Summary

Laser Doppler flux signals show temporal fluctuations caused by physiological phenomena like heartbeat, respiration, and local tissue vasomotion. These fluctuations are often filtered out in the data analysis process. This study investigated whether the laser Doppler fluctuations caused by the heartbeat contain clinically useful information. The dependence of these fluctuations on monitor type, fibre arrangement and probe location was also addressed. By using two perfusion monitors and two probes at the skin with different fibre arrangement, the flux signals during an occlusion test were analysed in subjects suffering from Fontaine class II-III peripheral arterial obstructive disease (PAOD) and patients with diabetes mellitus, and compared with those of healthy subjects. The flux signals were filtered with a band-pass filter between 0.5 and 3 Hz followed by root mean square processing and averaging over 4 seconds. The processed flux signals resembled the original tracing. The ratio between these signals, referred to as pulsatility, was shown to be significantly smaller in patients with PAOD (p < 0.05) on all channels at the dorsum of the foot, and for one of the fibre distances at the toe. For each probe, the pulsatility almost had the same value for the results for all detection fibres and instruments. Pulsatility during the hyperaemia peak showed a maximum value of ~0.3. From this study it is concluded that the laser Doppler fluctuations caused by the heartbeat contain clinically useful information.

Manuscript is to be submitted.
6.1 Introduction

Laser Doppler perfusion monitoring (LDPM) is a well-known non-invasive technique to assess tissue microcirculation allowing the continuous recording of tissue blood perfusion [16]. In a typical LDPM recording session temporal variations of the flux signal are commonly observed, which are mostly caused by physiological phenomena such as the cardiac rhythm, the respiration, and local tissue vasomotion [7,9,17]. Several studies have focused on the clinical relevance of assessing the low frequencies (< 0.1 Hz) contained in the LDPM-flux signal which are produced by the vasomotion [10,15,19]. Other studies have investigated characteristic mean flux values or the changes in tissue perfusion caused by provocations such as arterial occlusion [6,8] or local heating [3,20]. However, the question remains whether higher frequency oscillations of the flux signal (> 0.5 Hz) have clinical relevance. Recently, Rossi et al. studied the frequency interval from 0.6 to 1.8 Hz of LDPM flux signals obtained from patients with peripheral arterial obstructive disease (PAOD), and found impaired response during reactive hyperaemia [14].

The cardiac cycle provokes oscillatory changes in arterial pressure that are transmitted through the arterial tree as pulse waves. The resulting changes in the pulsatile blood flow are typically observed in distal places of extremities using methods such as photoplethysmography, Doppler ultrasound, dynamic intracapillary pressure [4], and laser Doppler perfusion monitoring. However, the use of the pulsatile changes caused by the heartbeat has been little addressed for LDPM [1,14].

A recently proposed model of the peripheral circulatory system used a basic electrical circuit of resistances and capacitors to explain the LDPM flux signals recorded during a post-occlusive reactive hyperaemia (PORH) test [5]. This model was applied to the results of measurements during PORH tests, which showed significant differences in the response for subjects with PAOD [12]. The model pays attention to the fact that the peripheral vascular network may act as a low-pass filter, which may be influenced by changes in the vascular resistances and compliances of the arteries and arterioles. These changes affect the propagation of heartbeat pulses as well. Therefore, if a situation of high arterial resistance and low capillary resistance exists, the pulses produced by the heart might be attenuated whereas the lower frequencies (vasomotion) should remain unchanged because they originate from the measuring location.

The PORH test is based on the vasodilator effect induced by a short period of ischaemia. This is a well-known method for assessing the microvascular
function. The rapid perfusion changes that occur during the PORH are easily measured with LDPM. Del Guercio et al. measured the PORH response with LDPM, and then calculated various time-dependent parameters [6]. This analysis method requires a smooth LDPM tracing that is typically obtained by averaging the signals over one second or more. Nevertheless, the information filtered by this analysis method may contain clinically useful information.

Other items of the present study with reference to the heartbeat fluctuations are the comparability of results obtained at the toe and the dorsum of the foot, the influence of the distance between the source and detector fibres at the skin surface, and the use of different LDPM monitors. Because every manufacturer of laser Doppler perfusion monitors uses a slightly different approach to process the signals, it is not certain that the results obtained can directly be compared.

For this study, LDPM flux signals were recorded from patients with peripheral arterial obstructive disease, diabetes mellitus and subjects without symptoms of peripheral vascular disease. Results from two instruments, from which one has four channels, have been compared using multi-fibre probes at the dorsum of the foot and the toe. The aim of this study is to investigate the clinical involvement of heart beat fluctuations in LDPM flux signals, which are normally filtered out.

6.2 Methods

6.2.1 Equipment

The LDPM monitors used were a dual-channel Pf4001 (Perimed AB, Järfalla, Sweden) and a four-channel moorLAB server system (Moor Instruments Ltd., Axminster, UK). In total, six channels were available, each consisting of one low-power 780 nm laser and one detector. For this study, only two lasers were used. The LDPM monitors were set to the minimum time constant (0.2 s for Pf4001, 0.03 s for moorLAB), and the maximal cut-off frequency was set at 12 kHz (fixed setting) for Pf4001, and at 22 kHz (selectable setting) for moorLab. The analogue output for flux and concentration signals of both monitors were connected to an analogue-to-digital acquisition card (PC-LPM-16/PnP, National Instruments Co., Austin, USA) with 12 bit of resolution. The recording software was developed with Labview v5.1 (National Instruments Co.) running in a laptop computer.

The signals from five channels were obtained using two custom-made probes placed at two different locations at the foot. The first probe was placed on the dorsum of the foot, between the second and third metatar-
It was a straight-cylindrical probe with one central fibre surrounded by six fibres hexagonally distributed (fig. 6.1). All the fibres had a core diameter of 0.125 mm and a separation between fibres (centre-to-centre) of 0.25 mm. These seven fibres had independent connectors to the LDPM monitor, so any combination of source and detection fibres was possible. For this probe, only one fibre was used as illuminating fibre and was connected to the moorLab server’s laser. Two of the fibres located at 0.25 mm from the illuminating fibre were plugged to the Pf4001’s detector (P1) and to the moorLab server’s detector (M1), respectively. The only fibre at 0.50 mm from the illuminating fibre was connected to the first moorLab satellite’s detector (M2).

The second probe was placed on the pulp of the hallux. It was a 90° angled probe with the illuminating fibre at the centre of a circle formed by eight detection fibres distanced one millimetre from the central fibre. A ninth detection fibre was located at 0.2 mm (centre-to-centre separation) from the illuminating central fibre. All fibres had a core diameter of 0.125 mm (fig. 6.1). Only one cord came out of the probe, and at the distal side it ended in three wires with ST-connectors carrying the 0.2 mm detector fibre (M3), the illuminating fibre, and the eight detection fibres at 1.0 mm (M4), respectively.

The monitors were calibrated using the probes and connections shown in Figure 6.1. Each monitor was calibrated using the calibration liquid (motility standard) provided by its manufacturer. The calibration was performed under low light conditions and at a room temperature of 22 ±1 °C. The resulting flux signals over 120 seconds, from the respective calibration liquids, were 250 perfusion units (PU) for Perimed and 220 PU for Moor. After the initial calibration, the flux output signal was regularly checked, against the respective motility standard, before the measurements were performed. In case the recorded flux signal changed more than ±50 PU from the resulted calibration value, a new calibration was performed following the above procedure.

Arterial occlusion was performed using an 18-cm wide pneumatic cuff (CC17, D. E. Hokanson Inc., Bellevue, USA) attached around the thigh of the measured leg. The cuff was driven by a rapid cuff inflator system (E-20 rapid cuff inflator and AG-101 air source, D.E. Hokanson) to quickly inflate/deflate the cuff.

6.2.2 Test subjects

Approval for this study was obtained from the medical ethical committee of the University Medical Centre Groningen. Thirty subjects of a comparable age were recruited from three groups:
• PAOD: Ten subjects of type II-III Fontaine class peripheral arterial obstructive disease (with claudication or rest pain, and no critical limb ischemia). PAOD was documented by an ABI (ankle-brachial index) < 0.9, and/or Doppler ultrasound or angiographic studies confirming a severe stenosis or occlusion. Most of the patients were participants in a so-called walking training program. The mean age was 61.3 ±8.7 years (range 49-76 years, six men).

• DM: Ten subjects with diabetes mellitus (type 1 and type 2) without symptoms of PAOD. The mean age was 51.7 ±6.6 years (range 43-63 years, seven men). Diabetes had been diagnosed using conventional ADA criteria [2].

• Controls: Ten subjects with no clinical symptoms of vascular disease. The mean age was 55.1 ±8.1 years (range 46-68 years, 10 men).

Subjects with a history of congestive heart failure were excluded. Furthermore, drugs with vasoactive effects were not allowed in the hours before the measurement.

Figure 6.1. Layout used for the LDPM probes. On the left is the straight cylindrical probe installed on the dorsum. On the right is the 90° probe used on the toe.
6.2.3 Experimental protocol

The LDPM recordings were performed in a quiet environment. The room had a temperature of 25 ±1 °C. All the subjects comfortably rested on a bed in the supine position. Each subject had 30 minutes for acclimatisation prior to the test. The recording session lasted 33 minutes without stops, and had three stages: resting, occlusion, and reactive hyperaemia. The first stage always lasted 15 min to record a stable resting flux reference signal for a sufficient period. After 3 minutes of occlusion, the signal was recorded for another 15 minutes to obtain the PORH response and its return to basal level.

6.2.4 Data processing and analysis

The analogue-signal outputs for flux and concentration of the LDPM monitors were recorded at a sample rate of 40 Hz, creating discrete data files per subject and measuring session. The data in millivolts were converted into perfusion units (10 mV = 1 PU) (fig. 6.2). These data, referred to as RAW-Flux, were processed in two different ways.

RMS-Flux signals: The frequency spectra of the RAW-Flux and concentration signals were investigated. By applying the FFT, the frequency spectra of the flux signals presented peaks at the heartbeat frequency and at the first harmonic. The heartbeat frequency range was 0.9-1.1 Hz (54-66 bpm). From these data we decided to use an 8th order band-pass filter created with Labview with cut-off frequencies of 0.5 and 3 Hz. Only the signal fluctuations in the band pass range were amplified, whereas the rest were attenuated. Next, the mean amplitude of the signal was obtained by calculating the root mean square (RMS) within a moving window of four seconds wide, in such a way that a new data point per second was obtained. This new signal is referred to as RMS-Flux. The same processing was also performed for the concentration signals. However, it appeared that the concentration signals did not show any frequency component in the 1 Hz-range. Hence, these results were not further considered.

Pulsatility signals: A pulsatility signal was obtained by dividing the RMS-Flux signal by the Average Flux signal, Avg-Flux. The latter was obtained by averaging the RAW-Flux signals over one second to remove the frequencies above ~0.5 Hz. Subsequently, these were plotted to calculate the biological zero, BZ [18] by averaging the lowest range of flux during occlusion. Then the BZ value was subtracted from every data point recorded. The resulting tracing from the ratio of RMS-Flux and Avg-Flux signals was referred to as the Pulsatility.
The following parameters were calculated from both the Avg-flux and RMS-flux tracings:

- **Resting flux** (RF) was defined as the mean flux calculated from the longest signal excerpt free of artefacts, and representing the pre-occlusion period.

- The **time to resting flux** ($t_{RF}$) was defined as the time when the first flux data-point was higher or equal to the RF value.

- The **time to maximum flux** ($t_{MF}$) and the **maximum flux** (MF) values were determined at the highest flux value.

- The **RF-pulsatility** and **MF-pulsatility** were calculated from the ratio between the RMS-Flux and the Avg-Flux during RF and MF conditions. Mean pulsatility for RF and the pulsatility at MF were denoted as RF-pulsatility and MF-Pulsatility, respectively.

- The **RMS-Flux ratio** was defined as the ratio between MF and RF of the RMS-Flux signal. This parameter shows the relative increase of RMS-Flux and gives an idea of the remaining reserve capacity of the microvasculature.

By means of the two-tailed Student’s t-test, comparisons of the calculated parameters were performed between the controls and each patient group (assuming different variances). The paired-samples t-test was used to compare the signals between channels within the same subject, with a confidence of 95%. Only normally distributed data were used in the test. A value of $p<0.05$ was considered significant, if not mentioned otherwise.

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**Figure 6.2. Schematic overview of the data processing used for the LDPM signals.**
6.3 Results

6.3.1 Influence of the signal processing methods

Figure 6.3 is an example of a flux signal excerpt recorded from the hallux of one healthy control subject including the occlusion and most of the PORH response. The RAW-Flux signal (fig. 6.3A) that was obtained directly from the laser Doppler perfusion monitor shows the rapid heartbeat variations. Slower fluctuations were caused by the respiration rhythm and the vasomotion.

Figure 6.3B shows the resulting 1-second averaged signal calculated from the RAW-Flux, here referred to as the Avg-Flux. Notice that the heartbeats have been removed and the tracing still shows most of the slower fluctuations. Furthermore, the RMS-Flux (fig. 6.3C) shows many similarities with the two above graphs. However, the most interesting difference between the figs. 6.3B-C is the rapid decrease of the RMS-flux at the beginning of the occlusion. In general, the contribution of the RMS-flux to the total Flux signal was smaller in the toe (up to 40%) than in the dorsum (up to 80%).

Table 6.1. Summary of RMS-Flux values and pulsatilities (mean ±SD) from the different channels for control subjects.

<table>
<thead>
<tr>
<th></th>
<th>Dorsum Probe</th>
<th></th>
<th>Hallux Probe</th>
<th></th>
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<tbody>
<tr>
<td></td>
<td>Moor 0.25 mm (ch. M1)</td>
<td>Moor 0.50 mm (ch. M2)</td>
<td>Perimed 0.25 mm (ch. P1)</td>
<td>Moor 1.0 mm (ch. M3)</td>
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<tr>
<td><strong>RMS RF</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Control</td>
<td>1.9 ±1.6</td>
<td>3.1 ±2.1</td>
<td>2.9 ±2.3</td>
<td>26.6 ±19.4</td>
</tr>
<tr>
<td>DM</td>
<td>1.9 ±1.7</td>
<td>3.1 ±2.4</td>
<td>2.1 ±2.3</td>
<td>23.7 ±21.2</td>
</tr>
<tr>
<td>PAOD</td>
<td>1.2 ±0.7</td>
<td>1.5 ±0.6(^a)</td>
<td>1.2 ±0.7(^a)</td>
<td>14.7 ±15.0</td>
</tr>
<tr>
<td><strong>Pulsatility RF</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>0.33 ±0.23</td>
<td>0.31 ±0.18</td>
<td>0.31 ±0.22</td>
<td>0.19 ±0.06</td>
</tr>
<tr>
<td>DM</td>
<td>0.22 ±0.12</td>
<td>0.23 ±0.11</td>
<td>0.22 ±0.14</td>
<td>0.18 ±0.09</td>
</tr>
<tr>
<td>PAOD</td>
<td>0.19 ±0.12(^a)</td>
<td>0.17 ±0.11(^a)</td>
<td>0.17 ±0.12(^a)</td>
<td>0.13 ±0.07</td>
</tr>
<tr>
<td><strong>RMS MF</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>4.6 ±4.0</td>
<td>7.3 ±6.1</td>
<td>5.3 ±2.7</td>
<td>43.0 ±17.1</td>
</tr>
<tr>
<td>DM</td>
<td>3.1 ±2.4</td>
<td>4.9 ±3.0</td>
<td>6.6 ±12.7</td>
<td>34.9 ±23.2</td>
</tr>
<tr>
<td>PAOD</td>
<td>2.8 ±1.7</td>
<td>3.3 ±1.5(^a)</td>
<td>2.8 ±2.1(^a)</td>
<td>26.4 ±19.5(^b)</td>
</tr>
<tr>
<td><strong>Pulsatility MF</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>0.18 ±0.08</td>
<td>0.18 ±0.07</td>
<td>0.17 ±0.07</td>
<td>0.17 ±0.07</td>
</tr>
<tr>
<td>DM</td>
<td>0.16 ±0.06</td>
<td>0.17 ±0.06</td>
<td>0.21 ±0.20</td>
<td>0.17 ±0.07</td>
</tr>
<tr>
<td>PAOD</td>
<td>0.15 ±0.08</td>
<td>0.13 ±0.07</td>
<td>0.13 ±0.06</td>
<td>0.12 ±0.05</td>
</tr>
</tbody>
</table>

\(^a\) Significance compared to Controls p < 0.05
\(^b\) Significance compared to Controls p < 0.02
Figure 6.3D shows the pulsatility calculated from the LDPM signal in Figure 6.3A, i.e. the ratio of the RMS-Flux and the Avg-Flux. Large signal fluctuations appeared during the occlusion part, because in that range the pulsatility was obtained by dividing two very small signals that contained noise.

6.3.2 RMS-Flux signals

In the dorsum, the Controls and DM patients had similar RMS-RF and RMS-MF values for all channels (Table 6.1), whereas significantly different values between Controls and PAOD patients were found for the M2 and P1 channels (p < 0.05 for RF and for MF). In the toe, only RMS-MF for channel M3 was significantly different between Controls and PAOD patients (p < 0.02).

6.3.3 Pulsatility

In Figure 6.4, the pulsatilities in the resting condition, RF-pulsatility, were compared pairwise for the fibres at the two probe locations (channels M1-M2 and M1-P1 at the dorsum, and M4-M3 at the toe), as well as the combination M1-M4. In general, the RF-pulsatilities for each subject were very similar when obtained from the channels at same probe location (Figs. 6.4A-C). For RF-pulsatility, significant differences between these values from the dorsum of the foot, as summarised in Table 6.1, were only found between channels M1-P1 (p < 0.05) for PAOD patients. The RF-pulsatilities between dorsum and toe locations were clearly less correlated (Fig. 6.4D). These were only significantly different for Controls (p < 0.05 for M1-M4), where higher values of the RF-pulsatilities were found in the dorsum of the foot (Table 6.1).

When comparing fibre distances within a patient group for the MF-pulsatility in the dorsum, the only significant differences found were between M1-P1 (p < 0.05) in the Controls and M1-M2 (p < 0.01) in the PAOD group. In the toe, the M3-M4 channels differed from each other for subjects with DM (p < 0.01) and PAOD (p < 0.05), not for Controls.

With respect to the comparison of the patient groups with Controls, it was observed that RF-pulsatilities below 0.13-0.15 at the dorsum of the foot were only observed in the DM and PAOD groups. Nevertheless, pulsatility values from the dorsum of the foot of all Controls and DM patients had similar values for all three channels. The latter was true for both the pulsatility in RF and that in MF. PAOD patients significantly differed from Controls for all channels (p < 0.05) except for M3. For MF-pulsatility significant differences were found for the M4 channel of PAOD only (p = 0.021).
Figure 6.3. Example of the signals obtained from the hallux of a healthy subject: the RAW-Flux (A), the Avg-Flux (B), the RMS-Flux (C), and the pulsatility (D).
Figure 6.5 shows the comparison of RF- and MF-pulsatilility for various channels at the dorsum (figs. 6.5A-C) and at the toe (fig. 6.5D). For lower RF-pulsatilities, these values roughly were the same. However, on the dorsum, a maximum value around 0.3 was found for the MF-pulsatility for both LDPM monitors, but not for the RF-pulsatility. This maximum value for MF-pulsatility was not observed at the toe where similar pulsatilities were observed in both toe channels for all values (M3 and M4). Although control subjects seldom showed pulsatilities below 0.15, no significant differences were found between the data of the patient groups and the control group.

6.3.4 Time-parameters

RMS-tRF was only significantly different between PAOD and controls for the M2 channel (p < 0.03). The RMS-tRF value could not be obtained from seven subjects (4 controls, 1 DM, and 2 PAOD).

The RMS-tMF was only significantly different between PAOD and Controls for the M1 channel (p < 0.03). Values of four subjects (1 control, 1 DM, and 2 PAOD) could not be obtained for the above parameters.

![Graphs showing comparison of RF-pulsatility values](image)

*Figure 6.4. Comparison of the RF-Pulsatility values of various fibre separations in the dorsum (A and B) and in the pulp of the hallux (C).*
6.4 Discussion and Conclusions

The digital filters used in this study to process the LDPM flux signal (fig. 6.3A) yielded two calculated flux signals, the Avg-Flux and RMS-Flux. It was remarkable that the variation of the amplitude of the cardiac pulses (fig. 6.3C) resembled the main variations in the Avg-Flux signal (fig. 6.3B). However, there were exceptions. When the occlusion started, the RMS flux signal dropped quicker than the Avg-Flux (compare figs. 6.3B and 6.3C). The heartbeat pulses immediately stopped with cuff inflation, however the Avg-flux signal remained for a while because some time is needed to reach equilibrium between the arterial and venous pressures. Nevertheless, the resemblance of the variations in the Avg-Flux signal and the RMS-Flux signals was the reason for introducing the pulsatility as a new parameter.

In our population, we found significantly different values for RMS-RF and/or RMS-MF between patients with PAOD and control subjects for the P1, M1 and M3 channels, whereas we were unable to find such differences for Avg-Flux, and Avg-MF (to be submitted elsewhere). Therefore, there may be some advantage in using RMS-Flux signals instead of Avg-Flux signals to discriminate patients with PAOD and Controls. This may be related to the somewhat smaller relative errors in Table 6.1 (SD/mean) for the values of RMS-RF when compared to Avg-RF. However, this advantage of RMS-RF over Avg-RF is small, as it has only led to a significant difference for channel P1.

The pulsatilities, which are the ratio of RMS-Fluxes and Avg-Fluxes have more advantage than the Avg-RF and RMS-RF, as the relative errors are even smaller, and show the most significant differences in the flux signals between control subjects and the PAOD patient group. The individual values of the RF-Pulsatility in part of the patients with PAOD, and of some with Diabetes Mellitus, were smaller than the values in the Control group. Similarly, Rossi et al. found decreased values of the mean peak power in the frequency band from 0.6 to 1.8 Hz of LDPM signals measured on PAOD patients [14]. Our observations of smaller pulsatilities in PAOD subjects compared to Controls are also in agreement with the decreased pulsatility from directly measured capillary blood pressure fluctuations [13] as well as with Doppler ultrasound tracings from large blood vessels distally from stenoses.

Our results also showed that RF-pulsatility has to be preferred over MF-pulsatility, which only gave significant results for one signal on the toe. The use of RF-pulsatility has other advantages as well. The calculated values for each channel at the dorsum of the foot were very similar for all combinations of fibre distances and instruments (see figs. 6.4A-C and Table 6.1).
With respect to comparability of laser Doppler instruments, the similar values of the RF-pulsatilities are interesting, as they indicate that LDPM monitors of different brands and with probes that measure at various fibre distances have an excellent comparability. Finally, the use of RF-pulsatility does not require a PORH test, which is an advantage when the patient experiences much pain from such occlusions.

As a larger fibre distance is also related to deeper penetration of part of the light, and thus a larger measuring depth [1,4,5,11], the similar values of RF-pulsatility at various fibre distances is an indication that the laser Doppler signals obtained at various (small) distances (and thus depths) from the source fibre are well related. From our results, we conclude that the fibre separations 0.25 mm and 0.50 mm did not correspond to volumes with different perfusion characteristics. These results agree with previous results (see in Chapter 3) that showed a similar ratio between the maximum flux (MF) during the PORH and the resting flux (RF) measured by two LDPM monitors. This cannot be concluded for pulsatility results at the dorsum of the foot in comparison with pulsatility results at the toe, as these results did not correlate well (fig. 6.4D). This is probably due to local differences in the microcirculation and the different microvasculature present in those two body areas.

Figure 6.5. Comparison of the MF- and RF-Pulsatility values in the dorsum (A, B, and C), and in the hallux (D).
It was observed that an apparent maximum value occurred for the MF pulsatility in the dorsum when comparing with RF pulsatility (fig. 6.5A-C). Because this happened for both instruments and with both fibre separations at the dorsum, we concluded that it is not due to the instruments. Most likely, the pulse propagation in the dorsum microvasculature was different during the PORH response compared to during the resting period. Furthermore, it is remarkable that the phenomenon described above was not observed from the toe measurements (fig. 6.5D).

The time-parameter RMS-t<sub>RF</sub>, that was derived from the RMS-RF signal of a PORH test, did not show advantages compared to the corresponding parameter derived from the Avg-RF signal in making distinction between the Patient groups and the Controls (fig. 3.2).

In summary, the contribution of the heartbeat pulses to the LDPM flux signal, as observed in this study shows interesting features. Not only the RMS-Flux signal, but also especially the pulsatility gives much better possibilities to discriminate between patients with PAOD and Controls. The similar RF-pulsatility results for both instruments and for the different fibre separations, especially at the dorsum of the foot, show that these instruments produce proportional results and show little variation upon variation of the distance between the illuminating and detecting fibres. Future, more clinically oriented studies may investigate whether the new parameter RF-pulsatility contributes to the evaluation of the status of patients with PAOD.

6.5 References


