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Lower Limb Revascularization and Pulse Wave Velocity of elastic and muscular arteries in Peripheral Arterial Disease

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1. Abstract

Background:

Peripheral arterial disease (PAD) may interfere with the assessment of pulse wave velocity (PWV) due to atherosclerotic lesions along the arterial tree.

Aim: To evaluate the effect of percutaneous transluminal angioplasty (PTA) of the lower limb on pulse wave velocity in patients with PAD.

Methods:

Pulse wave velocity (PWV) was measured prior and after lower limb angioplasty in patients with PAD. Aortic PWV was assessed with the Mobil-O-Graph (ABPM by IEM; Stolberg, Germany) derived from the brachial artery (aPWV) using a brachial cuff-based method. In addition, PWV was assessed with an oscillometric technique (Vicorder system SMT Medical, Würzburg, Germany) for the carotid-femoral (cfPWV) and femoro-tibial (ftPWV) segments in both limbs. Periinterventional changes were analysed by the Wilcoxon signed rank test.

Results:

Overall 58 patients were treated with angioplasty for Rutherford stage 2-5 with bilateral treatment in four patients. Treated limbs (n=62) were compared with control limbs (n=54). In the PTA-limbs, the ftPWV and the ankle-brachial arterial pressure index improved both from 7.6±2.5m/s to 9.2±2.3m/s ($p<0.0001$) and from 0.67±0.32 to 0.86±0.28 ($p<0.0001$), respectively. In the untreated limbs, ftPWV remained unchanged (9.1±2.7 m/sec and 9.2±2.6 m/sec, $p=0.334$). No significant changes were observed for the cfPWV for both the control-limb (8.6±2.5 m/s and 8.4±2.2m/s at follow-up, $p=0.744$) and the PTA–limb (8.2±2.2 m/s and 8.3±2 m/s at follow-up, $p=0.116$). Likewise, aPWV remained unchanged with 11.6± 2.7m/s and 11.5± 2.6m/s ($p=0.410$).

Conclusion:

Lower limb angioplasty in peripheral arterial disease leads to an increase in pulse wave velocity in the treated segments. This indicates that peripheral arterial obstructions lower PWV in the muscular-arterial segments. In contrast, lower limb angioplasty does not affect aortic and cfPWV.

2. Introduction

Arterial stiffness is an independent predictor of survival [1]. There are several non-invasive methods for assessment that are easily applied in clinical routine [1].

The gold standard for assessment of arterial stiffness is carotid-femoral pulse wave velocity (PWV), but other devices that assess brachial-ankle PWV are also frequently used [2].

Current guidelines of the European Societies of Hypertension and Cardiology recommend measurement of PWV in patients at intermediate risk for risk stratification and suggest a cut-off value for carotid femoral PWV of 10m/sec for arterial organ damage [3,4,5,6]. An increase in PWV of 1m/sec is associated with a 7% elevated risk for cardiovascular events in a 60-year-old man without cardiovascular risk factors [4].

The ankle brachial arterial pressures index (ABI) is an established vascular marker for the diagnosis of peripheral arterial disease (PAD) and is an independent predictor of cardiovascular events [7]. PAD affects more than 20% of the elderly population and prognosis in these patients is poor due to cardiovascular and cerebral events [7]. Furthermore, ABI reflects atherosclerosis along the arterial tree, and the degree of impairment correlates with a risk of all-cause and cardiovascular mortality [7].

Hence, ABI and PWV are both vascular markers, whereby ABI reflects peripheral arterial obstruction and carotid-femoral PWV stiffening of the aorta due to ageing (sclerosis) or atheroma (atherosis) [8]. In the Rotterdam study, PAD was associated with an increased carotid-femoral PWV [9]. Similarly, Catalano et al. found elevated PWV in PAD when using applanation tonometry [10]. In contrast, a

reduced brachial-ankle pulse wave velocity with lower ABI was found and improvement of both following angioplasty was reported [11].

To our knowledge so far, there is no data on the effect of percutaneous transluminal angioplasty (PTA) on both elastic and muscular arteries assessed as PWV. We hypothesized that PTA affects the PWV of the treated segments, i.e. carotid-femoral or femoro-tibial but not the aortic PWV.

Therefore, we evaluated the impact of PTA on carotid-femoral (cfPWV), femoral-tibial (ftPWV) and brachial-derived aortic PWV (aPWV) in patients with PAD.

3. Patients and Methods

3.1. Study design and patient selection

The study was conducted at a tertiary referral centre as a prospective single-centre evaluation of patients with PAD referred for PTA. The following data were collected for all patients: medical history, brachial systolic and diastolic blood pressures, heart rate, body mass index (weight/height^2), cardiovascular risk factors (arterial hypertension, diabetes mellitus, dyslipidemia, smoking, family history), comorbidities, medication, ankle-brachial index and pulse wave analysis as described below.

Patients continued to take their regular medication. Coronary and cerebrovascular diseases were defined for the purposes of this study by clinical history of either events or interventions. The glomerular filtration rate was estimated by Cockcroft-Gault equation on the basis of serum creatinine levels according to K/DOQI clinical practice guideline [12]. Participants were categorized as currently smoking, stopped smoking less than one year or more than one year ago. Dyslipidemia was defined by a total HDL- cholesterol ratio above 5 mmol/l or the use of lipid-lowering medications. Patients were classified as diabetics if their HbA1c was 6.5% and/or their serum glucose level were ≥7.00 mmol/L, if their physician had diagnosed them and/or if they were treated medically. Patients were considered hypertensive if brachial systolic blood pressure (SBP) was above 140 mmHg or brachial diastolic blood pressure (DBP) was above 90 mmHg or if they were on antihypertensive medications.

Only patients with chronic and stable PAD were eligible for the study. PAD was defined by ankle-brachial arterial pressure index (ABI) of < 0.9. Patients were

assigned in Rutherford stages according to medical history. Exclusion criteria were cardiac arrhythmia and chronic inflammatory vascular disorders.

The measurements of pulse wave velocity were performed before and after PTA with two different non-invasive methods: a brachial cuff-based method using the Mobil-O-Graph (IEM, Stolberg, Germany) which derives an estimation of aortic PWV (aPWV) from pressure waveforms recorded at the brachial artery and second carotid-femoral and femoro-tibial PWV were recorded using the Vicorder (SMT Medical, Würzburg, Germany) device, which detected transit time from cuffs placed around the neck, thigh and ankle [13,14,15,16,17,18,19]. All measurements were made by a single trained physician and a study nurse. The treatments and investigations were part of the standard of care. The study was approved by the ethical committee (2013-0575) and all patients had given written informed consent for procedure and evaluation of data.

3.2. Assessment of Hemodynamics and Arterial Stiffness

Patients were studied in supine position after 10 minutes of rest in a quiet room. SBP and DBP were measured three times in the right and left arm. The average of the last two measurements was recorded as representative of brachial blood pressure and the Mobil-O-Graph placed on the arm with the higher systolic blood pressure for further assessment.

Instantaneously, after brachial blood pressure estimation at least three measurements of carotid-femoral and femoral-tibial pulse wave velocity were obtained with the Vicorder and the mean value was used for analysis of each participant. While the carotid-femoral and the femoral-tibial pulse wave velocity with the Vicorder were performed, the Mobil-O-Graph measurements were recorded simultaneously.

3.3. Mobil-O-Graph

The Mobil-O-Graph (IEM, Stolberg, Germany) suggested by ARCSolver is based on an oscillometric method for assessment of pulse wave velocity via recording of blood pressure with a common cuff placed over A. brachialis. The diastolic blood pressure level is therefore measured for approximately 10 seconds with the conventional cuff and a MPX5050 pressure sensor (Freescale Inc., Tempe, AZ, USA). After digitalization, the single obtained pressure waves are verified for their plausibility, using pulse wave analysis and impedance wave separation. Thereafter they are compared with each other to detect artefacts and finally an aortic pulse wave is induced via general transfer function [13,14,15]. This operator-independent method was invasively validated by Hametner et al. for 24-hour ambulatory blood pressure and aortic PWV monitoring [16].

3.4. Vicorder

The Vicorder (SMT Medical, Würzburg, Germany) is an oscillometric method to record the carotid to femoral and femoral to tibial pulse wave velocity. First, a neck pad is placed other the patient’s neck with a partial inflatable 30-mm plethysmographic sensor positioned over the carotid artery, able to record the carotid pulse wave [17]. A second 100-mm wide blood pressure cuff is placed around the upper thigh to obtain the femoral pulse wave [17].

Both cuffs are inflated to about 60 mmHg and waveforms of the carotid and femoral artery are recorded simultaneously over about 10 consecutive heartbeats to estimate transit time [17]. The beginning of the systole is identified by an inbuilt foot-to-foot algorithm that is centred around the peak of the second derivative of pressure [17].

The path length was defined, as advised by the manufacturer and Hickson and colleagues, by direct measurement between the suprasternal notch to the top of the thigh cuff [17,18,19].

Carotid-femoral PWV velocities were calculated as path length divided by the transit time: cfPWV (m/s) = D (path length)/ΔT(transit time) [17,19]. The measurements of femoral to pedal velocities were taken similarly placing the proximal collar on the right/left upper thigh and the distal cuff respectively over the right/left ankle. The path length was defined between the top of the thigh and the top of the ankle cuff.

3.5. Ankle-Brachial Arterial Pressure Index Assessment

Ankle-brachial pressure index assessments were performed as part of the standard procedure. Systolic blood pressures were measured in both arms and in posterior tibial (PT), peroneal (P) and dorsal pedal (DP) arteries of both limbs using appropriate-sized cuffs and a hand–held 6-MHz Doppler probe (Kranzbühler, Logidop 2, Pilger Medical Electronics, Switzerland). The ABI was than calculated for each leg separately with the highest systolic blood pressure of the PT, P or DP divided by the highest brachial systolic pressures.

3.6. Statistical Analysis

Descriptive statistics for continuous variables are given as mean ± standard deviation. For categorical variables, results are presented as frequency and percentage. Periinterventional changes were analysed by the Wilcoxon signed rank test. Differences between carotid-femoral PWV and aortic PWV were compared by Mann Whitney U-Test.

All data were analyzed using the IBM SPSS software Version 22.0 statistical package for Windows (SPSS Inc. Armonk, NY: IBM Corp.). A value of $p<0.05$ was considered significant.

4. Results

Between January and April 2014, 63 patients fulfilled the clinical selection criteria of which 5 patients were excluded because of invalid readings of pulse volume recordings resulting in 58 patients finally analysed. Lesion localisations and characteristics are summarised in Figure 1. In all 58 patients aPWV were assessed per patient whereas ABI and cf- and ft-PWV were assessed per limb resulting in 115 limbs (1 below the knee amputation) evaluated either as control-limbs (n=53) or PTA-limbs (n=62).

The baseline characteristics of the study population are presented in Table 1. Mean age was 73.7±13 years and 44.8% (n=26) were female. In 57% (n=33) intermittent claudication (Rutherford 2 and 3) was the main complaint and 43% (n=25) had either rest pain or tissue lesions (Rutherford 4 and 5).

The majority of the treated lesions were located unilaterally (87%) and in the femoro-popliteal segments (53%). Only few lesions were located in the iliac (29%) and below-the-knee segments (18%).

4.1. Hemodynamics of the whole cohort

The systolic and diastolic blood pressure of the complete study group did not change significantly after PTA (systolic blood pressure baseline 152±25 mmHg, follow-up 149±21 mmHg, p=0.62; diastolic blood pressure baseline 80±13 mmHg, follow-up 76±9 mmHg, p=0.11). Heart rate also remained unchanged (baseline 68±10/min, follow-up 69±11/min, p=0.70).

There was a significant increase in ftPWV in the PTA-limb (baseline 7.6±2.5 m/sec, follow-up 9.2±2.3 m/sec, p<0.0001) (Figure 2). Similarly, ABI improved significantly in the PTA-limb (baseline 0.67±0.32, follow-up 0.86±0.28, p<0.0001). In

the control-limb, ftPWV (baseline 9.1±2.7m/sec, follow-up 9.2±2.6 m/sec, p=0.334) remained unchanged as well as the ABI (baseline 0.92±0.24, follow-up 0.94±0.25, p=0.405).

The aPWV (Mobil-O-Graph) remained unchanged (baseline 11.6±2.7 m/sec and follow-up 11.5±2.6 m/sec, p=0.410) (Figure 2). Likewise, there were no significant changes in cfPWV assessed by Vicorder in the control-limb (baseline 8.6±2.5 m/sec, follow-up 8.4±2.2 m/sec, p=0.774) as well as in the PTA-limb (baseline 8.2±2.2 m/sec, follow-up 8.3±2 m/sec, p=0.116).

PWV assessed as the carotid femoral travel time measured by the Vicorder device revealed lower velocities than aortic travel time by the Mobil-O-Graph. All p-values were <0.001 for the comparison of aPWV (prior and postinterventional) and both cfPWV of the control- and PTA-limb.

In the univariate regression analyses with PWV as the dependent and age as the independent variable cfPWV measured by Vicorder correlated significantly but not strongly with age in PTA- and control-limb (Figure 3). No significant correlations were found for age and ftPWV (control-limb at baseline and follow-up) nor in PTA-limb at baseline. Mild but significant correlation resulted for age and ftPWV of PTA-limb at follow-up (r=0.31, p<0.02).

4.2 Iliac angioplasty subgroup

When analysing the group of patients with obstructions in the iliac segment only, the results remained the same (Table 2). Revascularisation was associated with a significant increase of ftPWV (Baseline 7.5±2.6 m/sec, Follow-up 9±3 m/sec, p=0.011) and increase of the ABI (Baseline 0.69±0.27, Follow-up 0.85±0.27, p=0.021) in the PTA-limb. There was a non-significant increase in cfPWV in the PTA-limb and control-limb periinterventional (PTA-limb baseline 7.8±1.9 m/sec, follow-up

8.4±1.7 m/sec, p=0.093; control-limb from 7.8±1.9 m/sec to 8.4±2.1 m/sec, p=0.198) (Figure 4. A). Aortic PWV remained unchanged after revascularization (Baseline 10.9±2.9 m/sec, Follow-up 10.8±2.9 m/sec, p=0.448).

When the iliac subgroup was analysed for occluded (n=3) and stenosed (n=15) vessels, there was a trend toward acceleration of cfPWV following revascularization in occluded iliac arteries by Vicorder (occlusions: cfPWV baseline 7.0±2.7 m/s follow-up 8.6±1.4 m/s, p=0.18, stenosis: cfPWV baseline 8.0±1.6 m/s follow-up 8.3±1.7 m/s, p=0.27) (Figure 4. B).

5. Discussion

This study evaluated the influence of PTA in patients with PAD on brachial derived aortic, carotid-femoral and femoral-tibial PWV. The main findings of the study is that femoro-tibial PWV and ABI of the treated limbs increased significantly following lower limb angioplasty, whereas the carotid-femoral and aortic PWV as well as all other hemodynamic parameters did not change.

To the best of our knowledge, this is the first study that examined not only the effect of lower-limb revascularization on PWV of elastic (cfPWV and aPWV) and muscular arteries (ftPWV) but also used two different devices, the Vicorder and Mobil-O-Graph, for carotid-femoral and aortic PWV assessment in PAD patients.

The assessment of PWV with the Mobil-O-Graph (aPWV) revealed higher velocities than the Vicorder (cfPWV). The latter is considered as the gold standard and current guidelines suggest a cut-off value of >10m/sec to diagnose arterial organ damage (AOD). With a mean cfPWV between 8.2 and 8.6 m/sec, the PAD subjects of our study would not fulfill the criteria for AOD in absence of an ABI assessment. When using the more recent tool for aortic PWV with a mean aPWV ranging between 11.5- and 11.6m/sec, our PAD cohort would qualify for AOD. Despite this significant difference among the two methods, there were no periinterventional changes, except for a non-significant trend towards higher cfPWV when iliac obstructions were treated. The validation of the more recent Mobil-O-Graph is based on a comparison with Sphygmocor for central aortic blood pressure and with a cardiac magnetic resonance imaging for PWV showing agreement for both parameters, but direct comparison between Vicorder and Mobli-O-Graph in control subjects is lacking so far [20,21].

A potential shortcoming of the carotid-femoral PWV in PAD may be caused by aorto-iliac obstructions. One explanation for the lower cfPWV assessed by Vicorder in patients with PAD is that below the obstruction a decrease in distending pressure may develop [22]. Consequently, the femoralis pulse wave measured is flatter and the recorded systolic foot-point misrepresented in the oscillometric pulse wave. The transit time estimated with the foot-to-foot method may be underestimated leading to lower velocities in those segments. Brand et al. argued that decrease in distending pressure distal to arterial stenosis may effect a shift to less steep proportion of the exponential vascular pressure-volume relationship, with an attenuation of stiffness in these segments causing decreased cfPWV [22].

PAD represents an interesting subgroup of cardiovascular patients with the extent of atherosclerosis along the aortic and the peripheral arteries. Atherosclerosis is defined by thickening (atherosis) and stiffening (sclerosis) of the arterial wall [8]. Whereas the first is found in the inner layer (intima and media of the artery), the latter is confined to the media and adventitia of the arterial wall. Both structural changes may occur together or separate (i.e. mediacalcinosis) and have an impact on pulse wave propagation. ABI indicates severity of PAD and is an independent marker for prognosis [23]. With ageing, aortic stiffening increases aortic PWV (and pressure pulsatility) due to the loss in elastic properties mainly in the aorta and relates to reduced “Windkessel” effect [24]. In contrast, stiffening of muscular arteries occurs to a much lesser extent [24]. The relation between PWV and ABI in subjects with PAD is unclear [25]. PWV and ABI are both markers of vascular damage providing different information [26]. PWV is a marker of arterial stiffness, and ABI reflects obstruction or occlusion of peripheral arteries [25,26]. Accordingly, PWV is affected by stiffening in the aortic segment, whereas ABI remains unchanged [25].

In Asian countries, arterial stiffness is frequently measured by brachial-ankle (ba)PWV since it is more comfortable and easier to assess than cfPWV [27,28]. In contrast to our study where we evaluated elastic and muscular artery velocities separately, the measurement of baPWV includes elastic and muscular arteries [27]. Therefore potential interactions between a loss of elasticity with increased PWV for the aortic segment and a decrease in PWV due to arterial obstructions in muscular arteries have to be taken into consideration. Due to the shorter pulse wave travel time, cfPWV tends to be lower than baPWV as reported in previous studies [27,29]. However, this difference is not only due to distance but also to travelling speed that varies in different parts of the arterial bed with lower velocities in elastic than in muscular arteries [27]. The arterial obstructions in PAD lead to underestimation of baPWV in patients with advanced PAD [26,29]. This is supported by an analysis of the association of baPWV and ABI on mortality in hemodialysis patients that found a weaker predictive value for mortality for baPWV than for ABI [26]. But when patients with advanced PAD (ABI<0.9) were excluded, baPWV restored its predictive power indicating the interference between the two vascular biomarkers mentioned above [26]. Furthermore, Yokoyama et al. found that baPWV is increased in diabetic patients, whereas it is decreased in the affected legs in diabetic patients with PAD [11]. In accordance with our study, they found that limb angioplasty increased both ABI and baPWV. In addition to their data, we analyzed PWV in three different modes as aortic, carotid-femoral and femoro-tibial and are able to show that PAD in relation to its severity is associated with a lower muscular arterial PWV that is at least attenuated in part after angioplasty.

Calcification of the arteries of the lower extremities leads to a falsely increased ABI and results in underestimation of PAD severity [11,25,26]. Therefore, combined assessment of baPWV and ABI has been suggested [11,25]. The findings that

baPWV is a less powerful risk predictor than ABI in patients on hemodialysis may be due to the fact that blood pressure highly varies in these patients with changes in systolic pressure influencing PWV [29,30]. Applying simply ABI < 0.9 for assessing PAD leads to an underestimation of prevalence of PAD in 20% of the subjects due to normal elevated ABI [25]. Thus, in patients with normal ABI, ba/(ft)PWV may help identifying high-risk individuals [26]. Therefore, our findings have clinical implication in that the assessment of ftPWV may supply complementary information of vascular damage. Based on previous research and our own results, we suggest that ftPWV may be an important adjunct to ABI that may be applied in primary as well as secondary prevention for estimations of the severity of PAD and to assess periinterventional changes of arteries with mediacalcinosis, which needs to be confirmed in future studies.

The trend toward increase in cfPWV following PTA of occluded iliac vessels when assessed by the Vicorder device suggests that iliac obstruction has to be taken into account when cfPWV is assessed. The lack of significance is likely due to the low number of patients with iliac occlusion in our study. An alternative assessment of aortic pulse wave velocity is the brachial derived aortic PWV by the Mobil-O-Graph device, which turned out to be unaffected irrespective of vascular segments that were treated. Furthermore, the measurement with Mobil-O-Graph provided higher velocities than with the Vicorder. This might be due to the fact that PAD is usually restricted to the aorta and the lower limb arteries. The arteries of the upper extremities often remain unaffected. Therefore, assessment of PWV by Mobil-O-Graph in patients with PAD should be preferred although this needs to be supported by furthers studies [11]. Another advantage of assessing PWV by Mobil-O-Graph is that the device is independent of the user, requires no training, can provide simultaneously 24-hour blood pressure and based on an averaged PWV may give

additional reliable information for cardiovascular risk [13]. There is limited information on the association of PWV and PAD. Catalano et al. examined arterial stiffness in PAD patients with the SphymoCor device and found that aortic PWV was higher in patients with PAD than in the control group [10]. Interestingly they found that PAD patients showed no correlation between PWV and age in contrast to the controls indicating the interference of atherosclerosis and PWV mentioned above [10]. In our study there was a weak but significant correlation between cfPWV and age. As expected, we found a strong and significant correlation for age and aPWV (baseline $r=0.97$ $p<0.0001$, follow-up $r=0.96$, $p<0.0001$) which is based on the device algorithm taken age into the calculation and therefore is of limited value.

The limited number of subjects in this first study evaluating two devices, elastic and muscular arteries and the results of the iliac subgroup have to be considered with caution. It is quite likely, that in a larger cohort with iliac occlusions, the periinterventional changes of cfPWV would turn out to be significant for the treated limb. Further studies are needed to prove that and to substantiate the evidence that cfPWV may have to be considered with caution at least in the elderly population with a rather high prevalence of PAD. This goes in line with the methodological limitation of the Vicorder device in that aortic, iliac or femoral obstructions may reduce and delay the pressure wave with a femoral pressure waveform that is inaccurately sensed as reported by Van Bortel et al. [31]. Furthermore, our study included only Caucasian patients. Therefore, these findings have to be verified in other randomized studies with larger population with PAD.

In conclusion, endovascular lower-extremity revascularization significantly increased PWV in the femoral-tibial segment indicating that moderate and severe PAD is associated with lower muscular arterial PWV. In contrast, aortic PWV derived

from the brachial artery remained unchanged. Whether carotid-femoral PWV is altered in presence of PAD needs to be verified in future studies.

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7. Tables and Figures

Table 1. Patient's characteristics (n=58)

Age (years)	73.7 ±13
Male gender, n (%)	32 (55.2)
Height (cm)	168.7 ±10.5
Weight (kg)	75.5±18.4
Body-Mass-Index (kg/m²)	26.3±4.7
Cardiovascular risk factors n (%)	
Arterial Hypertension	49 (84.5)
Hyperlipidemia	29 (50)
Diabetes mellitus	13 (22.4)
Family history	14 (24.1)
Smoker	
current	21 (36.2)
former	15 (25.9)
never	22 (37.9)
Cardiovascular comorbidities n (%)	
Coronary artery disease	21 (36.2)
Cerebrovascular disease	11 (19)
Renal dysfunction	
GFR 50-69 ml/min	14 (24.1)
GFR 30-49 ml/min	17 (29.3)
GFR 15-30 ml/min	5 (8.6)
GFR <15 ml/min	1 (1.7)
Cardiovascular medication n (%)	
Aspirin	41 (70.7)
Clopidogrel	10 (17.2)
Phenprocoumon	7 (12.1)
Statin	31 (53.4)
ACE-I/AT-II-Antagonist	43 (74.1)
Beta blocker	25 (43.1)
Calcium channel blockers	19 (32.8)
Diuretics	24 (41.4)
Rutherford classification n (%)	
Category 2	11 (19)
Category 3	22 (37.9)
Category 4	8 (13.8)
Category 5	17 (29.3)

Data are mean (range or standard deviations) for quantitative variables and number (percentage) for categorical variables
Creatinine clearance according to the Cockcroft- Gault equation

Table 2. Baseline Hemodynamic parameters of the treated iliac arteries

	Baseline	Follow-up	p
Pulse Wave Velocity (m/sec)*			
Brachial	10.9±2.9	10.8±2.9	0.448
PWV-PTA-Limb (m/sec)**			
Carotid-femoral	7.8±1.9	8.4±1.7	0.093
Femoro-tibial	7.5±2.6	9±3	0.011
PWV-Control-Limb (m/sec)**			
Carotid-femoral	7.8±1.9	8.4±2.1	0.198
Femoro-tibial	8.6±2.2	8.8±3	0.396
Ankle-Brachial Arterial Pressure Index			
PTA-Limb	0.69±0.27	0.85±0.27	0.021
Control-Limb	0.87±0.21	0.93±0.22	0.315

Values refer either to subjects (n=16), to PTA- Limbs (n=18), or to Control- Limbs (n=14);
PWV: Pulse wave velocity, *Mobil-O-Graph, **Vicorder

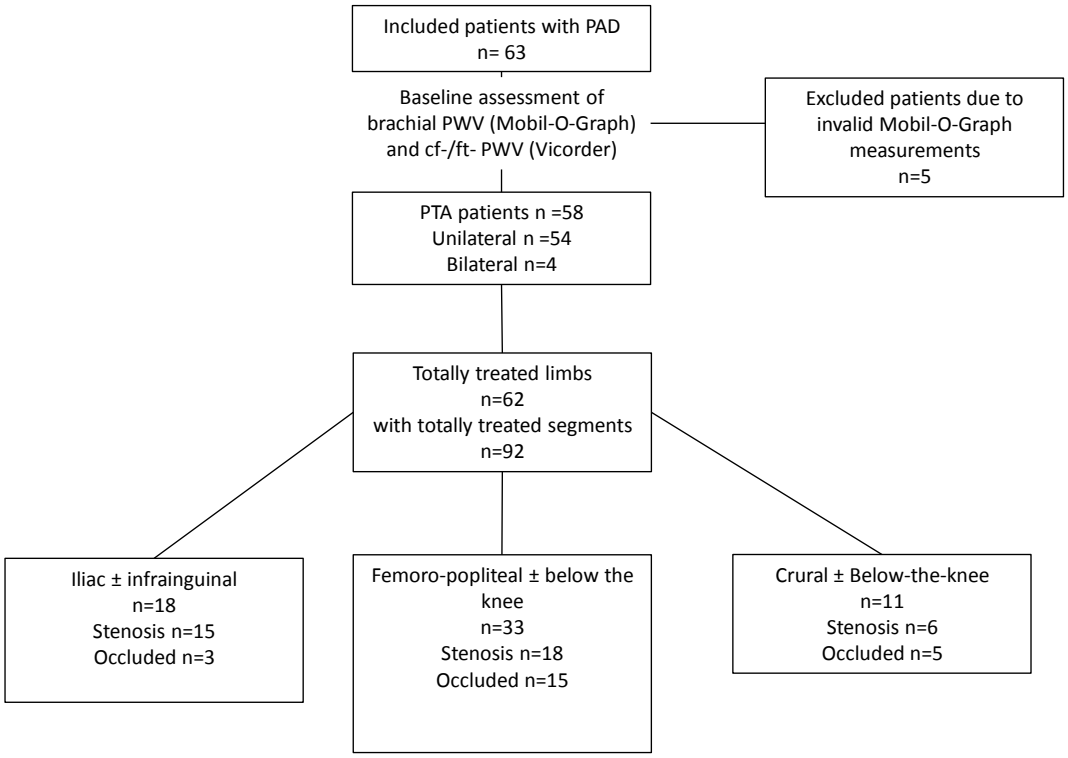

Fig. 1. Flow chart of included patients with peripheral arterial disease, treated limbs and vascular lesion. Obstruction characterstics refer to the proximal lesion treated.

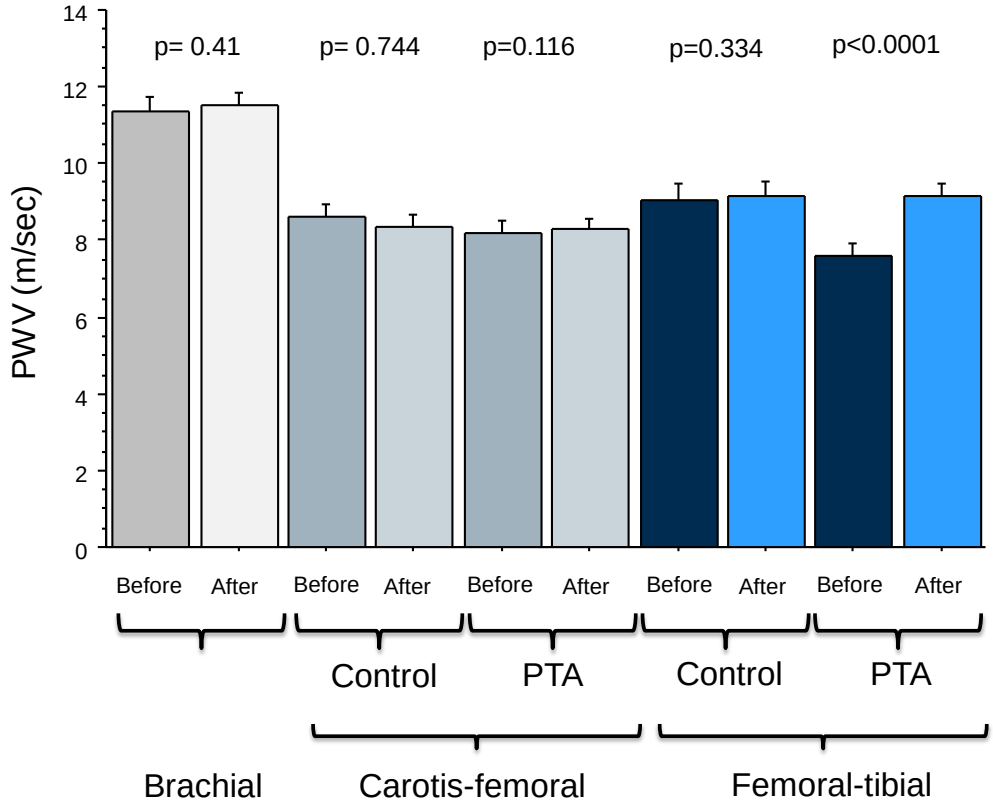

Fig. 2. Comparison of Brachial-, Carotis-femoral and Femoral-tibial PWV in m/sec before and after PTA in Control- and PTA-group. Error Bars: ± 1 Standard Deviation.

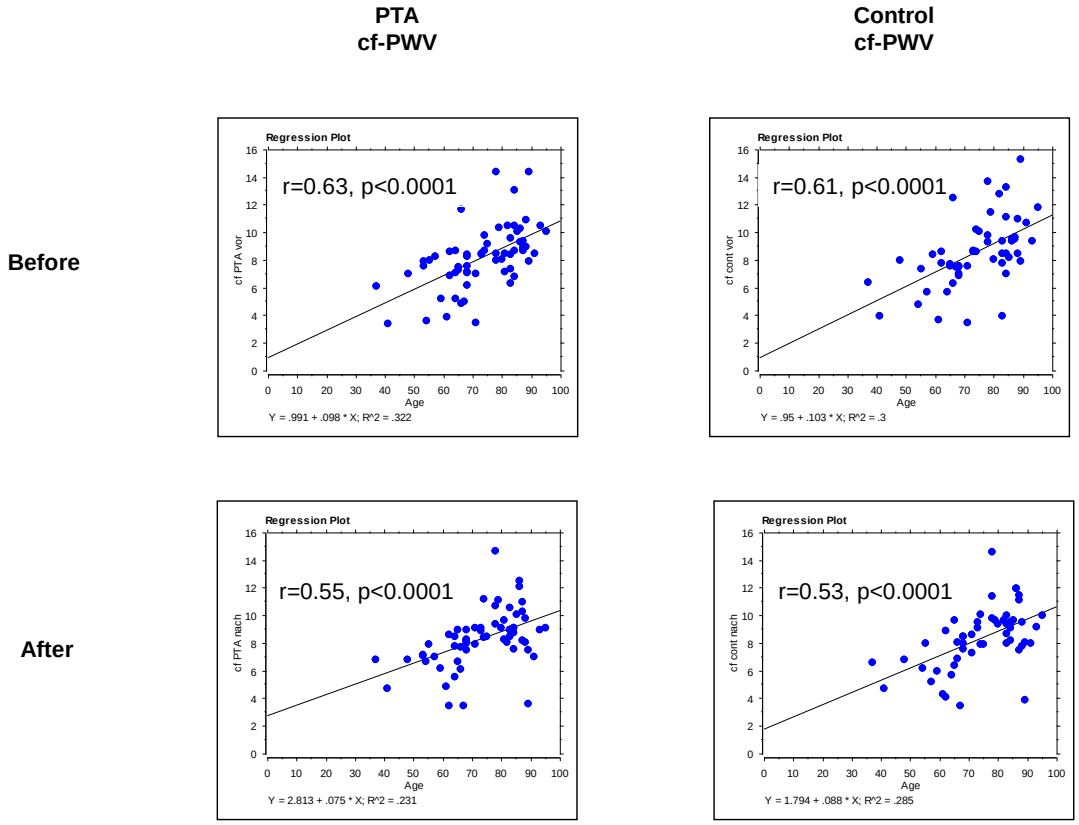

Fig. 3. Regression Plot for Pulse wave velocity by Mobil-O-Graph (Brachial PWV)/ Vicorder (cf-PWV) in PTA- versus Control-group and age in patients with peripher arterial disease before and after PTA.

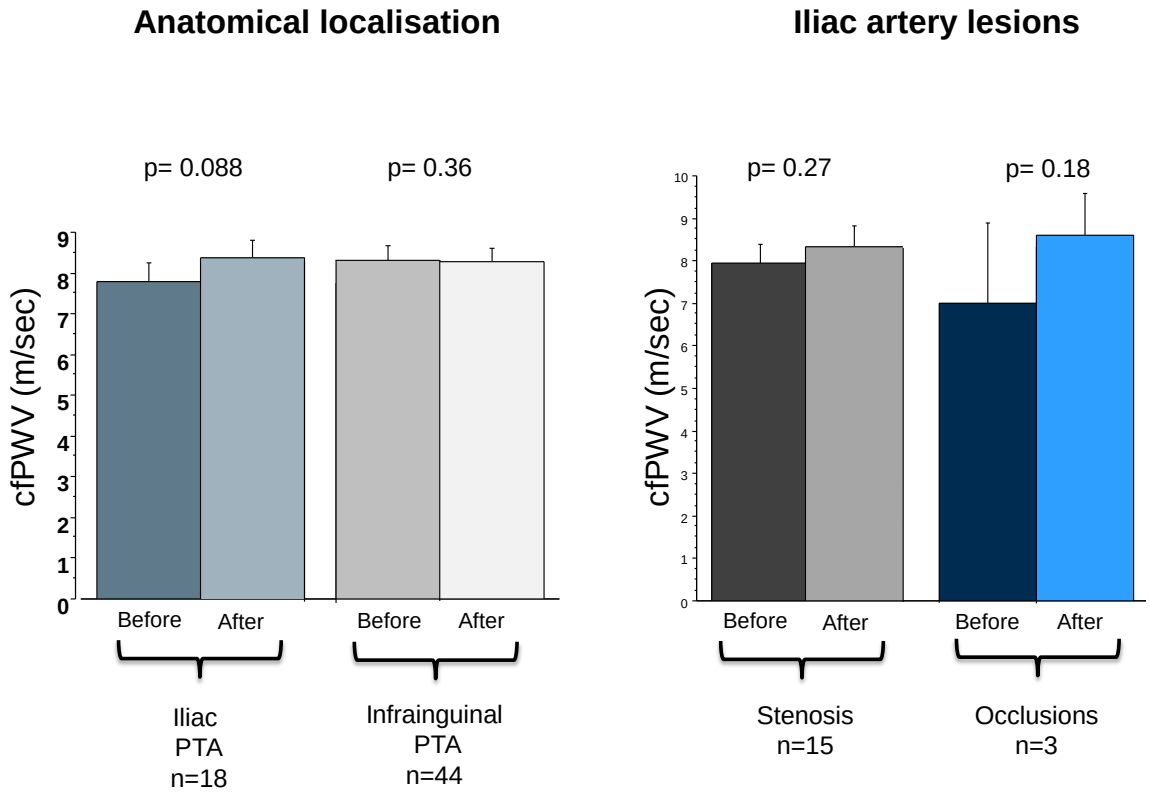

Fig. 4. A) **Fig. 4. B)**

Fig. 4. A) cfPWV in m/sec before and after PTA according to anatomic localisation of the lesion in n lower extremities. Error Bars: ± 1 Standard Deviation. **B)** cfPWV before and after PTA according to n iliacal obstructions (stenosis versus occlusions). Error bars: ± 1 standard deviations.

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