1. Introduction

Intravascular ultrasound is a clinical tool that has been used in a complimentary manner to contrast angiography in order to enhance the procedure success rate and patient outcomes. Particularly, there are some specific situations where IVUS can be very useful and information from IVUS overcome angiography limitations. These situations can be divided into three parts:

- pre-interventional
- during intervention
- post-interventional

Pre-interventional use of IVUS can help with assessment of hemodynamic significance of stenosis (in this place must be emphasized, that IVUS can estimate hemodynamic significance only non directly, but with acceptable correlation with fractional flow reserve and nuclear stress test), precise anatomic analysis (type of bifurcation, plaque burden in ostial part of side branch or left main) and can help with device selection: precise measurement of diameters in reference segments for better sizing of balloon or stent, type of lesion preparation such as dilatation with cutting balloon or atherectomy devices in bulky lesions, drug eluting stent for lesions with high probability of small in stent area achievement.

IVUS can be used during intervention for assessment of lesion preparation (proper position of wire; especially it’s relation to stent struts, effect of predilatation, assessment of side branch ostium) for guiding in more complicated intervention (left main stenting, navigation of wires during PCI of chronic total occlusion, trifurcation PCI).

Post-interventional use of IVUS can answer questions about result adequacy (stent expansion, stent apposition), angiographic filling defects (“hazy lesions”) after PCI (edge dissection, thrombus formation, inadequate stent expansion, prominent calcification) and diagnosis of complications (dissection, geographic mismatch, plaque protrusion inside the stent, inadequate stent expansion or apposition). IVUS can in most cases also reveal the reasons behind development of complications after PCI such is in stent thrombosis (mainly inadequate stent expansion or edge troubles such as dissection, or uncovered lesion) or unexpected early in stent restenosis (inadequate stent expansion).

IVUS is a mandatory tool in the cardiac catheterization laboratory today. Like all medical equipments it should be used by an experienced investigator for better understanding of PCI mechanisms, prevention and solution of complications. Nowadays it is possible to use many semi-automatic softwares for border detection, but all of them must be corrected.
manually to avoid serious mistakes and misunderstanding. The best way how to do that is just routine use of IVUS.

1.1 Limitation of angiography
Angiography is a gold standard for assessment of atherosclerotic impairment of coronary arteries and for guiding of coronary intervention. On the other hand it has many limitations. Widely accepted decision making point for diagnosis of flow-limiting lesion is lumen diameter less than 50% of the reference segment. However, it has been proven that there is no correlation between angiography assessment and functional measurement by fractional flow reserve (FFR) in intermediate lesions between 40-70%\(^1\).

Based on autopsy studies we know, that atherosclerosis is more often diffuse than focal process affecting coronary arteries. These findings are in good correlation with intravascular ultrasound. The reference segment, which is angiographically normal often contains atherosclerotic plaques visible on IVUS. From this point of view we are comparing less affected segments with more affected ones during angiography and thus underestimate stenosis significance\(^2,3\). Other challenging issues are assessment of ostial lesions and bifurcations.

2. Quantitative lesion assessment

2.1 Technique
The most important parameter for quantitative lesion assessment is minimal lumen area (MLA), which has the best correlation with presence of ischemia (see below). Common misunderstanding comes from the assessment of relative severity of stenosis in percentage of “normal” reference diameter. Angiographic stenosis assessment is based on comparison of lumen diameter in reference segment and lumen diameter in the lesion. The most corresponding IVUS parameter is lumen area stenosis (LAS), which is calculated as minimal lumen area in reference segment – minimal lumen area in lesion / minimal lumen area in reference segment. More frequently used description of stenosis is plaque burden (PB), computed as external elastic membrane area – lumen area / external elastic membrane area\(^4\). We must interrogate each frame and look for the smallest lumen area, use routinely nitrates before IVUS probe insertion for avoiding spasms. In case of uncertainty in lumen measurement, especially in present of soft plaque it is recommended to flush guiding catheter by saline for clearing of picture. Using of automatic pullback devices is not necessary for quantitative lesion assessment, but allows us to perform longitudinal diameters or volumetric analysis. Manual pullback is better for analysis of a precise part of plaque, where we can stop IVUS probe to obtain more frames from the region of interest. For imaging of aorto-ostial lesions it is necessary to retract the guiding catheter back into the aorta and verify that the path of the IVUS probe is still coaxial with the ostium of the vessel\(^5\).

2.2 Indication for coronary intervention
The main indication for coronary intervention is lesion producing myocardial ischemia, which can be detect either non-invasively by stress myocardial perfusion imaging (SPECT) or invasively by measurement of pressure gradient across stenosis (fraction flow reserve, FFR). IVUS measurement correlates with both of them (table one).
<table>
<thead>
<tr>
<th>Author</th>
<th>Description</th>
<th>Methods</th>
<th>No of patients</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Briguori⁶</td>
<td>angiographic stenosis 40-70%</td>
<td>IVUS vs. FFR</td>
<td>43</td>
<td>FFR &lt; 0.75 correlates with:</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- MLA ≤ 4 mm²</td>
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<td></td>
<td></td>
<td>- MLD ≤ 1.8 mm</td>
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<td></td>
<td></td>
<td>- PB &gt; 70%</td>
</tr>
<tr>
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<td></td>
<td></td>
<td>- Lesion length &gt; 10 mm</td>
</tr>
<tr>
<td>Abizaid⁷</td>
<td>patients indicated for PCI</td>
<td>IVUS vs. FFR</td>
<td>73</td>
<td>CFR ≥ 2 correlates with:</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- MLA ≥ 4 mm²</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- MLD ≥ 2 mm</td>
</tr>
<tr>
<td>Nishioka⁸</td>
<td>consecutive IVUS examinations</td>
<td>IVUS vs. myocardial SPECT</td>
<td>79</td>
<td>Positive scan correlates with:</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- MLA ≤ 4 mm²</td>
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<td></td>
<td></td>
<td></td>
<td>- PB &gt; 73%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- LAS &gt; 59%</td>
</tr>
<tr>
<td>Takagi⁹</td>
<td>consecutive IVUS examinations</td>
<td>IVUS vs. FFR</td>
<td>42</td>
<td>FFR &lt;0.75 correlates with:</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- MLA ≤ 3 mm²</td>
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<tr>
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<td></td>
<td></td>
<td></td>
<td>- LAS &gt; 60%</td>
</tr>
<tr>
<td>Lee¹⁰</td>
<td>vessels smaller than 3 mm</td>
<td>IVUS vs. FFR</td>
<td>94</td>
<td>FFR &lt; 0.75 correlates with:</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- MLA ≤ 2.0 mm</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- PB ≥ 80%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- Lesion length ≥ 20 mm</td>
</tr>
<tr>
<td>Abizaid¹¹</td>
<td>IVUS deferred PCI</td>
<td>IVUS</td>
<td>300</td>
<td>Safe deferral of PCI correlates with:</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- MLA ≥ 4 mm²</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- MLD ≥ 2 mm</td>
</tr>
<tr>
<td>Jasti¹²</td>
<td>ambiguous left main stenosis</td>
<td>IVUS vs. FFR</td>
<td>55</td>
<td>FFR &lt; 0.75 correlates with:</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- MLA ≤ 5.9 mm</td>
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<tr>
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<td></td>
<td></td>
<td></td>
<td>- MLD ≤ 2.8 mm</td>
</tr>
<tr>
<td>Abizaid¹³</td>
<td>patients with borderline left main stenosis</td>
<td>IVUS</td>
<td>122</td>
<td>MACE predictor :</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- MLD ≤ 3.0 mm</td>
</tr>
<tr>
<td>Kang¹⁴</td>
<td>Consecutive patients</td>
<td>IVUS vs. FFR</td>
<td>201</td>
<td>FFR &lt;0.80 correlates with:</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- MLA 2.4 mm²</td>
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<td></td>
<td></td>
<td></td>
<td>- PB ≥ 79%</td>
</tr>
<tr>
<td>Ben Dor¹⁵</td>
<td>patients with intermediate lesion during QCA</td>
<td>IVUS vs. FFR</td>
<td>84</td>
<td>FFR &lt; 0.8 correlates with:</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- MLA 2.4 mm² for vessels 2.5-3.0 mm</td>
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<td></td>
<td></td>
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<td></td>
<td>- MLA 2.7 mm² for vessels 3.0-3.5 mm</td>
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<td></td>
<td></td>
<td></td>
<td>- MLA 3.6 mm² for vessels &gt;3.5 mm</td>
</tr>
<tr>
<td>Ahn¹⁶</td>
<td>consecutive patients with SPECT and IVUS</td>
<td>IVUS vs. SPECT</td>
<td>150</td>
<td>Positive scan correlates with:</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- MLA 2.1 mm²</td>
</tr>
<tr>
<td>PROSPECT¹⁷</td>
<td>clinical follow-up after ACS</td>
<td>IVUS and SPECT</td>
<td>700</td>
<td>Predictors for MACE:</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- PB &gt; 70%</td>
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<tr>
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<td></td>
<td></td>
<td>- MLA &lt; 4 mm²</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- TCFA</td>
</tr>
</tbody>
</table>

Abbreviations: CFR - coronary flow reserve, MACE – major cardiac adverse event, MLA – minimal lumen area, MLD – minimal lumen diameter, PB - plaque burden, PCI- percutaneous coronary interventions, QCA – quantitative coronary angiography, SPECT – single photon emission computed tomography, TCFA thin cap fibroatheroma

Table 1. Studies with IVUS evaluation of hemodynamic significance
Based on older studies the following recommendations for IVUS detection of significant stenosis were established:

Main epicardial artery:
- MLA < 4 mm²
- MLD < 2 mm

Left main:
- MLA < 6 mm²
- MLD < 3 mm

However, recently published studies do not support these cut-off values. Instead of clear cut-off points there are number of different recommended values, which do not seem to be useful for routine practice. Based on these findings we must conclude that IVUS is not suitable for assessment of haemodynamic significance of intermediate lesions. IVUS can be only used for exclusion of haemodynamically significant lesion with MLA more than 4 mm² for main epicardial arteries and more than 6 mm² for left main. The haemodynamic significance of each lesion is caused not only by lumen area, but also by amount of viable myocardium supplied by this vessel and by presence or absence of collaterales. These facts simply cannot be examined solely by a morphologic modality like IVUS. On the other hand the PROSPECT trial with clinical endpoints confirmed, that large plaque compromising lumen to 4 mm² and less, especially together with higher content of necrotic tissue is a risk factor for future events. These issues will be matter of further studies.

There are no data about IVUS criteria for hemodynamic significance in saphenous vein graft, but it is recommend to use a cut-off point MLA 4 mm² for graft supplying one coronary artery and MLA 6 mm² for graft supplying two arteries.

The precise assessment of hemodynamic significance is a crucial point, because the rate of ischemic events is 5-10% / year in significant lesion and less than 1%/ year in non-ischemic lesion. Performing PCI in non-ischemic lesion increase risk of event to 2-3% / year and furthermore increases risk of periprocedural ischemia or myocardial necrosis during PCI.

Very old IVUS-skeptic sentence “If you want to stent, do IVUS” was quite recently supported by the work of Nam et al. Authors randomized 167 patients with intermediate coronary lesion between FFR guided (cut-off for PCI was FFR <0.8) and IVUS guided (cut-off MLA< 4.0 mm²) coronary intervention. PCI was performed in 33.7% lesion in FFR arm and in 91.5% in IVUS arm. This difference was highly statistically significant (p<0.001). On the other hand we ourselves have a different experience with IVUS assessment in borderline lesions. We estimate that we are performing PCI in 40-50% of borderline lesions based on IVUS criteria, which is closer to FRR guided arm in this study. The finding of 91.5% frequency of MLA < 4 mm² in lesion between 40-70% is in our eyes unrealistic.

2.3 Assessment of left main (LM)

Left main stenosis is a very important predictor for future cardiac events. Angiographic assessment of LM is often complicated for overlapping branches and short or no reference segment and can lead to inappropriate estimates of lesion severity. IVUS is more sensitive for left main atherosclerosis than angiography. Suter et al. found that in half of the patients with an inconclusive angigram IVUS detects a significant stenosis. There is no difference for left main assessment during pullback from left anterior descending artery (LAD) or left circumflex artery (LCX). On the other hand for accurate assessment of ostial part of LAD and LCX is necessary to perform two pullbacks from both daughter vessels,
because oblique view can overestimates lumen area\textsuperscript{25}. The main target in left main bifurcation (like in other bifurcation) is to make a decision between one or two stent strategy. Pullback from just one daughter artery can answer the question whether the ostium of second branch is affected or not. For a more precise evaluation of minimal lumen area it is necessary to perform pullback from both daughter branches. The precise coaxial position of guiding catheter is important for measurement or ostial left main stenosis, other than coaxial position of guiding catheter overestimates the lumen area. This type of inappropriate measurement can be detected by elliptical instead of spherical shape of ostial left main.

The ostium of LM can be influenced not only by atherosclerosis but also by external compression between enlarged pulmonary artery and aorta. This compression occur during systolic phase and lumen is enlarged during diastolic phase (figure 1,2). This finding can be seen in patients with severe pulmonary artery hypertension.

According to our experience we recommend to perform all PCI’s of LM with IVUS guidance. This is supported by results of MAIN-COMPARE registry, where IVUS guidance of left main PCI was superior to angiographic guidance\textsuperscript{26}.

Fig. 1. External compression of left main during systolic phase of cardiac cycle. “Kmen ACS” means left main coronary artery.

Fig. 2. No evidence of left main compression during diastolic phase of cardiac cycle.
3. Lesion morphology

3.1 IVUS in lesion with angiographic filling defect “hazy lesions”

The main finding in so-called hazy lesions is a defect in the contrast filling of coronary artery. The reasons for this include:

- eccentric calcification (figure 3)
- significant stenosis
- dissection (figure 4)
- thrombosis (figure 5)
- plaque rupture (figure 6)
- “flow phenomenon” (inadequate filling of big arteries during dye injection).

Thrombus is the most dangerous cause and many hazy lesions are treated like thrombus-containing lesions with administration of IIbIIIa glycoprotein inhibitors, use of embolic protection devices or covered stents. However, real presence of intracoronary thrombosis is 50-60% of all hazy lesions\(^{27}\). Sensitivity of IVUS for thrombus is low (about 50%), so from IVUS picture we simply cannot rule out the presence of thrombus (the highest sensitivity for thrombus has optical coherence tomography), but IVUS can confirm other causes and mainly causes which are not indicated for PCI. This strategy decreases frequency of PCI in hazy lesions to 15-20%\(^{28-30}\).

Fig. 3. Eccentric calcification.

Fig. 4. Dissection with visible tear (arrow) and small thrombus (in circle).
Fig. 5. Huge thrombus located on very small plaque causing acute coronary syndrome. Left side is cross sectional view, right side is longitudinal view (thrombus is located in the circle).

Fig. 6. Plaque rupture with visible cavity after embolization of plaque mass. Patient with acute myocardial infarction.

3.2 Aneurysms

True aneurysm is defined as both an external elastic membrane (EEM) and lumen area 50% larger than the proximal reference segment with intact three layers of vessel wall (figure 7). Lesions which seem to be the aneurysms from angiographic assessment are an true aneurysm in 37% of such findings. The rest are normal segments adjacent to plaque (53%), complex atherosclerotic plaque (16%) and pseudoaneurysm (4%)\textsuperscript{31}. Coronary pseudoaneurysm is due to rupture of vessel wall and in IVUS picture the three layers of vessel are not present, the shape of pseudoaneurysm is often irregular. From a practical point of view, pseudoaneurysms are seen only after coronary interventions causing trauma of vessel wall.
3.3 True versus false lumen
False lumen is created by dissection, either spontaneous or iatrogenic during coronary intervention (wire insertion or after balloon/stent dilatation). Dissections are treated by stent implantation and a flow non limiting dissection can be let to spontaneous healing. Stent insertion to the true lumen is a crucial point for proper treatment of dissection. True lumen can be identified by three-layered appearance and by origin of side branches. False lumen contains more echogenic blood flow\(^5\) (figure 8).

4. Balloon angioplasty
4.1 The mechanism of balloon PCI
The main mechanism of lumen enlargement during balloon dilatation is a plaque rupture, which enables lumen dilatation\(^3^{2}\). This finding was also confirmed by IVUS during in vivo studies, mainly in eccentric plaques (which are mainly present in coronary arteries). Another mechanism of lumen enlargement during balloon angioplasty is plaque compression and plaque redistribution, which are the main mechanism in concentric plaques\(^3^{3}\).
## 4.2 IVUS guided plain balloon PCI

Several studies have been conducted in this field. Their results are summarized in table 2.

<table>
<thead>
<tr>
<th>study</th>
<th>target</th>
<th>restenosis</th>
<th>MACE</th>
<th>comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>CLOUT(^{34}) 102 pts.</td>
<td>using larger balloon than angiographically measured vessel diameter</td>
<td>not declared</td>
<td>1.9(^{\circ})</td>
<td>plaques compensated by positive remodeling allow use of aggressive dilatation without risk of significant dissections</td>
</tr>
<tr>
<td>Haase(^{35}) 144 pts.</td>
<td>IVUS guided balloon dilatation</td>
<td>21(^{\circ})</td>
<td>12(^{\circ})</td>
<td>use of larger balloon than angiographic lumen diameter is safe and with low risk for restenosis</td>
</tr>
<tr>
<td>Schroeder(^{36}) 252 pts.</td>
<td>IVUS guided balloon dilatation</td>
<td>19(^{\circ})</td>
<td>14(^{\circ})</td>
<td>small dissection are not flow limiting IVUS decreases number of implanted stents low restenosis comparable with BMS</td>
</tr>
<tr>
<td>Abizaid(^{37}) 284 pts.</td>
<td>IVUS guided balloon dilatation and stent only in case of unsatisfactory result</td>
<td>8(^{\circ}) - PCI 16(^{\circ}) - stent</td>
<td>8(^{\circ}) - PCI 11(^{\circ}) - stent</td>
<td>„stent like“ effect after IVUS guided balloon dilatation has similar rate restenosis like a BMS</td>
</tr>
<tr>
<td>BEST(^{38}) 254 pts.</td>
<td>IVUS guided aggressive PCI (stent only when necessary) vs. routine stenting</td>
<td>16.8(^{\circ}) - aggressive PCI 18.1(^{\circ}) stent</td>
<td>16(^{\circ}) - aggressive PCI 20(^{\circ}) - stent</td>
<td>IVUS guided aggressive PCI with provisional stenting is safe and with the same results like routine stenting</td>
</tr>
<tr>
<td>SIPS(^{39}) 269 pts.</td>
<td>IVUS vs. angio guided PCI</td>
<td>29(^{\circ}) - IVUS, 35(^{\circ}) angio</td>
<td>30(^{\circ}) - IVUS, 37(^{\circ}) angio</td>
<td>same restenosis rate. Lower TLR in IVUS group</td>
</tr>
<tr>
<td>Gaster(^{40}) 108 pts.</td>
<td>IVUS guided vs. angio guided (IVUS controlled) PCI</td>
<td>not declared</td>
<td>22(^{\circ}) - IVUS 41(^{\circ}) angio</td>
<td>lower MACE in IVUS guided group, better C/E(^{3}) ratio in IVUS group</td>
</tr>
<tr>
<td>Meuller(^{41}) C/E analysis of SIPS study</td>
<td>IVUS guided vs. angio guided PCI</td>
<td>not declared</td>
<td>19.8(^{\circ}) - IVUS 31.1(^{\circ}) angio</td>
<td>lower MACE in IVUS guided group, better C/E(^{4}) ratio in IVUS group</td>
</tr>
<tr>
<td>Colombo(^{42}) 130 pts.</td>
<td>IVUS guided intervention in lesions longer than 15 mm</td>
<td>25(^{\circ}) - IVUS 39(^{\circ}) angio (p&lt; 0.05)</td>
<td>22(^{\circ}) - IVUS 38(^{\circ}) angio (p&lt; 0.05)</td>
<td>Lower restenosis, MACE and number of stents in IVUS group</td>
</tr>
</tbody>
</table>

\(^{1}\) 12 months MACE, \(^{\circ}\) 6 months MACE, \(^{\bullet}\) 2.5 year, C/E cost/effectiveness ratio, \(^{\n}\) 2 years, TLR: target lesion revascularization, BMS: bare metal stents

* non randomized study, control group is composed by similar lesions

Table 2. IVUS guided coronary intervention
The table 3 summarizes the strategy for choosing balloon diameter, acute complications and frequency of stenting in aforesaid studies.

<table>
<thead>
<tr>
<th>study</th>
<th>B/A ratio</th>
<th>equation for balloon diameter</th>
<th>occurrence of acute severe dissection</th>
<th>stenting</th>
</tr>
</thead>
<tbody>
<tr>
<td>CLOUT34</td>
<td>1.3 * 0.8-1**</td>
<td>mean MLDref + mean MVDref / 2</td>
<td>5%</td>
<td>not declared</td>
</tr>
<tr>
<td>Haase35</td>
<td>0.8-1 **</td>
<td>EEM in lesion</td>
<td>11%</td>
<td>0%</td>
</tr>
<tr>
<td>Schroeder36</td>
<td>1.4 * 0.88**</td>
<td>(2xEEM refprox + 2xEEMrefdist + 2xEEM lesion)/ 6</td>
<td>12%</td>
<td>2%</td>
</tr>
<tr>
<td>Abizaid37</td>
<td>1.34 *</td>
<td>(EEMmaxlesion + EEM minlesion) / 2</td>
<td>28%</td>
<td>53%</td>
</tr>
<tr>
<td>BEST38</td>
<td>1.25 *</td>
<td>(EEM refprox + EEMrefdist) / 2</td>
<td>18%</td>
<td>44% (in IVUS group)</td>
</tr>
<tr>
<td>SIPS39</td>
<td>1.23 * 1.03 angio</td>
<td>MLDref + MVDref / 2 (in smallest ref. segment)</td>
<td>3% IVUS, 3.2% angio</td>
<td>49.5% angio, 49.7% IVUS</td>
</tr>
<tr>
<td>Gaster40</td>
<td>not declared</td>
<td>(MLArefprox + MLArefdist) / 2</td>
<td>not declared</td>
<td>85% angio, 87% IVUS</td>
</tr>
<tr>
<td>Meuller41</td>
<td>not declared</td>
<td>not declared</td>
<td>not declared</td>
<td>49.5% angio, 49.7% IVUS</td>
</tr>
<tr>
<td>Colombo42 °</td>
<td>1.23 **</td>
<td>EEM in lesion</td>
<td>4.6%</td>
<td>51.5%</td>
</tr>
</tbody>
</table>

Abbreviations: ref – reference segment, refprox – reference proximal segment, refdist – reference distal segment, * angiographic, ** IVUS, B/A - balloon artery ratio, mean MLAref – mean lumen in reference segments, QCA = quantitative coronary angiography - EEMmax lesion – maximal EEM in lesions, EEM min lesion – minimal EEM in lesions, ° long lesion intervention

Table 3. Different strategies for choosing of balloon diameter.

The most frequent formula for choosing of balloon diameter is \((EEM_{refprox} + EEM_{refdist}) / 2\) coming from BEST study or just \(EEM_{refdist}\), which is adapted form the study SIPS. IVUS criteria for optimal result after balloon dilatation are listed in table 4.

<table>
<thead>
<tr>
<th>study</th>
<th>criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>CLOUT34</td>
<td>MLA (\geq 65%) mean MLA_{ref}, no signs of flow limiting dissection</td>
</tr>
<tr>
<td>Haase35</td>
<td>increasing of MLA by at least 20% of EEM, no signs of flow limiting dissection</td>
</tr>
<tr>
<td>Abizaid37</td>
<td>MLA (\geq 65%) mean MLA_{ref} or MLA (\geq 6\text{mm}^2), no signs of flow limiting dissection</td>
</tr>
<tr>
<td>BEST38</td>
<td>residual stenosis (&lt; 30%) (IVUS and angio), MLA &gt; 6\text{mm}^2, no signs of flow limiting dissection</td>
</tr>
<tr>
<td>SIPS39</td>
<td>MLA (\geq 65%) mean MLA_{ref}, no signs of flow limiting dissection</td>
</tr>
<tr>
<td>Mueller41</td>
<td>residual stenosis (\leq 35%), MLA &gt; 65% meanMLA_{ref}, no signs of flow limiting dissection</td>
</tr>
<tr>
<td>Colombo42 °</td>
<td>MLA (\geq 50%) EEM_{lesion}, MLA (\geq 5.5\text{mm}^2)</td>
</tr>
</tbody>
</table>

Abbreviations: mean MLA_{ref} - mean minimal lumen area in reference segment, QCA - quantitative coronary angiography, ° long lesion intervention

Table 4. IVUS criteria of optimal result after balloon dilatation.
Based on these studies with IVUS guided balloon dilatation we can summarize:

1. Using of larger balloons (balloon/artery ratio more than 1 according to angiographic assessment) is safe and without increased risk for acute severe complications. In hospital MACE were 1.4-3.9%. Occurrence of significant dissection varies in large range, probably for different definition of this kind of complication. However, all authors declare low risk for severe acute dissection.

2. The rate of in stent restenosis is consistently low in all studies. It means that it is safe to avoid stent implantation after fulfilling of IVUS criteria for adequate results after balloon dilatation. Surprisingly, higher in stent restenosis and higher MACE were found in some studies in patients with stent implantation compared to plain balloon dilatation. The reason for this finding is probably for bias; according to the study design stents were implanted to lesions with non satisfactory results after balloon dilatation or for treatment of complications after balloon dilatation. Higher occurrence of in stent restenosis and MACE is expected in these types of lesions.

3. The dark side of IVUS guided balloon angioplasty is a prolonged procedural time (5-13 minutes), increased X-ray time (2-3.6 minutes) and higher amount of contrast dye (12-34 ml).\(^{38-41}\)

4. Routine IVUS guided intervention can be beneficial from financial point of view. Optimal decision making decreases number of implanted stent and frequency of in stent restenosis and avoids peri-procedural complications from non-indicated coronary interventions\(^{40,41}\).

5. IVUS guided intervention should be used for avoiding stent implantation in patients unsuitable for dual antiplatelet therapy.

5. Bare-metal stent implantation

5.1 Mechanism of lumen enlargement during stent implantation
The most important mechanisms are plaque redistribution (inside stent) and plaque extrusion (outside stent) to the reference segments (more frequently to the distal one)\(^{43}\). Plaque redistribution and extrusion play role during restenosis in edge segment of stent\(^{44}\). Less important factors are plaque compression, plaque embolization and vessel enlargement (more in vessel with negative remodeling before PCI)\(^{43}\).

Final stent diameter is a result of interplay between pressure during implantation and vessel wall resistance. Declared stent diameters for different pressures during dilatation do not correlate with real stent diameter after implantation. These numbers for stent diameter come from \textit{in vitro} tests in water and do not reflect real situation in the vessel. Costa et al.\(^{45}\) compared 200 drug eluting stent diameters from IVUS measurement immediately after implantation and declared stent diameter for nominal pressure. At least 90% of minimal lumen diameter for nominal pressure was reached only in 4% of stents. In stent area can be decreased immediately after implantation also by protrusion of plaque material through the struts\(^{46}\) (\textit{figure 9}).

It is important to distinguish between stent expansion and stent apposition. Expansion means ratio between minimal stent area (MSA) and lumen area in reference segment. Inadequate expansion (\textit{figure 10}) can be improved by high pressure postdilatation. Apposition reflects contact between stent struts and vessel wall. Inadequate apposition (\textit{figure 11}) can be solved by low pressure dilatation with a bigger balloon.
5.2 High pressure stent dilatation
The first implantations of Palmaz-Schatz and Gianturco-Roubin stents were complicated by high occurrence of subacute stent thrombosis. Colombo and coworkers started IVUS guided...
high pressure dilatation, which (together with dual antiaggregation) decreased the rate of subacute stent thrombosis to 0.9%. Routinely using high pressure dilatation (up to 20 atm.) improves stent expansion and apposition without increasing acute complications. Choi et al. found, that only 54% of angiographically adequately expanded stents fulfill IVUS criteria for optimal stent expansion. Authors performed high pressure postdilatation and final MACE was only 11% in 6 months. This study confirms minimal stent area (MSA) as the most important risk factor of restenosis (figure 12).

Fig. 12. In stent restenosis

5.3 IVUS guided stent implantation
There are many studies focusing on impact of IVUS guidance on stent implantation. They are summarized in table 5. Different criteria for optimal stent expansion were used in published trials, details are summarized in table 6.

<table>
<thead>
<tr>
<th>study name</th>
<th>study goal</th>
<th>restenosis</th>
<th>MACE</th>
<th>comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>MUSIC</td>
<td>restenosis during IVUS guided stent implantation</td>
<td>9.7%</td>
<td>12.1%</td>
<td>the second lowest restenosis rate in studies with BMS</td>
</tr>
<tr>
<td>161 pts.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blasini</td>
<td>IVUS vs. angio* guided stent implantation</td>
<td>20.9% IVUS</td>
<td>29.9% angio (p=0.033)</td>
<td>Restenosis 13.5% in stent fulfilling IVUS criteria</td>
</tr>
<tr>
<td>105 pts.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>study name</td>
<td>study goal</td>
<td>restenosis</td>
<td>MACE</td>
<td>comments</td>
</tr>
<tr>
<td>------------</td>
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<td>------------</td>
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<td>----------</td>
</tr>
<tr>
<td>RESIST$^{32}$ study</td>
<td>IVUS vs. angio guided (IVUS controlled) stent implantation</td>
<td>22.5% IVUS 28.8% angio (p=0.25)</td>
<td>not declared</td>
<td>IVUS guidance does not decrease rate of in stent restenosis</td>
</tr>
<tr>
<td>OPTICUS$^{33}$ 550 pts.</td>
<td>IVUS vs. angio guided stent implantation</td>
<td>24.5% IVUS 22.5% angio (p=0.68)</td>
<td>17.9%* IVUS 15.3% angio (p=0.4)</td>
<td>IVUS guiding do not decrease neither rate of in stent restenosis nor MACE</td>
</tr>
<tr>
<td>CRUISE$^{34}$ 499 pts.</td>
<td>IVUS vs. angio guided (IVUS controlled) stent implantation</td>
<td>not declared</td>
<td>TVR 8.5% in IVUS group 15.3% in angio group (p=0.019)</td>
<td>IVUS guided stenting leads to lower TVR</td>
</tr>
<tr>
<td>Choi$^{49}$ 278 pts.</td>
<td>IVUS vs. angio guided stent implantation</td>
<td>not declared</td>
<td>12%*IVUS 19% angio (p=0.11)</td>
<td>IVUS leads to lower periprocedural complications with a trend for lower TVR in IVUS group(p=0.08)</td>
</tr>
<tr>
<td>AVID$^{35}$ 800 pts.</td>
<td>IVUS vs. angio guided stent implantation</td>
<td>not declared</td>
<td>18% IVUS 19% angio</td>
<td>IVUS leads to larger lumen, but without any effect on 30 days and 12 months MACE</td>
</tr>
<tr>
<td>TULIP$^{36}$ 150 pts.</td>
<td>IVUS vs. angio guided stenting of long lesions</td>
<td>23%* IVUS 46% angio (p=0.008)</td>
<td>6%* IVUS 20% angio (p=0.01)</td>
<td>Better clinical and angiographic results in IVUS group despite more implanted stents</td>
</tr>
<tr>
<td>PRESTO$^{57}$ 796 pts with IVUS vs. 8274 with angio</td>
<td>IVUS vs. angio guided stenting</td>
<td>not declared</td>
<td>TVR 13.8% IVUS 12.2% angio (p=0.9)</td>
<td>Larger MLD in IVUS group, but without difference in MACE. Criteria for adequate IVUS stent expansion was let only on operators discretion</td>
</tr>
<tr>
<td>DIPOL$^{58}$ 163 pts.</td>
<td>IVUS vs. angio guided stenting</td>
<td>10% IVUS 27% angio</td>
<td>7.3% IVUS 16% angio</td>
<td>Best results for IVUS guided in randomized trial</td>
</tr>
<tr>
<td>Gaster$^{55,60}$ 108 pts</td>
<td>IVUS vs. angio guided stenting</td>
<td>16% IVUS 25% angio</td>
<td>22% IVUS 41% angio</td>
<td>IVUS guiding is cost saving</td>
</tr>
</tbody>
</table>

* non randomized trial, angio group is a historic control, ** IVUS criteria was fulfilled in 49.5% patients and in these ones was restenosis 13.5%, * 6 months, * 12 months, * 9 months, TVR - target vessel revascularization

Table 5. Trials assessing IVUS guided stent implantation.
Table 6. Different criteria for optimal stent expansion.

<table>
<thead>
<tr>
<th>study</th>
<th>criteria for optimal expanded stent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Katritsis</td>
<td>full stent apposition, MSA ≥ 90% meanMLA&lt;sub&gt;ref&lt;/sub&gt;, symmetrical expansion (min SD/maxSD &gt; 0.7) (correlation with FFR 0.94)</td>
</tr>
<tr>
<td>Fearon</td>
<td>MSA 7 mm&lt;sup&gt;2&lt;/sup&gt; (correlates with FFR 0.96)</td>
</tr>
<tr>
<td>Hanekamp</td>
<td>full stent apposition, MSA ≥ 90% meanMLA&lt;sub&gt;ref&lt;/sub&gt;, or MSA ≥ 100% minMLA&lt;sub&gt;ref&lt;/sub&gt;, symmetrical expansion (minSD/maxSD &gt; 0.7), (correlation with FFR 0.94)</td>
</tr>
<tr>
<td>Gorge</td>
<td>symmetrical stent expansion, MSD ≥ 3 mm, full apposition</td>
</tr>
<tr>
<td>Choi</td>
<td>complete stent apposition, MSA ≥ 80% distalMLA&lt;sub&gt;ref&lt;/sub&gt;, symmetrical expansion (minSD/maxSD &gt; 0.8)</td>
</tr>
<tr>
<td>Colombo</td>
<td>full stent apposition, MSA ≥ minMLA&lt;sub&gt;ref&lt;/sub&gt;, MLA &gt; 60% MLA&lt;sub&gt;ref&lt;/sub&gt; in edge regions</td>
</tr>
<tr>
<td>Ahmed</td>
<td>complete stent apposition, MSA ≥ 80% meanMLA&lt;sub&gt;ref&lt;/sub&gt; or MSA 7.5 mm&lt;sup&gt;2&lt;/sup&gt;</td>
</tr>
</tbody>
</table>
| MUSIC*           | - complete stent apposition, MSA ≥ 90 meanMLA<sub>ref</sub> or ≥ 100 minMLA<sub>ref</sub>  
|                  | - in case of > 9 mm<sup>2</sup>:  
|                  | MSA ≥ 80% meanMLA<sub>ref</sub> or ≥ 90% minMLA<sub>ref</sub>  
|                  | MSA in proximal part of stent ≥ 90% proxMLA<sub>ref</sub>, symmetrical expansion (minSD/maxSD > 0.7) |
| Blasini          | full stent apposition, MSA > 8 mm<sup>2</sup>, or MSA ≥ 90% meanMLA<sub>ref</sub> complete coverage of dissection |
| RESIST           | MSA ≥ 80% meanMLA<sub>ref</sub>                                                                    |
| OPTICUS          | MUSIC criteria, residual stenosis less than 10% (angiographically)                                 |
| CRUISE           | residual stenosis < 10% (angiographically)                                                        |
| AVID             | full stent apposition, residual stenosis < 10%, no signs of dissection                            |
| TULIP            | complete stent apposition, MSD ≥ 80% meanMLD<sub>ref</sub>, MSA ≥ 90% distalMLA<sub>ref</sub>      |

Abbreviations: SD - stent diameter, meanMLA<sub>ref</sub> - mean minimal lumen area in reference segments, minMLA<sub>ref</sub> minimal - minimal lumen area i reference segments, MSD - minimal stent diameter, MSA - minimal stent area  
* all criteria were fulfilled in 81% patients and these patients were treated only with acetylosalycilic acid and rate of subacute stent thrombosis was 1.3%

Table 7. Risk of ISR and achievement of different IVUS parameters.

<table>
<thead>
<tr>
<th>criteria</th>
<th>restenosis rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>MSA &gt; 9 mm&lt;sup&gt;2&lt;/sup&gt;</td>
<td>11%</td>
</tr>
<tr>
<td>MSA &gt; 9 mm&lt;sup&gt;2&lt;/sup&gt; a MSA ≥ 80% meanMLA&lt;sub&gt;ref&lt;/sub&gt;</td>
<td>12.5%</td>
</tr>
<tr>
<td>MSA ≥ 55% meanEEM&lt;sub&gt;ref&lt;/sub&gt;</td>
<td>17%</td>
</tr>
<tr>
<td>MSA ≥ 90% meanMLA&lt;sub&gt;ref&lt;/sub&gt;</td>
<td>21%</td>
</tr>
<tr>
<td>MSA ≥ 90% distal MLA&lt;sub&gt;ref&lt;/sub&gt;</td>
<td>22%</td>
</tr>
</tbody>
</table>

Abbrevation: MSA - minimal stent area

Table 7. Risk of ISR and achievement of different IVUS parameters.
The lowest rate of ISR is in stents with MSA > 9mm². However, for achievement of this MSA it is necessary to use a 3.5mm stent (with ideal stent area 9.6mm²), because 3mm stent has an ideal stent area 7.1mm². MSA 8 mm² as a sufficient post-stenting area was confirmed by Hoffmann et al⁶⁵. They found a mean MSA in stent with ISR 7.1mm² and 8.1mm² in stents without development of ISR. This cut-off MSA is also a predictor for development of ISR in long stents⁶⁶. Further predictors for ISR are:
- length of stents⁶⁷
- strong calcification⁶⁸
- bulky plaque compensated by positive vessel remodelation⁶⁹-⁷² (figure 13)
- plaque protrusion through the stent struts⁷³

MSA is the most important factor for development of in stent thrombosis (IST)⁷⁴. The incidence of this feared complication is less than 0.5%⁷⁴. Parise et al.⁷⁵ published recently a meta-analysis of all randomized studies comparing IVUS guided vs. angio guided stenting in pre-drug-eluting stent era. They concluded that IVUS guided stenting significantly lowered the 6-month angiographic restenosis rates, 12-month revascularization and MACE rate. They did not find any effect of IVUS guidance on death or myocardial infarction.

![Fig. 13. Large plaque compensated by positive vessel remodeling](image)

### 6. Drug-eluting stent implantation

Drug eluting stents (DES) significantly reduce the risk of in stent restenosis compared to BMS⁷⁶-⁷⁸. IVUS guidance seemed to be needless for DES excellent results. However, several issues are still a problem even with the use of DES and some of them, like in stent thrombosis, seem to be more important than in BMS.

In stent restenosis still exists and its incidence is 5-10% in selected populations⁷⁹-⁸⁰. It seems to be more pronounced in paclitaxel eluting stent than sirolimus eluting stents⁸¹. Minimum stent area that best separates restenosis from no restenosis in DES is between 5.0-6.0 mm²⁸²,⁸³. The occurrence of in stent thrombosis (IST) in DES is 1-1.5%⁸⁴. The main cause of IST is stent under expansion. DES with further IST development showed significantly lower MSA (4.3-4.6 mm²). Further risk factor for IST development is residual edge stenosis, defined as a MLA < 4 mm² and a plaque burden > 70%⁸⁵,⁷⁴. These risk factors remain the same like in BMS, where the presence of dissection, thrombus or tissue prolaps into the stent were
recognized as further risk factors for IST development\textsuperscript{86,87}. The occurrence of IST in BMS is estimated at 0.9\%\textsuperscript{88}. The risk factors for IST development can be revealed by IVUS control after stent implantation. Roy et al.\textsuperscript{89} published study, where IVUS guidance of DES implantation reduced development of IST in 30 days (0.5\% in IVUS vs. 1.5\% in angio guided group) and 12 month (0.7\% in IVUS vs. 2.0\% in angio guided group), as well as decreased need for revascularization and MACE in 30 day. Unfortunately, there were no clear criteria for adequate stent deployment in this study. Claessen et al.\textsuperscript{90} published a study comparing angio and IVUS guidance in 1504 sirolimus eluting stent implantation in the MATRIX (Comprehensive Assessment of Sirolimus-Eluting Stents in Complex Lesions) registry. They found significantly lower occurrence of myocardial infarction and combined endpoint of myocardial infarction and death in IVUS guided group. Hur et al.\textsuperscript{91} published their enormous registry of angio (3744 patients) versus IVUS (4627 patients) guided stent implantation. The main finding of this study is lower mortality in IVUS guided group. This result was seen mainly in DES implantation, because mortality in BMS group was the same. However, the explanation of this interesting finding is not clear, because the occurrence of myocardial infarction, target vessel revascularization and in stent thrombosis was without significance differences between angio and IVUS group. Based on aforesaid trials we can conclude, that IVUS guidance of DES implantation probably improves patient’s outcome, but further trials are needed for better understanding of this phenomenon.

Important phenomenon in DES implantation is incomplete stent apposition (ISA), which means, that at least one strut is not adequately apposed to vessel wall. One should distinguish between acute stent malapposition, which is present immediately after stent implantation, late stent malapposition (LSM), which can be persistent, it means that acute malapposition is not healed and late-acquired stent malapposition (LASM), which develops despite normal finding after stent implantation. The mechanism of LASM development is not known, several mechanisms are thought:
- positive vessel remodeling leading to vessel enlargement
- decrease of plaque volume behind the stent caused by antiproliferative effect of DES
- thrombus dissolution after PCI of thrombus containing lesions

The frequency of LSM is not clear, a quite broad range for this phenomenon was published (4-21\%)\textsuperscript{92,93}. LSM is not found only in DES, but also in BMS, where it’s incidence is 4.4-5.4\%.\textsuperscript{94,95} Empty space behind stent struts can lead to decrease of blood flow in this region and cause development of thrombosis. However, this theoretic concept was not proved in any study following the natural course of patients with LSM. Hong et al.\textsuperscript{96,97} did not find any clinical adverse event in patient with LSM during 10 months follow up. On the other hand Cook et al.\textsuperscript{98} published correlation between LSM and very late IST. Moreover, Hassan et al.\textsuperscript{99} published meta-analysis of 17 randomized trials focusing on LSM in BMS as well as DES. They found four times higher risk of LASM in patients with DES compared to BMS and LSM (acquired or persistent) increased significantly risk for (very) late in stent thrombosis (OR 6,51).

A rare complication (1.25\%) of DES implantation is development of coronary aneurysm. The definition of aneurysm is focal enlargement of vessel lumen, which is 50\% larger than adjacent reference vessel. Coronary aneurysm is not a benign finding as up to 40\% of patients needed revascularization in a study done by Alfonso et al.\textsuperscript{100}.
7. Ivus guided PCI in specific situation

7.1 PCI of bifurcation lesions

PCI in bifurcation is a more challenging procedure with a risk of compromising flow in side branch (SB) and with higher rate of restenosis. The “classical” mechanisms of worsening flow in side branch were thought to be:

- plaque compression in ostial part of SB during dilatation in main vessel (MV). The presence of diffuse ostial plaque (figure 14) has higher risk for TIMI 2 flow after stenting than presence of eccentric plaque.
- plaque shifting from the MV to SB during dilatation in MV (“snow-plow” phenomenon)
- interposition of stent struts across the ostium of SB

Plaque in bifurcation is mainly localized in counter carina. However, Medina et al. proved the presence of plaque also in carina. They found plaque in 32% of bifurcations and moreover, in 16% of bifurcations the plaque in the carina was larger than in counter carina area. Furthermore, authors showed, that the mechanism of ostial SB damage after stent implantation in the MV was always due to displacement of the carina and no cases of plaque shifting were found. Further interesting finding of this study is that plaque in the carina was greater in the bifurcations that had maximum stenosis located distal to the carina in the MV. Authors explain this finding by hypothesis of different flow velocity patterns in these lesions. Atheroprotective high shear stress can be transformed into an atero-prone low shear stress. Authors describe less damage of SB ostium after stenting of lesions with plaque at the carina. Plaque probably makes carina more resistant and does not allow carina shifting, which is now thought to be a dominant mechanism causing damage to the SB ostium when a stent is implanted into the MV.

Important contribution for planning of PCI in bifurcations is a study done by Costa et al. They found that the part of MV just behind the origin of SB (so called “lower diamond” or “polygon of confluence”) is more prone for small minimal stent area after dilatation of SB ostium, which is not reverted even after kissing balloon dilatation. Kang et al. published a study assessing the IVUS predictors for side branch compromise after single-stent crossover technique and found two predictors for post stenting FFR<0.8: MLA 2.4mm² and PB > 51% in SB ostium.
Main role of IVUS guidance in bifurcation PCI is precise assessment of atherosclerotic burden of main vessel, carina, ostial part of side branch and the choice of the best strategy based on these findings. IVUS can improve the choice of ideal stent diameter, because bifurcation area is frequently affected by negative remodeling, which is not visible in routine angiography\textsuperscript{107}.

IVUS should be used of most cases of uncertain atherosclerotic distribution in bifurcation lesions, because in case of favorable finding in SB ostium a planned complex procedure can be converted to a simple one with just stenting of MV.

7.2 PCI of chronic total occlusion
The main disadvantage for IVUS guidance of chronic total occlusion (CTO) interventions is the lateral view of IVUS probe. A prototype of forward-looking IVUS system was developed, but till now it has never been commercialized. IVUS can help during wire introduction to the proximal fibrous cap of CTO in presence of side branch just proximal to occluded segment. IVUS is introduced to this side branch and from this location the wire reaching the origin of total occlusion can be visualized (figure 15). IVUS also can help with re-introduction of wire from false lumen to the true lumen, but it is necessary to introduce the IVUS probe to the false lumen after its predilatation with small balloon\textsuperscript{108}. This technique unfortunately increases risk of vessel perforation.

Fig. 15. Schema of IVUS guidance during PCI of chronic total occlusion.

8. Complications
8.1 Dissections
Dissections are tears in the plaque that are parallel to the vessel wall with blood flow in the false lumen and tend to occur at the junction of elements with different compliance (plaque and normal vessel wall, edges of stents) (figure 16, 17). Dissections can be described as proximal or distal to the lesion; epicardial or myocardial; and according to length, circumferential arc, depth, lumen compromise, bulkiness and mobility of the flap\textsuperscript{5}. The major predictor of an unfavorable prognosis of dissection is a decreased blood flow in affected coronary artery. This situation must be solved immediately. IVUS can also detect dissections, which are not visible on angiography and it is questionable how many of them should be
treated. Nishida et al. followed 124 patients with non-flow limiting dissection (65% of them were after stenting). They found that dissection can be left untreated if the residual lumen is more than 6 mm$^2$ and lumen area is more than 40% of the EEM area. The edge dissections after stenting, which can be visualized only by IVUS and are not apparent during angiography, are not indicated for any intervention, because of good prognosis by spontaneous healing.

![Fig. 16. Dissection with tear reaching the media.](image1)

Fig. 16. Dissection with tear reaching the media.

![Fig. 17. Complex dissection with IVUS probe in true lumen, one false lumen is located down to true lumen (♦) and another entry to the false lumen is located above the true lumen (∗).](image2)

Fig. 17. Complex dissection with IVUS probe in true lumen, one false lumen is located down to true lumen (♦) and another entry to the false lumen is located above the true lumen (∗).

### 8.2 Intramural hematomas
Intramural hematoma is a variant of a dissection. The angiographic appearance ranges from a dissection, thrombus, and abrupt closure to non-significant abnormality. The EEM expands outwards and the intima is pushed inwards and straightens to cause lumen compromise (figure 18). Blood accumulates in the space caused by the split in the media and becomes static and echogenic. The hematoma can propagate antegrade or retrograde, but tends to be stopped by branches or severely diseased parts of the vessel (particularly calcified plaques). In a study, which included more than 1000 patients with IVUS control after PCI, an intramural hematoma was found in 6.7% with a high rate of clinical events.
8.3 Haziness
IVUS is a very useful tool for diagnosis of underlying processes causing a hazy appearance of lesions after PCI. The most common findings are large residual plaque burden, dissection and plaque protrusion. Rarely stent deformation or intramural hematoma can be found. Intimal wrinkling can occur when a vessel is straightened by a guidewire. It reverses when the wire is removed or when the stiff wire is exchanged for a soft one. A narrowing of the lumen with a straightening of a normal-looking intima, behind which is an echoluent zone can be seen in IVUS picture. Discontinuity of the EEM can be found, which is caused by simultaneously visualized both sides of fold in the artery. Finally, it is important to recognize an angiographic pseudo complication with normal IVUS finding5.

9. Conclusions
Based on studies focusing on IVUS guidance of stent implantation we can summarize:
1. Based on IVUS criteria we can probably safely defer PCI
2. Satisfactory IVUS finding after balloon dilatation has same results as a stenting with BMS
3. IVUS guidance decreases angiographic restenosis and consequently the revascularization and MACE rate
4. IVUS guidance decreases rate of in-stent thrombosis
5. Contraindication for IVUS guidance are small vessels, tortuous vessels and degenerated vein grafts

This summary raises the question why IVUS is not used routinely during all PCI procedures. The answers can be divided into two groups. First there are the lessons we have learned from IVUS and this phenomenon is known as an “IVUS eye”. We know that vessels are bigger than they look on angio and we are using bigger stents than before IVUS studies. We know, that without high pressure post dilatation we are not able to achieve sufficient stent diameter despite information derived from manufacturer’s compliance charts. These factors dramatically contributed to lowering of in-stent restenosis and therefore it is difficult to find statistical significant difference between IVUS and angio guided interventions, like it was described in OPTICUS trial. However, IVUS is still irreplaceable during investigation of unclear finding during angiography (like hazy lesions) or after stenting. IVUS is especially recommended during PCI of left main, the last remaining coronary artery and PCI in patients who are contraindicated for dual antiplatelet therapy for avoiding stent implantation. Furthermore, precise assessment of bifurcation can change the strategy from more complex to simple one in case of low risk profile of ostial part of side branch.

The group of arguments against routine use of IVUS during PCI is cost of IVUS catheter, longer procedural time and higher contrast dye consumption. Moreover IVUS guided PCI are more complex and need more skills than angio guided ones. However, in indicated cases IVUS guidance undoubtedly improves short as well as long term of coronary interventions.

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IVUS Guided PCI


Intravascular Ultrasound


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Intravascular ultrasound (IVUS) is a cardiovascular imaging technology using a specially designed catheter with a miniaturized ultrasound probe for the assessment of vascular anatomy with detailed visualization of arterial layers. Over the past two decades, this technology has developed into an indispensable tool for research and clinical practice in cardiovascular medicine, offering the opportunity to gather diagnostic information about the process of atherosclerosis in vivo, and to directly observe the effects of various interventions on the plaque and arterial wall. This book aims to give a comprehensive overview of this rapidly evolving technique from basic principles and instrumentation to research and clinical applications with future perspectives.

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