensin II Type 1 receptor gene polymorphisms on aortic stiffness in normotensive and hypertensive patients. *Circulation*, **94** (4), 698–703.

Bone, R. C. (1991). The pathogenesis of sepsis. *Ann. Intern. Med*., **115**, 457–469.

Blacher, J., Asmar, R., Djane, S., London, G. M., Safar, M. E. (1999). Aortic pulse wave velocity as a marker of cardiovascular risk in hypertensive patients. *Hypertension*, **33** (5), 1111–1117.

Blacher, J., Guerin, A. P., Pannier, B., Marchais, S. J., Safar, M. E., London, G. M. (1999). Impact of aortic stiffness on survival in end-stage renal disease. *Circulation*, **99** (18), 2434–2439.

Booth, A. D., Wallace, S., McEniery, C. M., Yasmin, Brown, J., Jayne, D. R. W., Wilkinson, I. B. (2004). Inflammation and arterial stiffness in systemic vasculitis: A model of vascular inflammation. *Arthritis Rheum*., **50** (2), 581–588.

Boutouyrie, P., Tropeano, A. I., Asmar, R., Gautier, I., Benetos, A., Lacolley, P., Laurent, S. (2002). Aortic stiffness is an independent predictor of primary coronary events in hypertensive patients: A longitudinal study. *Hypertension*, **39** (1), 10–15.

Cohen, J. (2002). The immunopathogenesis of sepsis. *Nature*, **420** (6917), 885–891.

Erts, R., Spigulis, J., Kukulis, I., Ozols, M. (2005). Bilateral photoplethysmography studies of the leg arterial stenosis. *Physiol. Meas*., **26**, 865–874

Huybrechts, S. A. M., Devos, D. G., Vermeersch, S. J., Mahieu, D., Achten, E., de Backer, T. L. M., Segers, P., van Bortel, L. M. (2011). Carotid to femoral pulse wave velocity: A comparison of real travelled aortic path lengths determined by MRI and superficial measurements. *J. Hypertens*., **29** (8). http://journals.lww.com/jhypertension/Fulltext/2011/08000/Carotid_to_femoral_pulse_wave_velocity___a.16.aspx

Kool, M. J., Hoeks, A. P., Struijker-Boudier, H. A., Reneman, R. S., Van Bortel, L. M. (1993). Short- and long-term effects of smoking on arterial wall properties in habitual smokers. *J. Amer. Coll. Cardiol*., **22**, 1881–1886.

Klocke, R., Cockcroft, J. R., Taylor, G. J., Hall, I. R., Blake, D. R. (2003). Arterial stiffness and central blood pressure, as determined by pulse wave analysis, in rheumatoid arthritis. *Ann. Rheum. Dis*., **62** (5), 414–418.

Knaus, W. A., Draper, E. A., Wagner, D. P., Zimmerman, J. E. (1985). APACHE II: A severity of disease classification system. *Crit. Care Med*., **13**, 818–829.

Levy, M. M., Fink, M. P., Marshall, J. C., Abraham, E., Angus, D., Cook, D., Cohen, J., Opal, S. M., Vincent, J. L., Ramsay, G. (2003) 2001 SCCM/ESICM/ACCP/ATS/SIS International Sepsis Definitions Conference. *Int. Care Med*., **29**, 530–538

Mahmud, A., Feely, J. (2005). Arterial stiffness is related to systemic inflammation in essential hypertension. *Hypertension*, **46** (5), 1118–1122.

Mäki-Petäjä, K. M., Hall, F. C., Booth, A. D., Wallace, S. M. L., Yasmin, Bearcroft, P. W. P., Wilkinson, I. B. (2006). Rheumatoid arthritis is associated with increased aortic pulse-wave velocity, which is reduced by anti-tumor necrosis factor-α therapy. *Circulation*, **114** (11), 1185–1192.

Nyström, P. O. (1998). The systemic inflammatory response syndrome: Definitions and aetiology. *J. Antimicrob. Chemother*., **41** (suppl 1), 1–7.

Oliver, J. J., Webb, D. J. (2003). Noninvasive assessment of arterial stiffness and risk of atherosclerotic events. *Arterioscler. Thromb. Vasc. Biol*., **23** (4), 554–566.

Riley, W. A., Freedman, D. S., Higgs, N. A., Barnes, R. W., Zinkgraf, S. A., Berenson, G. S. (1986). Decreased arterial elasticity associated with cardiovascular disease risk factors in the young. Bogalusa Heart Study. *Arterioscler. Thromb. Vasc. Biol*., **6** (4), 378–386.

Roman, M. J., Devereux, R. B., Schwartz, J. E., Lockshin, M. D., Paget, S. A., Davis, A., Salmon, J. E. (2005). Arterial stiffness in chronic inflammatory diseases. *Hypertension*, **46** (1), 194–199.

Stefanadis, C., Dernellis, J., Tsiamis, E., Stratos, C., Diamantopoulos, L., Michaelides, A., Toutouzas, P. (2000). Aortic stiffness as a risk factor for recurrent acute coronary events in patients with ischaemic heart disease. *Eur. Heart J*., **21** (5), 390–396.

Selzer, F., Sutton-Tyrrell, K., Fitzgerald, S., Tracy, R., Kuller, L., Manzi, S. (2001). Vascular stiffness in women with systemic lupus erythematosus. *Hypertension*, **37** (4), 1075–1082.

Tanaka, H., DeSouza, C. A., Seals, D. R. (1998). Absence of age-related increase in central arterial stiffness in physically active women. *Arterioscler. Thromb. Vasc. Biol*., **18** (1), 127–132.

Turesson, C., Jacobsson, L., Rydén Ahlgren, Å., Sturfelt, G., Wollmer, P., Länne, T. (2005). Increased stiffness of the abdominal aorta in women with rheumatoid arthritis. *Rheumatology*, **44** (7), 896–901.

Vincent, J. L., Moreno, R., Takala, J., Willatts, S., Mendonēa, A. de, Bruining, H., Reinhart, C. K., Suter, P. M., Thijs, L. G. (1996). The SOFA (Sepsis-related Organ Failure Assessment) score to describe organ dysfunction/failure. *Int. Care Med*., **22** (7), 707–710.

Vlachopoulos, C., Dima, I., Aznaouridis, K., Vasiliadou, C., Ioakeimidis, N., Aggeli, C., Stefanadis, C. (2005). Acute systemic inflammation increases arterial stiffness and decreases wave reflections in healthy individuals. *Circulation*, **112** (14), 2193–2200.

Yasmin, McEniery, C. M., Wallace, S., Mackenzie, I. S., Cockcroft, J. R., Wilkinson, I. B. (2004). C-Reactive protein is associated with arterial stiffness in apparently healthy individuals. *Arterioscler. Thromb. Vasc. Biol*., **24** (5), 969–974.

Zieman, S. J., Melenovsky, V., Kass, D. A. (2005). Mechanisms, pathophysiology, and therapy of arterial stiffness. *Arterioscler. Thromb. Vasc. Biol*., **25** (5), 932–943.

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ARTĒRIJU CIETĪBA UN PULSA VIĻŅA IZPLATĪŠANĀS ĀTRUMS PACIENTIEM AR AGRĪNU SEPSI

Sepse izraisa masīvu iekaisuma reakciju, kas var ietekmēt asinsvadu funkciju. Šī pētījuma mērķis ir noteikt pulsa viļņa izplatīšanās ātrumu (PVIĀ), artēriju cietības objektīvu rādītāju, un tā dinamiku pacientiem ar agrīnu smagu sepsi un septisku šoku un salīdzināt artēriju elastīgās īpašības ar sepses iznākumu. Pētījumā tika iekļauti 12 pacienti ar smagu sepsi un 22 pacienti ar septisku šoku. PVIĀ tika noteikts miega artērijas-femorālajā un miega artērijas-brahiālajā baseinā pirmajās 24 stundās pēc stacionēšanas intensīvās terapijas nodaļā un atkārtoti pēc 48 stundām. PVIĀ pacientiem ar smagu sepsi un septisku šoku neatšķīrās ne miega artērijas-femorālajā (11.7 ± 2.2 vs. 11.3 ± 3.6 m/s), ne miega artērijas-brahiālajā baseinā (12.0 ± 3.8 vs. 9.5 ± 2.2 m/s). Atkārtotā mērījumā pēc 48 stundām PVIĀ bija līdzīgs 25 izdzīvojušo un 9 neizdzīvojušo pacientu grupās. Pozitīvu korelāciju novēroja starp PVIĀ un pulsa spiedienu visās pacientu grupās ($r = 0.35$, $p = 0.05$). Zemāks PVIĀ korelēja ar augstāku C reaktīvā proteīna koncentrāciju ($r = -0.43$, $p = 0.04$). Pacientiem ar agrīnu sepsi PVIĀ ietekmē hemodinamiskie (pulsa spiediens) un iekaisuma (C reaktīvais olbaltums) faktori, bet ne slimības smagums vai prognoze.