**Coronary artery height differences and their effect on fractional flow reserve**

**Running Title: Hydrostatic pressure and FFR**

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**Abstract**

**Background:** Fractional flow reserve (FFR) uses pressure-based measurements to assess the severity of a coronary stenosis. Distal pressure (Pd) is often at a different vertical height to that of the proximal pressure (Pa). The difference in pressure between Pd and Pa due to hydrostatic pressure, may impact FFR calculation.

**Methods:** One hundred CT coronary angiographies were used to measure height differences between the coronary ostia and points in the coronary tree. Mean heights were used to calculate the hydrostatic pressure effect in each artery, using a correction factor of 0.8mmHg/cm. This was tested in a simulation of intermediate coronary stenosis to give the “corrected FFR” (cFFR) and percentage of values, which crossed a threshold of 0.8.

**Results:**The mean height from coronary ostium to distal LAD was +5.26cm, distal Cx -3.35cm, distal RCA-PLV -5.74cm and distal RCA-PDA +1.83cm.For the LAD, correction resulted in a mean change in FFR of +0.042, -0.027 in the Cx, -0.046 in the PLV and +0.015 in the PDA. Using 200 random FFR values between 0.75 and 0.85, the resulting cFFR crossed the clinical treatment threshold of 0.8 in 43% of LAD, 27% of Cx, 47% of PLV and 15% of PDA cases.

**Conclusions:** There are significant vertical height differences between the distal artery (Pd) and its point of normalisation (Pa). This is likely to have a modest effect on FFR calculation and the results in values crossing the treatment threshold. Operators should be mindful of this phenomenon when interpreting FFR values.

**Keywords:** Hydrostatic Pressure, CT Coronary Angiography, Coronary Stenosis

**Introduction**

Fractional flow reserve (FFR) is the gold standard for invasive assessment of flow limitation caused by a coronary stenosis and it has been shown to improve clinical outcomes in randomised clinical trials [1, 2, 3]. In practice, FFR is calculated as the ratio of the distal trans-stenotic pressure to the proximal coronary or aortic pressure during pharmacological hyperaemia. The hydrostatic consequences of the wire position are one of the recognised pitfalls when FFR measurements are performed. Coronary arteries lie in different vertical planes and height variations are part of normal anatomy. Thus, the pressure wire sensor measuring distal pressure (Pd) is seldom at the same level with the coronary ostium where aortic pressure (Pa) is measured and where the Pd and Pa were previously equalised. This effect is present in any pressure based measurement, including the resting indices such as instantaneous wave free ratio (iFR) [4]. Despite strong evidence for its use, FFR remains underutilised [5]. Avoiding confounding factors when using pressure based indices is crucial in accurate stenosis assessment.

In clinical practice hydrostatic effect produces FFR values higher than 1.00 in a non-diseased vessels, most commonly positioned posteriorly [6]. A recent study documented coronary ostia and distal vessels height differences in an elderly patient cohort with aortic stenosis [7]. Furthermore, the investigators used an *in vitro* model to calculate the impact of their observed height difference in pressure derived physiological indices. The observed changes were small meaning that it is unlikely to cause a significant change of FFR value in clinical practice. However, when using a binary cut-off for flow limitation for a given coronary stenosis, even a change of 0.02 can change the classification of FFR from ischaemic to non-ischaemic (FFR form 0.79 to 0.81).

In this study, we aimed to quantify the height differences between the distal coronary vessels and the corresponding coronary ostia in a supine position in a real life cohort of patients undergoing investigations for coronary artery disease. Based on these measurements, we tried to quantify the effect of coronary anatomical variations on FFR values around the ischaemic cut-off point of 0.80.

**Methods**

We conducted a retrospective analysis of 100 patients undergoing CT coronary angiograms from August 2016 to April 2017 for new onset chest pain suspected to be angina. Vertical coronary height measurements were recorded in all coronary arteries and then used to calculate the potential hydrostatic effect on that specific point in the artery. The effect of the calculated pressure difference and hence effect on FFR was applied to a model of two hundred randomly generated FFR values. FFR was compared pre- and post-correction for hydrostatic force.

*Inclusion and Exclusion Criteria*

All patients were elective outpatients under investigation for angina. Patients with previous bypass grafting or valve surgery were excluded. Scans, which did not show the upper rim of the CT table could not be analysed (as this was the reference point for measurement). Coronary visualisations with poor contrast penetration, or significant artefact were excluded. Finally, left dominant coronary circulations were not included in analysis.

*CT Coronary Angiogram*

CT coronary angiography was performed as per local criteria at our institution using a 64-slice CT scanner. A resting heart rate of less than 80 beats per minute was required. Intravenous metoprolol was administered for heart rate reduction if necessary.

*Coronary Height Analysis*

Using an electronic radiology reporting program (Agfa IMPAX™) and a measuring calliper, distance from the upper rim of the CT table to multiple points in the coronary tree were obtained. Arterial measurement points included;

1. Left coronary ostium
2. Right coronary ostium
3. Ostial left anterior descending (LAD)
4. Distal LAD - at its highest point
5. Distal circumflex (Cx) - at its lowest point
6. Right coronary artery bifurcation
7. Distal posterior descending artery (PDA) - at its highest point
8. Distal posterior left ventricular artery (PLV) - at its lowest point

Measurements were in millimetres and taken at the furthest point of contrast penetration visible in the vessel.

*FFR Impact Analysis*

The difference in height between the coronary ostium and the measurement point in the artery is the calculated height difference. This was multiplied by 0.8 (according to Pascal's Law and adjusting for blood density) to give a positive or negative change in pressure - in mmHg. This is the theoretical effect on Pd. The denominator (Pa) is assumed to be 100 in the following calculation model. The resulting value was factored into 200 random computer generated FFR values between 0.75 and 0.85 to give a corrected FFR (cFFR) using Microsoft Excel™. Corrected FFR was compared with baseline FFR and the percentage of values that crossed the threshold of 0.8 (from positive to negative or vice versa) was calculated.

*Statistical Analysis*

Continuous variables are expressed as mean values plus or minus standard deviation. Categorical variables are described as numbers and percentages. Statistical significance of coronary height variations were calculated using the Student *t-test*.

**Results**

*Study Population*

Patient demographics are summarised in table 1.

All patients had a resting heart rate below 80 beats per minute before scanning.

*Coronary Height Data*

Figure 1 shows an example of coronary height measurement. The measuring calliper in green calculates height from the upper rim of the CT table to the corresponding point in the coronary artery. In this particular example the calliper is measuring from ostial left main stem.

Results are displayed below of all measurement points within the coronary tree (Table 2, Figure 2). Height measurement is taken from the upper rim of the CT table.

Table 3 summarises data points from each coronary artery with regard to their respective coronary ostia. The height difference between the coronary specific coronary ostium (Pa) and the vessel containing the height measurement point (Pd), is the value used to calculate effect on FFR and hence, the cFFR.

*Hydrostatic effect and cFFR*

The corresponding hydrostatic effect of distal LAD, distal Cx, distal PDA and distal PLV were factored into the FFR equation to give the cFFR (Table 3). For anterior vessels, the FFR increased, for posterior vessels, it fell. Out of the 200randomly generated FFR values, 45.5% were below 0.8 and 55.5% above. After correction and calculation of cFFR, these percentages changed substantially. Those that crossed from positive to negative, or vice versa were calculated. Table 4 summarises the results.

*Clinical Case Example*

An *in vivo* example demonstrating the effect of wire position is presented of a 73-year old male with a lesion in the mid right coronary artery (RCA) (figure 3). The patient presented with typical stable angina. There is a background history of inflammatory bowel disease, but no typical cardiac risk factors. Ejection fraction was normal. A combined pressure and velocity wire (Combowire, Volcano Corporation™, San Diego, California, USA) is passed through a 6F guiding catheter. The wire is passed beyond the lesion and FFR is measured firstly in the PDA (as distal as a clear velocity tracing allowed), followed by the PLV (distally as per PDA) and lastly placed 3 vessel diameters beyond the stenosis in the main mid RCA. 400 micrograms of intra-arterial nitrates were administered before FFR measurement. Intravenous adenosine at 140mcg/kg was used to induce a steady state of hyperaemia. There was no drift with any of the acquired measurements. Invasive measurements are presented in Table 5.

For the same lesion, placement of the wire in the PDA or PLV altered FFR by 0.05. Placing the wire 3 vessel diameters beyond the stenosis, gives an FFR of 0.79. The small flow variations measured on each occasion are not significantly different, and within normal variations expected during doppler measurements [8].

**Discussion**

In summary, our findings show that coronary anatomy results in statistically significant height variations between proximal (Pa) and distal vessel (Pd). There is a potential change in FFR of 0.02-0.05, causing a number of 'grey-zone' FFR results to cross a binary cut-off point.

In our cohort, the most superior points in a supine patient were the distal LAD, followed by distal PDA. The most inferior points were the distal Cx and distal PLV. All measurements were statistically significant when compared to the respective ostium, apart from the ostial LAD. Even though the mean height of PLV and Cx were identical with reference to the CT table, when compared to their respective ostium (Pa), the PLV had a larger height difference, owing to the more superior position of the RCA ostium. In turn, the hydrostatic pressure effect was more pronounced in the PLV. More proximal points in a vessel, e.g ostial LAD or RCA bifurcation had a smaller height variation when compared to their respective coronary artery ostium. In general there is a gradual change in height from proximal to distal vessel. Note however, that the most distal point in the vessel does not always have the greatest height variation. An example of this is in a 'wrap around' LAD, where the vessel height falls after reaching the apex. This occurs in over half of patients in one study [9].

CT coronary angiography can accurately map the course of coronary vessels and their vertical heights. Subsequently, the height of the distal vessel (i.e the position of the pressure wire, or Pd) may be higher, or lower than its origin (Pa), depending on the course it takes. This may explain observed changes in groups of patients with 'moderate' coronary stenoses in which posterior vessels (those vertically lower when supine - Circumflex, Posterior left ventricular) have higher mean FFR values than anterior vessels (those that are vertically higher - left anterior descending, posterior descending) [10]. Resting Pd/Pa can also often be seen above 1.0. Studies have identified this phenomenon [6] and it is caused by the distal pressure sensor sitting vertically lower than the aortic pressure sensor (and original point of normalisation). For a resting index to be above one, disease in the vessel is usually mild. While often attributed to drift, physical principles can predict this concept. It is useful to note this phenomenon rather than assume the physiology wire is at fault.

A recent study assessing coronary artery height variations using CT coronary angiograms has been conducted recently in a group composed predominantly of transcutaneous aortic valve implantation (TAVI) patients [5]. Hydrostatic pressure effects were then confirmed using an *in vitro* model. The anatomy of these patients with severe aortic stenosis may slightly alter the anatomy of the coronary arteries themselves due to changes in the aortic root. Our assessment of coronary height variations in a more heterogeneous group of patients presenting with stable cardiac chest pain was thought to be a useful addition to current knowledge. In general, our patients were younger females in keeping with the low to intermediate risk group initially assessed with CT coronary angiography at the time. There were some differences in height measurements from CT scans between our study and Härle et al. Measurements from ostial left coronary artery to LAD and Cx were similar (5.3cm vs. 4.9 and 3.4 vs. 3.9 respectively). There were however more pronounced differences in the measurement of PLV and PDA from the right coronary ostium (5.7 vs. 2.6 and 1.8 vs. 3.8). There are potential explanations. Observer variation between two studies may account for some of the change. Contrast penetration into the distal vessel can significantly alter the measurement point within the artery, leading to error in measurements in both studies. Finally, the patient cohort varies between the studies. One anticipates that coronary height measurements may vary between a predominantly older population with aortic stenosis, and a younger cohort without.

Pressure based invasive physiology such as FFR, has been well validated over many years. However, pressure-based measurements are subject to the potential effects of hydrostatic pressure. If hydrostatic forces alter distal pressure recordings FFR will in turn change. The change may be small (0.02 - 0.05) but useful to know in FFR values circling the cut-off point (0.75-0.85) [11]. In theory, the addition of adenosine should not alter the physical hydrostatic pressure effect in a coronary vessel *in vivo*, as height, fluid density and gravitational effect have not changed. An important consideration is the hypotensive effect and hence reduction in Pa during adenosine infusion. Pa pressure may fall below 100mmHg during hyperaemia, meaning alterations in Pd have a larger effect on overall Pd/Pa. Hydrostatic effect is constant across resting and hyperaemic states. A change in Pd of 5mmHg is therefore of greater relative importance in resting indices (where a transtenotic gradient of 10mmHg is considered abnormal) compared to hyperaemic indices (where 20mmHg is considered abnormal)

Whilst the effect of hydrostatic pressure upon FFR is described, we believe that this novel data demonstrates that depending on the coronary artery in question and its anatomical course the physiological significance of a coronary stenosis can be both over or under-estimated. Treatment of intermediate coronary stenoses therefore must not be a binary decision, and the operator must exert clinical judgment when faced with grey zone physiology values.

The exact position of the pressure sensor of the physiology wire is often not considered. Hydrostatic effect becomes more pronounced as the pressure sensor is positioned more distally. Avoiding an unnecessarily distal wire position will minimise the hydrostatic effect on obtained measurements by reducing the guide to pressure sensor distance.

By changing patient position during angiography, (i.e turning onto one side), and leaving the wire in exactly the same position in the artery, FFR values have been shown to change [12]. Correcting for the presumed hydrostatic effect due to this position change (by using measured height difference between guide and wire), abolished the difference between the two FFR recordings, seemingly explaining the difference.

Another important observation is the pressure change along the longitudinal length of a coronary artery, which has been attributed to diffuse atherosclerosis [13]. The additive effect of hydrostatic pressure however cannot be excluded, as vertical height also gradually changes along the length of an artery. This along with other confounding factors, such chronic kidney disease [14] may also impact stenosis assessment. Finally, hydrostatic pressure effects may also contribute to measurements that use mean distal pressure, such as the index of microvascular resistance (IMR) measured using thremodilution.

In our clinical case example, wire placement altered FFR by 0.05 (PDA vs. PLV placement). Flow within the artery does not change in our case study as coronary autoregulation maintains flow over a wide pressure range when these mechanisms are intact [15] . Using our coronary CT data, the mean height difference between the PLV and PDA was 7.57cm, equating into a potential distal pressure difference (Pd) of 6.06mmHg. Therefore a change in FFR of up to 0.06 is possible on average. Of course this is a mean change, and patient factors such as height, play a role in individual FFR measurements [7]. Although clinical decision-making takes into account multiple factors and is not a binary process revolving around a cut-off point, one should recognise the potential effects of wire position and hydrostatic pressure.

**Study Limitations**

The study group consisted of low to intermediate risk patients, meaning the majority were younger females. This is not in keeping with a typical demographics of patients who require invasive treatment for coronary artery disease.

The visualisation of the coronary artery in question was limited by contrast penetration into the distal vessel. Some vessels were not completely opacified, meaning a potential underestimation of height measurements. This seemed especially prominent in the PDA where contrast did not penetrate to the distal vessel in 15% of cases. Measurements for these patients were excluded.

Height was measured at distal sections in the coronary artery, as this was the point of maximal height variation. In clinical practice the wire is often not positioned as far distal as these measurements were taken, meaning a potential overestimation of the hydrostatic effect.

With regards to the 200 random FFR results generated, it can be seen that 54.5% of FFR values generated were over 0.8. This was obviously a chance occurrence, but the lack of a more linear 50/50 split of values will effect subsequent analysis.

The hydrostatic effect on FFR in this study takes into account a Pa pressure of 100mmHg.Further data on alterations in Pa and the subsequent impact on FFR may have been a useful addition.

The calculated hydrostatic effect is theoretical, and needs further investigation *in vivo*. Recent trials have upheld anticipated changes in pressure based measurements due to hydrostatic forces [12].

**Conclusion**

The anatomical path of coronary arteries results in a significant vertical height difference between the distal artery (Pd) and its point of normalisation (Pa). According to our hydrostatic pressure model, this is likely to have a modest effect on FFR calculation, which in turn could result in values crossing the treatment threshold. Operators should be mindful of this phenomenon when interpreting FFR values, particularly in the LAD and RCA-PLV.

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**Statement of Competing Interests**

The authors report no competing interests

**List of Abbreviations**

FFR - Fractional Flow Reserve

Pd - Distal Pressure

Pa - Proximal Pressure

cFFR - corrected FFR

iFR - instantaneous wave free ratio

TAVI - transcutaneous aortic valve implantation

**References**

1. Zimmermann FM, Ferrara A, Johnson NP, van Nunen LX, Escaned J, Albertsson P, et al. Deferral vs. performance of percutaneous coronary intervention of functionally non-significant coronary stenosis: 15-year follow-up of the DEFER trial. Eur Heart J. 2015 Dec 1;36(45):3182–8.

2. Pijls NHJ, Fearon WF, Tonino PAL, Siebert U, Ikeno F, Bornschein B, et al. Fractional flow reserve versus angiography for guiding percutaneous coronary intervention in patients with multivessel coronary artery disease: 2-year follow-up of the FAME (Fractional Flow Reserve Versus Angiography for Multivessel Evaluation) study. J Am Coll Cardiol. 2010 Jul 13;56(3):177–84.

3. Xaplanteris P, Fournier S, Pijls NHJ, Fearon WF, Barbato E, Tonino PAL, et al. Five-Year Outcomes with PCI Guided by Fractional Flow Reserve. N Engl J Med. 2018 May 22;

4. Davies JE, Sen S, Dehbi H-M, Al-Lamee R, Petraco R, Nijjer SS, et al. Use of the Instantaneous Wave-free Ratio or Fractional Flow Reserve in PCI. N Engl J Med. 2017 11;376(19):1824–34.

5. Tebaldi M, Biscaglia S, Fineschi M, Musumeci G, Marchese A, Leone AM, et al. Evolving Routine Standards in Invasive Hemodynamic Assessment of Coronary Stenosis: The Nationwide Italian SICI-GISE Cross-Sectional ERIS Study. JACC Cardiovasc Interv. 2018 Aug 13;11(15):1482–91.

6. Nijjer SS, de Waard GA, Sen S, van de Hoef TP, Petraco R, Echavarría-Pinto M, et al. Coronary pressure and flow relationships in humans: phasic analysis of normal and pathological vessels and the implications for stenosis assessment: a report from the Iberian-Dutch-English (IDEAL) collaborators. Eur Heart J. 2016 Jul 7;37(26):2069–80.

7. Härle T, Luz M, Meyer S, Kronberg K, Nickau B, Escaned J, et al. Effect of Coronary Anatomy and Hydrostatic Pressure on Intracoronary Indices of Stenosis Severity. JACC Cardiovasc Interv. 2017 Apr 24;10(8):764–73.

8. Davies JE, Whinnett ZI, Francis DP, Manisty CH, Aguado-Sierra J, Willson K, et al. Evidence of a dominant backward-propagating ‘suction’ wave responsible for diastolic coronary filling in humans, attenuated in left ventricular hypertrophy. Circulation. 2006 Apr 11;113(14):1768–78.

9. Kobayashi N, Maehara A, Brener SJ, Généreux P, Witzenbichler B, Guagliumi G, et al. Usefulness of the Left Anterior Descending Coronary Artery Wrapping Around the Left Ventricular Apex to Predict Adverse Clinical Outcomes in Patients With Anterior Wall ST-Segment Elevation Myocardial Infarction (from the Harmonizing Outcomes With Revascularization and Stents in Acute Myocardial Infarction Trial). Am J Cardiol. 2015 Dec 1;116(11):1658–65.

10. Härle T, Meyer S, Bojara W, Vahldiek F, Elsässer A. Intracoronary pressure measurement differences between anter﻿ior and posterior coronary territories. Herz. 2017 Jun;42(4):395–402.

11. Petraco R, Escaned J, Sen S, Nijjer S, Asrress KN, Echavarria-Pinto M, et al. Classification performance of instantaneous wave-free ratio (iFR) and fractional flow reserve in a clinical population of intermediate coronary stenoses: results of the ADVISE registry. EuroIntervention. 2013 May 20;9(1):91–101.

12. Härle T, Luz M, Meyer S, Vahldiek F, van der Harst P, van Dijk R, et al. Influence of hydrostatic pressure on intracoronary indices of stenosis severity in vivo. Clin Res Cardiol. 2018 Mar;107(3):222–32.

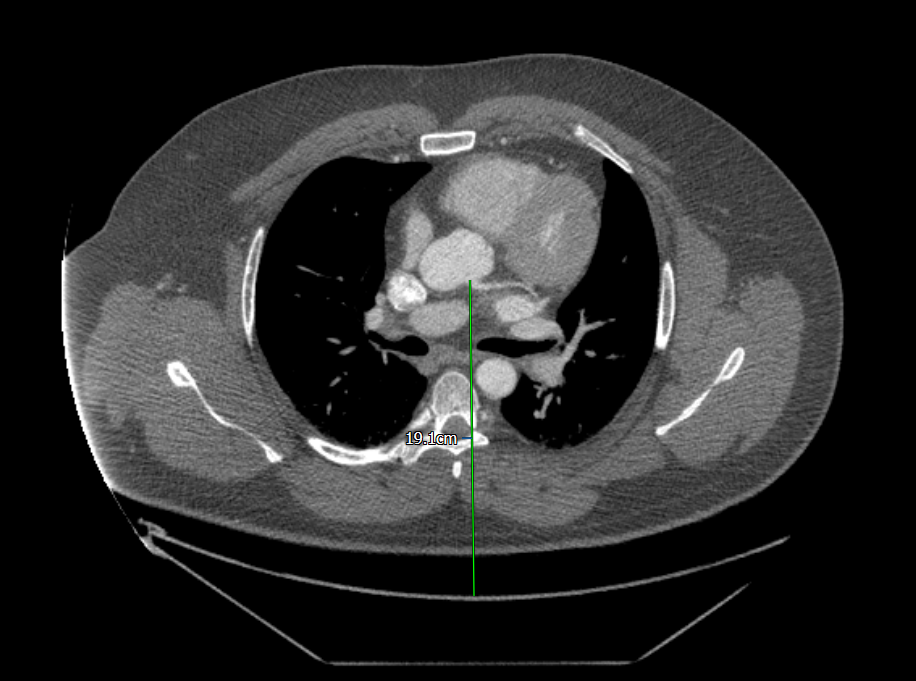
13. Bruyne BD, Hersbach F, Pijls NHJ, Bartunek J, Bech J-W, Heyndrickx GR, et al. Abnormal Epicardial Coronary Resistance in Patients With Diffuse Atherosclerosis but “Normal” Coronary Angiography. Circulation. 2001 Nov 13;104(20):2401–6.

14. Tebaldi M, Biscaglia S, Fineschi M, Manari A, Menozzi M, Secco GG, et al. Fractional Flow Reserve Evaluation and Chronic Kidney Disease: Analysis From a Multicenter Italian Registry (the FREAK Study). Catheter Cardiovasc Interv. 2016 Oct;88(4):555–62.

15. Ramanathan T, Skinner H. Coronary blood flow. Contin Educ Anaesth Crit Care Pain. 2005 Apr 1;5(2):61–4.

**Figure Legend**

Figure 1

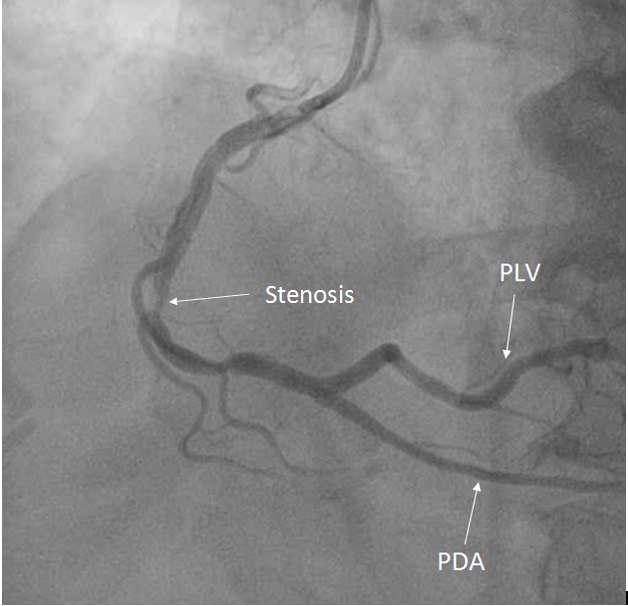


Vessel height measurement illustration on coronary CT  
The image demonstrates the measurement calliper from the left main stem ostium, to the upper rim of the CT table

Figure 2

Coronary height variation from their respective ostium  
Figure 2 demonstrates the height variation of the distal vessel from its respective ostium. \*\* These measurements were statistically significant.

Figure 3



Mid right coronary artery stenosis  
The stenosis is shown in the mid right coronary artery, with arrows indicating the PLV and PDA.

**Table Legend**

Table 1

|  |  |
| --- | --- |
| Characteristic | Number (also % as n=100) |
| Age | 55.9 |
| Female | 68 |
| Current smoker | 12 |
| Ex-smoker | 19 |
| Hypertension | 33 |
| Hypercholesterolaemia | 25 |
| Family History | 24 |
| Ejection Fraction | 54.8% |

Patient Demographics

Demographics of 100 study patients

Table 2

|  |  |  |
| --- | --- | --- |
| Measurement Point | Mean height from Upper Rim of CT Table (mm) (Standard Deviation in mm) | P Value compared to vessel ostium |
| *Left Coronary Circulation* |  |  |
| LCA Ostium | 170.0 (+/- 19.6) | N/A |
| LAD Ostium | 167.9 (+/-19.6) | 0.06 |
| Distal LAD | 222.5 (+/- 28.3) | <0.0001 |
| Distal Cx | 136.4 (+/- 20.4) | <0.0001 |
| *Right Coronary Circulation* |  |  |
| RCA Ostium | 193.8 (+/- 21.1) | N/A |
| RCA bifurcation | 175.6 (+/- 28.3) | <0.0001 |
| Distal PDA | 212.1 (+/-30.7) | <0.0001 |
| Distal PLV | 136.4 (+/-26.1) | <0.0001 |

CT Height measurements

The vertical height measurements are shown from the upper rim of the CT table. P values are calculated for each point to the respective vessel ostium.

Table 3

|  |  |  |  |
| --- | --- | --- | --- |
| Measurement Point | Height from respective coronary ostium (mm) | Height effect on distal pressure (Pd) - mmHg | FFR Correction factor |
| *Height from Left Coronary Ostium* |  |  |  |
| LAD Ostium | +2.1 | -0.2 | **+0.002** |
| Distal LAD | +52.5 | +4.2 | **+0.04** |
| Distal Cx | -33.6 | -2.7 | **-0.03** |
| *Height from Right Coronary Ostium* |  |  |  |
| RCA bifurcation | -18.2 | -1.5 | **-0.02** |
| Distal PDA | +18.3 | +1.5 | **+0.02** |
| Distal PLV | -57.4 | -4.6 | **-0.05** |

FFR effect

The height variations have been converted into pressure effect in mmHg. The impact on FFR with a Pa of 100 is shown in the far right column.  
  
Table 4

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| Vessel point  (+change in Pd pressure) | % FFR below 0.8 | % FFR above 0.8 | % cFFR below 0.8 | % cFFR above 0.8 | % Crossing 0.8 |
| Distal LAD  (-0.04) | 45.5 | 54.5 | 6 | 94 | 42.5 |
| Distal Cx  (+0.03) | 72 | 28 | 26.5 |
| Distal PLV  (+0.05) | 92 | 8 | 46.5 |
| Distal PDA  (-0.02) | 30.5 | 69.5 | 15 |

Effect on FFR measurements between 0.75 and 0.85  
The effect on 200 randomly generated FFR measurements is shown for each vessel point. % values crossing a threshold of 0.8 is shown in the far right column

Table 5

|  |  |  |
| --- | --- | --- |
| Measurement point | FFR | Flow (cm/s) |
| PDA | 0.75 | 17.1 |
| PLV | 0.8 | 19.1 |
| 3 vessel diameters beyond stenosis (mid RCA) | 0.79 | 18.6 |

Clinical case data

The data from the clinical case described is shown in table 5. FFR measurement varied by 0.05 between PLV and PDA. Velocity measurements did not vary significantly. This is due to the vertical height differences in both vessels and in turn the hydrostatic effect.