ACCEPTED MANUSCRIPT

Accepted manuscripts are the articles in press that have been peer reviewed and accepted for publication by the Editorial Board of the Vojnosanitetski Pregled. They have not yet been copy edited and/or formatted in the publication house style, and the text could still be changed before final publication.

Although accepted manuscripts do not yet have all bibliographic details available, they can already be cited using the year of online publication and the DOI, as follows: article title, the author(s), publication (year), the DOI.

Please cite this article: ARTERIAL STIFFNESS AS PREDICTIVE FACTOR OF CARDIOVASCULAR DISEASES

ARTERIJSKA KRUTOST KAO PREDIKTIVNI FAKTOR KARDIOVASKULARNIH OBOLJENJA

Authors: Stojanov Vesna*, Radivojević Nenad†; Vojnosanitetski pregled (2017); Online First November, 2017.

UDC:

DOI: https://doi.org/10.2298/VSP170701161S

When the final article is assigned to volumes/issues of the Journal, the Article in Press version will be removed and the final version appear in the associated published volumes/issues of the Journal. The date the article was made available online first will be carried over.
ARTERIAL STIFFNESS AS PREDICTIVE FACTOR OF CARDIOVASCULAR DISEASES

Arterijska krutost kao prediktivni faktor kardiovaskularnih oboljenja

Stojanov Vesna*, Radivojević Nenad†

*Multidisciplinary Center for Polyclinic Diagnostics, Assessment and Treatment of Blood Pressure Disorders, Clinic for Cardiology, Clinical Center of Serbia; Faculty of Medicine, University of Belgrade, Belgrade, Serbia
†Multidisciplinary Center for Polyclinic Diagnostics, Assessment and Treatment of Blood Pressure Disorders, Clinic for Cardiology, Clinical Center of Serbia

Corresponding author:
Prof. dr Vesna Stojanov
Multidisciplinarni centar za polikliničku dijagnostiku, praćenje i lečenje poremećaja krvnog pritiska
Klinika za kardiologiju
Klinički centar Srbije
Pasterova 2
11000 Beograd
Tel: 011 366 3402
Email: stojanovves@eunet.rs

Kratak naslov rada: Arterial stiffness in cardiovascular diseases
Introduction

Understanding the basic hemodynamic principles is necessary for the assessment of arterial stiffness and its possible clinical practice. Hydraulic and elasticity theory was established by Young in 1840, Moens in 1878, and Korteweg in 1878 (1). The main determinants of PWV relates the velocity of pulse wave travel in a vessel to the distenbility of that vessel (2,3):

\[ \text{PWV} = \sqrt{\frac{Eh}{2R\rho}} \]

(E is Young’s modulus in the circumferential direction, h is the wall thickness, R is the vessel radius and \( \rho \) is the density of fluid).

Later, the doctors-physiologists, Marey in 1860, Mahomed in 1872 and Mackenzia in 1902, developed various types of Sphygmocor and, thus, made significant progress in pressure waveform analysis (4). Clinical application was discovered by Safar (5) and O’Rourke (6), which turned out to be very useful in the prognosis of the outcome and the correction of therapy.

In healthy persons, peripheral arteries are stiffer than the central and that leads to an increase of amplitude of pulse wave in blood vessels, from heart to periphery, which is known as pressure amplification (7). The stiffness of mid-size arteries is assessed by vasomotor tone which depends on the endothelial function or the sympathetic nervous system (8,9,10) or on renin-angiotensin system (11).

Elastic properties of arteries vary along the arterial tree, with more elastic proximal and stiffer distal arteries. In humans, pulse wave velocity (PWV) increases from 4-5 m/s in ascending aorta to 5-6 m/s in abdominal aorta, and 8-9 m/s in iliac and femoral arteries (11). This difference in arterial stiffness has significant physiological and pathophysiological consequences.

Due to increase of pulse pressure between central and peripheral arteries, brachial pulse pressure should not be used as a replacement for aortic or carotid pulse pressure especially in younger persons.

Stiffness of common carotid artery is approximately six times higher in 70-year old persons than in a 20-year old person (6,12). In older patients with hypertension or diabetes, carotid arteries may become stiffer than femoral or radial arteries which become slightly stiffer with age or due to hypertension.
Non-invasive assessment of arterial stiffness
Regional and local arterial stiffness may be measured directly in a non-invasive manner, in various locations along the arterial tree and it is based on measuring of parameters which are directly connected to arterial stiffness.

Local assessment of arterial stiffness
Arterial stiffness can be assessed locally through the use of ultrasound device which also provides precise measures of intima-media thickness (IMT) which allows the assessment of elastic properties of arterial vessel according to the Young’s elastic modulus (13). The assessment of carotid stiffness and thickness is important for the development of atherosclerosis. Local arterial stiffness of deep arteries such as aorta may also be measured with magnetic resonance imaging (MRI), but this is not commonly applied in routine practice. Aortic pulse wave velocity assessed by transformation of the brachial pressure waveform didn’t show significant different comparing with the CMR-derived transit time method (14).

Local measurement of arterial stiffness is important in pathophysiological, pharmacological and therapy studies.
Although the carotid-femoral PWV and carotid stiffness provide similar data on how age affects stiffness of big arteries in healthy persons, this is not the case with persons with hypertension and/or diabetes. With age and other cardiovascular risk factors, aorta gets stiffer than carotid arteries and that is why aortic and carotid stiffness cannot be used as variable predictors of high risk patients (15).

Regional assessment of arterial stiffness
Aorta is the main blood vessel of interest in the assessment of regional arterial stiffness, but all of the arterial branches have their own impact. Arteria brachialis, where the pressure is most often measured, and lower extremity arteries get especially altered by atherosclerosis.
Measurement of local carotid stiffness may also provide significant prognostic information since the carotid artery is often the place where atheroma appears.

**PWV measuring**

Pulse wave velocity measurement is the simplest, non-invasive method for the assessment of arterial stiffness. Carotid-femoral PWV is a direct measurement and is considered a golden standard for the assessment of arterial stiffness, but it is important to precisely measure distance between carotid and femoral artery, because even small mistakes may affect the absolute value of PWV (16,47,49). Measuring along aorta or aortoiliac path is clinically the most relevant, since aorta and its first branches are connected to the left ventricle and therefore are the most responsible for most of the arterial stiffness effects. Contrary to that, PWV measured outside of aortic system, that is, in upper extremities (brachial PWV) or lower extremities (femorotibial PWV) do not have good predictive value in patients with terminal kidney disease (17).

It is difficult to register femoral pressure waveform in persons with metabolic syndrome, excess body mass, diabetes and peripheral arterial disease (18). In case of aortic, iliac or stenosis of proximal segment of femoral artery, pressure waveform may be delayed or reduced. Abdominal obesity, especially in men, and large breasts in women may cause inaccurate measuring of distance (19).

**Arterial stiffness measuring devices**

SphygmoCor® is a tonometric device; PWV is calculated on the basis of successive waves produced in short time interval in two arterial locations (most often carotid and femoral artery) through use of R waves in the electrocardiogram (ECG) for the calculation of the delay (20).

Measurements are most often done at the root of the left subclavian artery (suprasternal space on the skin) or close to abdominal aortic bifurcation (at the level of umbilicus). Transit time is automatically calculated by the recognition of the beginning of pulse. This method is used for the assessment of predictive value of aortic PWV for cardiovascular events in patients with diabetes and it gives more precise assessment of aortic PWV when compared to carotid-femoral PWV. Aortic brachial PWV is an important predictor of cardiovascular events in patients with hypertension (21).
TensioMed Arteriograph® is a device for measuring stiffness of arterial blood vessels that works according to patented oscillometric principle. Data received via Arteriograph (augmentation index (Aix), PWV, central systolic blood pressure (SBP) and pulse pressure (PP)) match the data received via brachial artery catheter. Comparison was also made with Applanation Tonometer Sphygmocor® and no significant difference was observed. For the assessment of arterial stiffness parameters with these devices, it is necessary to prepare the patient adequately: the use of alcohol 10 hours before and the use coffee 3 hours before the measurement are strictly forbidden; patients must be in a semi-supine position 10 minutes before the measurement. The assessment is performed in supine position at the room temperature of 22±1°C. Arteriograph device measures aortic pulse wave velocity (PWVao), augmentation index (Aix) and central blood pressure (SBPao) values simultaneously with the peripheral blood pressure. By inflating the cuff on upper-arm to suprasystolic pressure the brachial artery becomes occluded. The brachial flow is stopped, therefore the brachial wall characteristics are excluded (no significant wall movement), consequently the gained information relate to the systematic circulation. For calculating arterial function parameters the recorded pulse waveform is analyzed and the characteristic points of the first and reflected waves are determined. The true aortic length is estimated with the jugulum-symphysis distance (Jug-Sy).

Optimal values: Aix < -30%, PWV <7 m/s. Pathological values: Aix >10%, PWV>12 m/s.

Besides the classic 24-hour blood pressure measurement, the latest device with HMS CS program combined with Mobil-O-Graph® also has an integrated system for 24-hour ambulatory monitoring of arterial stiffness through the use of oscillometric method. Mobil-O-Graph measures standard blood pressure parameters, central aortic pressure, central pulse pressure (PP), augmentation index standardized to a heart rate of 75 beats through empirical regression (AIX@75), pulse wave velocity (PWV), cardiac output, cardiac index, and total vascular resistance reflection magnitude. Bioelectrical impedance gives us these values at present time (22,23). 24-hour monitoring of both arterial stiffness and hemodynamic enables the provision of more precise parameters in cardiovascular prediction. There is no specific preparation of patients for wearing this device and one advantage is that all parameters are monitored 24 hours during usual daytime and nighttime
values. In addition, therapeutic effect of medicine can be assessed much better, with a much better prognostic effect on damage to target organs.

**Figure 1.** Mobil-O-Graph® parameters

There is no difference in the value of central aortic pressure measured through oscillometric non-invasive method with the Mobil-O-Graph® device and tonometric method with the SphygmoCor® device. Mobil-O-Graph® combines the advantages of assessing brachial pressure and central blood pressure in one measurement (18). As with other populations, acceptability of Mobil-O-Graph® and SphygmoCor® is evident for central SBP and AIX@75 in dialysis patients; PWV is slightly underestimated by Mobil-O-Graph (20). Non-invasive 24-hour assessment of blood pressure level and blood vessel stiffness is of prognostic value for cardiovascular risk (24). According to the SAFER study, left ventricular hypertrophy (LVH) is more associated with 24-hour aortic pressure than with 24-hour brachial pressure in patients with hypertension (25). Aortic pressure correlates better with all, especially cardiovascular mortality caused by brachial artery pressure (26).

**Non-invasive assessment of arterial stiffness parameters**

*Central pulse wave analysis*

Arterial pressure waveform consists of the pulse wave initiated by ventricular contraction and reflected wave. The waves are reflected from periphery, mostly from the branching point. In elastic blood vessels, due to low PWV, reflected wave has a tendency to return to aortic root during diastole. In case of stiff arteries, PWV increases and reflected wave arrives to central artery earlier, where it acts as an enhancement to the initiated wave and increases systolic pressure. This phenomenon may be quantified through Augmentation Index (AIx) – which is defined as a difference between the second and first systolic peak (P2-P1) expressed as a percentage of the pulse pressure (Figure 1) (4,16). Regardless of high PWV, changes in the location of reflection may affect AIx. In clinical research, the main AIx determinants are not only diastolic blood pressure and height, but also age and aortic PWV.
Central AIx and central pulse pressure have shown an independent predictive value when it comes to all-cause mortality in dialysis patients (26) and cardiovascular events in patients who underwent PCI, as well as in patients with hypertension in the CAFE study (27).

**Figure 2.** Augmentation Index (AIx) – which is defined as difference between the second and first systolic peak (P2-P1) expressed as a percentage of the pulse pressure

*Central and peripheral systolic and pulse pressure*

Peripheral pressure SBP and pulse pressure (measured on brachial artery) should not be equated with central SBP and PP measured on carotids, since the peripheral pressure is higher than the central and PP, especially in younger persons, due to less stiff central artery in younger persons.

*Central pulse pressure, AIx and arterial stiffness*

Central SBP and PP, AIx and PWV increase with age, hypertension, diabetes mellitus and hypercholesterolemia, and they are connected with damage to target organs (left ventricular hypertrophy, microalbuminuria, carotid plaques, and endothelial dysfunction). Central SBP, PP and AIx depend on the speed at which the wave travels, amplitude of the reflected wave, point of reflection, duration and size of ventricular ejection, especially in relation to heart rate and ventricular contractility (28), while aortic PWV which is the wave propagation speed, presents internal arterial stiffness according to Bramwell-Hill equation (Figure 2). Pathophysiological conditions and medicines can change central pulse pressure and AIx without changing aortic PWV, which shows the dominant influence of the reflecting wave, heart rate or ventricular ejection, without the change of arterial stiffness (29,30,31). AIx is much more sensitive to heart rate than PWV. In the Anglo-Cardiff Collaborative Trial conducted in the general population it is shown that younger than age affects AIx more than it affects PWV 50 in people below 50 years of age, but it is the opposite after the age of 50 years (32).

Central pressure and AIx have a significant predictive value in patients with hypertension, coronary disease and kidney diseases (33).
Clinical application

Pathophysiology

Stiffness of vascular wall depends on two main fiber proteins which are part of its composition: collagen and elastin. Excessive production of abnormal collagen and reduction of normal elastin plus their inadequate spatial organization lead to arterial stiffness (34).

There are many risk factors for increased arterial stiffness: age, low birth weight, menstrual cycle, menopause, lack of physical activity, genetic predisposition to hypertension, diabetes, myocardial infarction, genetic polymorphism, obesity, smoking, hypercholesterolemia (35), glucose intolerance, metabolic syndrome, hyperhomocysteinemia, high levels of C-reactive protein (CRP). Also, cardiovascular diseases, such as coronary disease, congestive heart failure (2), stroke, as well as non-cardiovascular diseases, such as moderate chronic kidney disease stage 3 (36), rheumatoid arthritis, systemic vasculitis and systemic lupus erythematosus (28).

Arterial stiffness and reflection of waves are important for increase of systolic pressure in elderly persons, and they play important role in the occurrence of cerebrovascular stroke and myocardial infarction.

Arterial stiffness causes premature return of reflected wave in early systole, which increases central pulse pressure and then systolic blood pressure which increases left ventricular burden and increases the need for oxygen. In addition, arterial stiffness is combined with left ventricular hypertrophy (25), which is a significant risk factor for coronary disease in normotensive and hypertensive patients. Increase of central PP and decrease of diastolic BP can directly cause subendocardial ischemia. Increase of aortic stiffness, together with aging and risk factors for cardiovascular diseases, is caused by various mechanisms including degradation of elastic fibers, collagen accumulation, fibrosis, inflammation, medial smooth muscle cell necrosis, calcification and diffusion of macromolecules across the arterial wall (27). Increased arterial stiffness may increase the risk of stroke through several mechanisms, including the increase of central PP, influence on remodeling of intra and extra cranial arteries, increase of carotid wall thickness, and...
development of stenosis and plaques (27,28,37), with plaque rupture and damage to brain’s white matter. In addition, coronary disease and heart weakness with high PP and arterial stiffness are risk factors for the occurrence of stroke.

*Routine application of arterial stiffness*

Aortic stiffness has an independent and more significant predictive value than classical risk factors for all-cause and cardiovascular mortality in patients with hypertension, type 2 diabetes, patients on dialysis, elderly persons, since it shows damage to blood vessel through risk factors during longer time period (20).

Aortic PWV has a better predictive value than classical cardiovascular risk factors. Central AIx and pulse pressure have shown independent predictive value for all-cause mortality in patients on dialysis or after kidney transplantation, and for cardiovascular events in persons with hypertension and coronary disease, after percutaneous coronary intervention (38). It was shown in meta-analysis that aortic stiffness expressed as aortic PWV is a strong predictor of future CV events and all-cause mortality. The predictive ability of arterial stiffness is higher in subjects with a higher baseline CV risk (39,40). Also, aortic PWV may enable better identification of high-risk populations that might benefit from more aggressive CVD risk factor management (41).

*Predictive value of pulse wave velocity for cardiovascular event reduction*

An important question is whether the reduction of PWV is associated with the accompanying reduction of cardiovascular events, regardless of the normalization of classical risk factors?

The reduction of arterial stiffness may indicate an actual reduction of damage to blood vessel wall, while blood pressure, glycaemia and lipids may be normalized in several weeks through medical therapy resulting in significant reduction of cardiovascular risk score, but without improvement of atherosclerotic lesion and arterial stiffness which require long-term correction of biochemical parameters. Therefore, a temporary dissociation between reduction of cardiovascular risk factor and further present arterial stiffness is to be expected.

It is still to be proven whether reduction of central PP is associated with the accompanying reduction of cardiovascular events, regardless of normalization of classical risk factors. There is indirect evidence of this. In the REASON study (36,42), only the combination of
perindopril/indapamide managed to significantly reduce reflection of carotid wave with the resulting relative reduction of central SBP and PP and subsequent reduction of LVH (42) as opposed to no reduction of carotid PP and LVH in patients who used atenolol in their therapy. CAFE study (38) and ASCOT study (43) have shown that central pressure, augmentation index and pulse pressure were independent predictors of cardiovascular events in hypertensive patients and that reduction of central SBP and PP was higher in the amlodipine/perindopril group than in the atenolol/thiazide group, despite of similar reduction of SBP and PP of brachial artery.

How to reduce arterial stiffness (arterial stiffness therapy)

Non-pharmacological and pharmacological therapies have an important place in the arterial stiffness reduction.

Non-pharmacological therapy includes a regular moderate physical activity, weight loss, reduced salt intake, moderate consumption of alcohol, garlic powder, alpha linoleic acid and fish oil, as well as hormone replacement therapy (44,45,46).

Pharmacological therapy includes the application of antihypertensive medicines and medicines for treatment of cardiac insufficiency: diuretics, beta blockers, angiotensin-converting-enzyme (ACE) inhibitors, type I angiotensin-receptor blockers (AT1 blockers) and calcium antagonists, nitrates, hypolipidemic medicines such as statins and fibrates, antidiabetics such as thiazolidinediones, as well as sildenafil (47,48).

The majority of antihypertensive drugs have the main influence on the dynamic component of arterial stiffness and in some part on the structural component in arterial wall remodeling (49).

In general, the renin-angiotensin system inhibitors are superior to all other antihypertensive drugs in reducing arterial stiffness. One reason is the profibrotic action of the renin-angiotensin system, as the turnover of the extracellular matrix in the arterial wall per se leads to a change in the properties of the vessel (50,51).

REASON study showed positive effects of ACE inhibitors on arterial stiffness, especially on AIX. The effects lasted even after nine months of treatment (52,53). Positive effects have been shown for most drugs in this group, including for lisinopril (54).

If reduction of AIX is in focus, losartan in LIFE (55), OPTIMAAL (56) study and candesartan (I57, 58) showed positive effect on reduction. Some others ARBs valsartan
(VALUE study) (59,60) and telmisartan (61) reduce Aix and PWV but increase pulse pressure.

**Figure 4.** Case from our practice (before therapy with losartan)

**Figure 5.** Case from our practice (six months on losartan therapy)

Beta-blockers without vasodilating effects have a weaker effect on arterial stiffness and central pulsatile hemodynamics than vasodilating drugs of other antihypertensive groups. The mechanism of action is through heart rate reduction, as this influences the viscoelastic properties of the arterial wall. Reduced heart rate also leads to increased wave reflections, a lower reduction in aortic than brachial systolic blood pressure, and reduced pulse pressure amplification. Peripheral vasoconstriction, achieved by, atenolol, is an additional mechanism responsible for the negative effect on wave reflections (62,63). New agents such as nebivolol and which have vasodilating effects seem to be more effective in improving central pulsatility. These effects appear to be related to their ability to donate NO, which dilates the small resistance arteries. The effects observed lead to pulse pressure amplification, but Aix reduction (64,62,63).

Calcium channel blockers also lower PWV and reduce wave reflections, but to a lesser degree than renin-angiotensin inhibitors. The largest amount of evidence is for amlodipine. In the CAFE study (65), was showed to reduce central blood pressure more than peripheral blood pressure; it amplified pulse pressure and reduced Aix.

Diuretics seem to have no beneficial effect on pulsatile hemodynamics. Hydrochlorothiazide showed a neutral effect on reduction of central blood pressure and a neutral effect on pulse pressure amplification (66, 67).

**Arterial stiffness and damage to target organs**
Arterial stiffness also provides data on the damage of target organs, which is of great importance for assessment of total cardiovascular risk in patients with hypertension.
In case of primary coronary event in hypertensive patients, the value of aortic PWV is much more important for low-risk patients (first or second tertile of the Framingham risk score) than for high-risk patients (third or fourth tertile), which shows that the population with low to medium risk benefits the most from the value of PWV (28).

The latest results show that increased aortic stiffness is an independent predictor of major adverse cardiac and cerebrovascular events (MACCE) after acute ST-elevation myocardial infarction. The assessment of aortic stiffness in addition to classical risk factors significantly improved early risk stratification (68).

Concerning diastolic dysfunction, arterial stiffness was correlated with elevated left ventricular filling pressure (LVFP) and it was shown that increased level was associated with an elevated LVFP in patients with preserved systolic function. It was hypothesized that increased arterial stiffness is of pathophysiological relevance for diastolic dysfunction (69).

**Conclusion**

The existing European (70) and American (71) recommendations for the diagnosis and treatment of hypertension define LVH and albuminuria as evidence of damage of target organ, as well as arterial stiffness and reflection of waves. Assessment of arterial stiffness and central pressure should be considered as a recommended test for assessment of cardiovascular risk, especially in patients with damage to target organs that went undetected during routine examinations. According to the recommendations of the American Society of Hypertension (ASH), the assessment of blood vessels’ stiffness should be performed by hypertension specialists in hypertension centers. Introduction of this diagnostic procedure in routine practice is still being considered (71).

According to the European Society of Hypertension (ESH) guidelines of 2013, stiffness of large arteries and wave reflection phenomenon are important pathophysiological indicators of isolated systolic hypertension, and, together with pulse pressure, they increase with age (72,73). Carotid-femoral PWV is a golden standard for measuring aortic stiffness (73). According to the common guidelines issued by the European Society of Hypertension and European Society of Cardiology (ESC) (74), PWV value of >12m/s indicates significant deterioration of aortic function in middle-aged hypertension patients. According to recent
research, due to use of correct carotid-femoral distance and taking into consideration 20% anatomically shorter pulse wave distance, PWV value of >10m/s indicates increase of aortic stiffness (75). Central pressure, especially central systolic pressure and pulse pressure are more important predictors of occurrence of cardiovascular events and assessment of cardiovascular risk from brachial pressure (74). For valid measuring of central pressure, you can use not only tonometric devices (SphygmoCorCP®), but also oscillometric ones (SphygmoCor XCEL®) (76), as well as 24-hour arterial stiffness monitoring device, Mobil-O-Graph® (18).

REFERENCES


Figure 1. Mobil-O-Graph parameters
Figure 2. Augmentation Index (AIx) – which is defined as difference between the second and first systolic peak (P2-P1) expressed as a percentage of the pulse pressure.
Figure 3. The foot to foot method for measurement of carotid-femoral Pulse Wave Velocity
Figure 4. Case from our practice (before therapy with losartan)

<table>
<thead>
<tr>
<th>Measurement data</th>
<th>29/03/2017 07:48</th>
<th>Height: 159cm</th>
<th>Arm circ.: 28cm</th>
<th>Right</th>
</tr>
</thead>
<tbody>
<tr>
<td>Operator: ARTERIOGRAM</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Suprasystolic record**

<table>
<thead>
<tr>
<th>RT</th>
<th>S36</th>
<th>ED</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Brachial Blood Pressure and Pulse Wave Analysis**

<table>
<thead>
<tr>
<th>Sys: 165 mmHg</th>
<th>Dia: 93 mmHg</th>
<th>PP: 72 mmHg</th>
<th>MAP: 117 mmHg</th>
<th>HR: 76 /min</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBPao: 173.3 mmHg</td>
<td>PPao: 80.3 mmHg</td>
<td>Aix aortic: 46.2 %</td>
<td>Aix aortic (75): 46.8 %</td>
<td></td>
</tr>
</tbody>
</table>

**Lower limb circulation**

<table>
<thead>
<tr>
<th>ABI:</th>
<th>ED: 310 ms</th>
</tr>
</thead>
</table>

Figure 5. Case from our practice (six months on losartan therapy)

<table>
<thead>
<tr>
<th>Measurement data</th>
<th>22/09/2017 12:29</th>
<th>Height: 159cm</th>
<th>Arm circ.: 28cm</th>
<th>Right</th>
</tr>
</thead>
<tbody>
<tr>
<td>Operator: ARTERIOGRAM</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Suprasystolic record**

<table>
<thead>
<tr>
<th>RT</th>
<th>S36</th>
<th>ED</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Brachial Blood Pressure and Pulse Wave Analysis**

<table>
<thead>
<tr>
<th>Sys: 152 mmHg</th>
<th>Dia: 83 mmHg</th>
<th>PP: 69 mmHg</th>
<th>MAP: 106 mmHg</th>
<th>HR: 80 /min</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBPao: 149.4 mmHg</td>
<td>PPao: 66.4 mmHg</td>
<td>Aix aortic: 30.8 %</td>
<td>Aix aortic (75): 33.6 %</td>
<td></td>
</tr>
</tbody>
</table>

**Lower limb circulation**

<table>
<thead>
<tr>
<th>ABI:</th>
<th>ED: 280 ms</th>
</tr>
</thead>
</table>