Coronary bifurcations stenosis: definition, classification of lesions, classification of techniques, measurements

Y. Louvard, ICPS, Massy, Générale de Santé, France

European Bifurcation Club 10th anniversary meeting
Bordeaux, France, October 17-18th 2014
European Bifurcation Club

1st Meeting, Bordeaux,
September 15-16, 2005

Organised by:

Olivier Darremon, Thierry Lefèvre, Yves Leuvarc, Goran Stankovic, Remo Albiero, Manuel Pan
Thursday 15th September 2005:

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<td>16:30 - 18:30</td>
<td>Registration, welcome drink</td>
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<td>19:00 - 21:30</td>
<td>Evening 'cine-dinner' (Case review session):</td>
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<td>11:50 - 12:05</td>
<td>Dedicated stents and delivery systems: advantages and drawbacks: D. Dudek</td>
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<td>12:05 - 12:20</td>
<td>What should be the ideal stent?: E. Albiero</td>
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<td>12:20 - 12:25</td>
<td>Lunch</td>
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<td>12:25 - 13:15</td>
<td>Photo, Lunch</td>
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<td>13:35 - 14:50</td>
<td>THIRD SESSION- Chairman: E. Garcia, R. Reifort, G. Stankovic</td>
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<td>13:35 - 13:45</td>
<td>Debate- The case of distal left main lesions: Should we treat it?</td>
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<td>13:55 - 14:05</td>
<td>Yes. M. Silvestri</td>
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<td>14:05 - 14:15</td>
<td>Debate- Which approach for LH stenting with DES?</td>
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<td>14:15 - 14:25</td>
<td>Insights from the Thrombin center DES registries: A. Haye</td>
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<td>Two DES are not better than one. T. Lefèvre</td>
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<td>14:35 - 14:50</td>
<td>We need a dedicated stent: P. Guyon</td>
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<td>Coffee break</td>
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Friday 16th September 2005:

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<tr>
<td>08:00 - 08:30</td>
<td>FIRST SESSION- Chairman: R. Albiero, D. Hildick-Smith, M. Pan</td>
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<td>08:00 - 08:20</td>
<td>Definition, epidemiology, classification, differentiation from standard lesions: B. Chevalier</td>
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<td>08:20 - 08:40</td>
<td>Review of bifurcation treatment techniques: Theoretical advantages and drawbacks: Y. Leonard</td>
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<td>09:00 - 09:10</td>
<td>Lessons from bench testing: J. Ornstein</td>
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<td>09:10 - 09:20</td>
<td>The issue of side branch protection, side branch access and side branch patency: R. Brunel</td>
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<td>09:20 - 09:30</td>
<td>Lessons from QCA in bifurcation lesions: M. Costa</td>
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<td>09:30 - 09:45</td>
<td>Lessons from IVUS in bifurcation lesions: G. Fontel</td>
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<td>09:45 - 10:00</td>
<td>Decision QCA+IVUS</td>
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<td>10:00 - 10:15</td>
<td>State of the art Bifurcation: I. Sheblan</td>
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<td>10:15 - 10:40</td>
<td>Coffee Break</td>
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<td>10:40 - 12:25</td>
<td>SECOND SESSION- Chairman: S. Cartier, O. Barremont, J. Ludwig</td>
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<td>10:40 - 10:50</td>
<td>Two stents always: A. Colombo</td>
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<td>10:50 - 11:10</td>
<td>One stent only: M. Pan</td>
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<td>11:10 - 11:15</td>
<td>Discussion</td>
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<td>11:10 - 11:50</td>
<td>Technique and Insights from China, India, Latin America, North America:</td>
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<td>L. Shuicheng, S. Mathew, MA Perin, S. Cartier</td>
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<td>17:15 - 17:30</td>
<td>Missing Studies: M. Thomas</td>
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<td>17:30 - 18:00</td>
<td>CONSENSUS</td>
</tr>
<tr>
<td></td>
<td>We have been able to reach a consensus on the following issues: Y. Legend</td>
</tr>
<tr>
<td></td>
<td>The following issues remain unresolved: B. de Breyne</td>
</tr>
<tr>
<td>18:00 - 18:10</td>
<td>Closing remarks: Y. Louvard</td>
</tr>
<tr>
<td>18:15 - 18:30</td>
<td>Departure from the Mercure Cite Mandiol to the Chateau Pope-Clement</td>
</tr>
</tbody>
</table>
Medina classification

Presented by Bernard Chevalier in the 1st EBC meeting, September 16th 2005
Definition of a coronary bifurcation stenosis
Classification of Coronary Artery Bifurcation Lesions and Treatments: Time for a Consensus!

Yves Louvard,1* MD, Martyn Thomas,2 MD, Vladimir Dzavik,3 MD, David Hildick-Smith,4 MD, Alfredo R. Galassi,5 MD, Manuel Pan,6 MD, Francisco Burzotta,7 MD, Michael Zelizko,8 MD, Darius Dudek,9 MD, Peter Ludman,10 MD, Imaed Sheiban,11 MD, Jens F. Lassen,12 MD, Olivier Darremont,13 MD, Adnan Kastrati,14 MD, Josef Ludwig,15 MD, Ioannis Iakovou,16 MD, Philippe Brunel,17 MD, Alexandra Lansky,18 MD, David Meerkin,19 MD, Victor Legrand,20 MD, Alfonso Medina,21 MD, and Thierry Lefèvre,1 MD

Background: Percutaneous coronary intervention (PCI) of coronary bifurcation lesions remains a subject of debate. Many studies have been published in this setting. They are often small scale and display methodological flaws and other shortcomings such as inaccurate designation of lesions, heterogeneity, and inadequate description of techniques implemented. Methods: The aim is to propose a consensus established by the European Bifurcation Club (EBC), on the definition and classification of bifurcation lesions and treatments implemented with the purpose of allowing comparisons between techniques in various anatomical and clinical settings. Results: A bifurcation lesion is a coronary artery narrowing occurring adjacent to, and/or involving, the origin of a significant side branch. The simple lesion classification proposed by Medina has been adopted. To analyze the outcomes of different techniques by intention to treat, it is necessary to clearly define which vessel is the distal main branch and which is (are) the side branch(es) and give each branch a distinct name. Each segment of the bifurcation has been named following the same pattern as the Medina classification. The classification of the techniques (MADS: Main, Across, Distal, Side) is based on the manner in which the first stent has been implanted. A visual presentation of PCI techniques and devices used should allow the development of a software describing quickly and accurately the procedure performed. Conclusion: The EBC proposes a new classification of bifurcation lesions and their treatments to permit accurate comparisons of well described techniques in homogeneous lesion groups.

Key words: bifurcation lesions; QCA; classification of bifurcation lesions; classification of treatments
Functional hierarchy of coronary circulation: direct evidence of a structure-function relation

Ghassan S. Kassab

Department of Biomedical Engineering, University of California, Irvine, California

Submitted 31 May 2005; accepted in final form 10 August 2005

Kassab, Ghassan S. Functional hierarchy of coronary circulation: direct evidence of a structure-function relation. Am J Physiol Heart Circ Physiol 289: H2559–H2565, 2005. First published August 19, 2005; doi:10.1152/ajpheart.00561.2005.—The heart muscle is nourished by a complex system of blood vessels that make up the coronary circulation. Here we show that the design of the coronary circulation has a functional hierarchy. A full anatomic model of the coronary arterial tree, containing millions of blood vessels down to the capillary vessels, was simulated based on previously measured porcine morphometric data. A network analysis of blood flow through every vessel segment was carried out based on the laws of fluid mechanics and appropriate boundary conditions. Our results show an abrupt change in cross-sectional area that demarcates the transition from epicardial (EPCA) to intramyocardial (IMCA) coronary arteries. Furthermore, a similar pattern of blood flow was observed with a corresponding transition from EPCA to IMCA. These results suggest functional differences between the two types of vessels. An additional abrupt change occurs in the IMCA in relation to flow velocity. The velocity is fairly uniform proximal to these vessels but drops significantly distal to those vessels toward the capillary branches. This finding suggests functional differences between large and small IMCA. Collectively, these observations suggest a novel functional hierarchy of the coronary vascular tree and provide direct evidence of a structure-function relation.

Vascular design, coronary vasculature, morphometry, network analysis, blood flow

for the differences in the branching pattern of EPCA and IMCA came from X-ray studies of Tanaka et al. (28). They found that the self-similar branching pattern of coronary arteries was discrete at the connection between the EPCA and the IMCA (28). Finally, Ritman and colleagues (2, 3) found a similar discontinuity between EPCA and IMCA vessels with micro-computed tomography.

Although these past studies suggest the existence of anatomic differences between EPCA and IMCA, these differences were not connected to the hemodynamics (flow, velocity, etc.) of the coronary circulation. The hypothesis of the present study is that these differences in the hemodynamics (flow, velocity, etc.) of the coronary circulation. The hypothesis of the present study is that there exists not only a characteristic variation in cross-sectional area (CSA) that distinguishes EPCA from IMCA but also a similar characteristic pattern of blood flow. An additional hypothesis is that there exists a functional difference between large and small IMCA that demarcates a possible transition from vessels involved in conduction to those vessels involved in transport based on the behavior of flow velocity. To test these hypotheses, a network analysis of coronary arterial blood flow was carried out based on a full set of anatomic data (5, 8, 11, 12). A functional hierarchy of the coronary arterial tree emerges that distinguishes EPCA from IMCA and further distinguishes two functional classes of IMCA. These findings provide direct evidence of a structure-function relation of the coronary circulation.
Fonctional hierarchy of coronary circulation: direct evidence of structure-fonction relation

Fig. 3. Relationship between normalized stem flow and crown length. A linear relation is observed, consistent with Eq. 7.

Fig. 4. Relationship between normalized crown volume and length. A power law relation is observed, consistent with Eq. 2.

Fig. 5. Relationship between normalized stem flow and diameter. A power law relation is observed, consistent with Eq. 4.

Fig. 6. Relationship between normalized minimum power consumption and crown length as expressed by Eq. 13.
Scaling laws of vascular trees: of form and function

Ghassan S. Kassab
Department of Biomedical Engineering, University of California, Irvine, California
Submitted 1 June 2005; accepted in final form 26 August 2005

Kassab, Ghassan S. Scaling laws of vascular trees: of form and function. Am J Physiol Heart Circ Physiol 290: H894–H903, 2006. First published September 2, 2005; doi:10.1152/ajpheart.00579.2005.—The branching pattern and vascular geometry of biological tree structure are complex. Here we show that the design of all vascular trees for which there exist morphometric data in the literature (e.g., coronary, pulmonary; vessels of various skeletal muscles, mesentery, omentum, and conjunctiva) obeys a set of scaling laws that are based on the hypothesis that the cost of construction of the tree structure and operation of fluid conduction is minimized. The laws consist of scaling relationships between 1) length and vascular volume of the tree, 2) lumen diameter and blood flow rate in each branch, and 3) diameter and length of vessel branches. The exponent of the diameter-flow rate relation is not necessarily equal to 3.0 as required by Murray’s law but depends on the ratio of metabolic to viscous power dissipation of the tree of interest. The major significance of the present analysis is to show that the design of various vascular trees of different organs and species can be deduced on the basis of the minimum energy hypothesis and conservation of energy under steady-state conditions. The present study reveals the similarity of nature’s scaling laws that dictate the design of various vascular trees and the underlying physical and physiological principles.

Kassab, Ghassan S. Scaling laws of vascular trees: of form and function. Am J Physiol Heart Circ Physiol 290: H894–H903, 2006. First published September 2, 2005; doi:10.1152/ajpheart.00579.2005.—The branching pattern and vascular geometry of biological tree structure are complex. Here we show that the design of all vascular trees for which there exist morphometric data in the literature (e.g., coronary, pulmonary; vessels of various skeletal muscles, mesentery, omentum, and conjunctiva) obeys a set of scaling laws that are based on the hypothesis that the cost of construction of the tree structure and operation of fluid conduction is minimized. The laws consist of scaling relationships between 1) length and vascular volume of the tree, 2) lumen diameter and blood flow rate in each branch, and 3) diameter and length of vessel branches. The exponent of the diameter-flow rate relation is not necessarily equal to 3.0 as required by Murray’s law but depends on the ratio of metabolic to viscous power dissipation of the tree of interest. The major significance of the present analysis is to show that the design of various vascular trees of different organs and species can be deduced on the basis of the minimum energy hypothesis and conservation of energy under steady-state conditions. The present study reveals the similarity of nature’s scaling laws that dictate the design of various vascular trees and the underlying physical and physiological principles.

suggests that animals and plants have reached similar solutions for efficient conduction of fluid (28).

Both Murray’s formulation and Uylings’ modification are focused on a particular vessel segment. The flow rate through a vessel branch, however, depends not only on the resistance of that branch but also on the total resistance of the tree distal to that branch. Hence, the formulation of an optimization principle requires the treatment of a tree structure as an integrated whole. Zhou, Kassab, and Molloy (47) (ZKM model) generalized the “minimum energy hypothesis” to an entire coronary arterial tree. In the process, a vessel segment was defined as a stem and the entire tree distal to the stem was defined as a crown (39). Obviously, the entire tree consists of many stem-crown units down to the capillary vessels as shown in Fig. 1. At each bifurcation, there is a unique stem-crown unit that continues down to the smallest unit: an arteriole with two capillaries for an arterial tree or a venule and two capillaries for a venous tree. Functionally, each stem supplies or collects blood from the crown for an arterial or venous tree, respectively. The details of the capillary network (nontree structure) beyond the first bifurcation are excluded from the present...
\[ f = \frac{F(L, V)}{K_m V_{\text{max}}} = \frac{Q_{\text{max}}^2 R_{\text{max}}}{K_m V_{\text{max}}} \left( \frac{L}{L_{\text{max}}} \right)^5 \left( \frac{V}{V_{\text{max}}} \right)^{\epsilon + 1} + \frac{V}{V_{\text{max}}} \]  

(8)

Next, we minimize the cost function for a given blood volume, i.e.,

\[ \frac{\partial f}{\partial \left( \frac{V}{V_{\text{max}}} \right)} = 0 \]

to yield the desired relation:

\[ \left( \frac{L}{L_{\text{max}}} \right)^5 \left( \frac{Q_{\text{max}}^2 R_{\text{max}}}{K_m V_{\text{max}}} \right) \epsilon' = 1 \]

(9)

Two important results follow from Eq. 9: the crown volume-length relation as expressed by Eq. 2 and the following equation for the crown resistance parameter as

\[ \epsilon' = \frac{K_m V_{\text{max}}}{Q_{\text{max}}^2 R_{\text{max}}} \]  

(10)

Hence, \( \epsilon' \) represents the ratio of maximum metabolic to viscous power dissipation for a given tree.

In the ZKM model (47), conservation of energy imposed on a stem-crown system under steady-state and isothermal conditions that neglect the elasticity of the vessel wall and gravitational potential energy results in

\[ \int_{A_s} \frac{1}{2} \rho q^2 u dA = K_m V \]

(11)
Scaling laws of vascular trees: of form and function

Scaling of myocardial mass to flow and morphometry of coronary arteries

Jenny Susana Choy and Ghassan S. Kassab

Department of Biomedical Engineering, Indiana University-Purdue University, Indianapolis, Indiana

Submitted 28 November 2007; accepted in final form 27 February 2008

Choy JS, Kassab GS. Scaling of myocardial mass to flow and morphometry of coronary arteries. J Appl Physiol 104: 1281–1286, 2008. First published March 6, 2008; doi:10.1152/japplphysiol.01261.2007.—There is no doubt that scaling relations exist between myocardial mass and morphometry of coronary vasculature. The purpose of this study is to quantify several morphological (diameter, length, and volume) and functional (flow) parameters of the coronary arterial tree in relation to myocardial mass. Eight normal porcine hearts of 117–244 g (mean of 177.5 ± 32.7) were used in this study. Various coronary subtrees of the left anterior descending, right coronary, and left circumflex arteries were perfused at pressure of 100 mmHg with different colors of a polymer (Microfil) to obtain rubber casts of arterial trees corresponding to different regions of myocardial mass. Volume, diameter, and cumulative length of coronary arteries were reconstructed from casts to analyze their relationship to the perfused myocardial mass. Volumetric flow was measured in relationship with perfused myocardial mass. Our results show that arterial volume is linearly related to regional myocardial mass, whereas the sum of coronary arterial branch lengths, vessel diameters, and volumetric flow show an ~3/4, 3/8, and 3/4 power-law relationship, respectively, in relation to myocardial mass. These scaling laws suggest fundamental design principles underlying the structure-function relationship of the coronary arterial tree that may facilitate diagnosis and management of diffuse coronary artery disease.

body mass (1). The model proposed by West and colleagues (30, 31) addresses the supply of materials (e.g., oxygen) to cells through hierarchical networks of branching tubes (e.g., the circulatory system). Although this model has invited critical remarks when extended to all biologic levels, its validity for mammals is widely accepted.

There is evidence indicating that scaling relationships exist based on basic design principles underlying the coronary vascular tree structure. For example, the sum of arterial branch lengths distal to the point of occlusion has been proposed for estimating the corresponding regional myocardial mass at risk (27). The total volume of blood in mammals has been found to scale proportionately with body mass (28, 31). More recently, Karalis and colleagues (11) indicated that the fractal volume scales proportionally to mass.

Here, we hypothesize that \( V \propto m^{1/4}, L \propto m^{3/8}, D \propto m^{3/8}, \) and \( Q \propto m^{3/4} \), where \( V \) and \( L \) correspond to cumulative arterial volume and length, respectively; \( D \) as arterial diameter, \( Q \) the volumetric flow, and \( m \) the myocardial mass. To verify the hypothesis, the relation between various morphological quantities, flow, and myocardial mass was determined in the swine heart. The limitations and implications of these scaling laws are enumerated.
Scaling of myocardial mass to flow and morphometry of coronary arteries

Relation of angiographic side branch calibre to myocardial mass: a proof of concept myocardial infarct index

Ghassan S. Kassab¹²³, MD, PhD; Deepak L. Bhatt⁴, MD; Thierry Lefèvre⁵, MD; Yves Louvard⁶, MD

¹. Department of Biomedical Engineering, Indiana-Purdue University, Indianapolis, IN, USA; ². Department of Surgery, Indiana-Purdue University, Indianapolis, IN, USA; ³. Department of Cellular and Integrative Physiology, Indiana-Purdue University, Indianapolis, IN, USA; ⁴. VA Boston Healthcare System, Brigham and Women’s Hospital, and Harvard Medical School, Boston, MA, USA; ⁵. Institut Cardiovasculaire Paris Sud, Massy, France

How to define a bifurcation lesion?

- A coronary artery narrowing occurring adjacent to, and/or involving, the origin of a significant side branch.
- A significant SB is a branch that you don't want to lose in the global context of a particular patient.
Bifurcation or not?
Classification of bifurcation lesions
Classifications of bifurcation lesions

Duke
Sanborn
Safian
Icps-Lefevre

Syntax classification

Type A
- Prebranch stenosis not involving the ostium of the side branch

Type B
- Postbranch stenosis of the parent vessel not involving the ostium of the side branch

Type C
- Stenosis of the parent vessel not involving the ostium of the side branch

Type D
- Stenosis involving the parent vessel and the ostium of the side branch

Type E
- Stenosis involving the ostium of the side branch only

Type F
- Stenosis discretely involving the parent vessel and ostium of the side branch

Type IV
- Lesion in the parent vessel that may or may not have additional ostial disease

Type I
- Parent vessel stenosis proximal to bifurcation

Type II
- Parent vessel stenosis distal to bifurcation

Type III
- Branch bifurcation lesion where parent vessel is free of disease and both branches have ostial disease

Type IV
- True bifurcation lesion

Type 1
- Lesion located in the main branch proximal and distal and the ostium of side branch

Type 2
- Lesion located only in the main branch proximal and distal and not the ostium of side branch

Type 3
- Lesion located in the main branch proximal to the bifurcation

Type 4
- Only the ostium of each branch of the bifurcation involved with ostial disease

Type 4a
- Lesion located only in the ostium of main branch

Type 4b
- Lesion located only in the ostium of side branch

Chen - Gao

Plaque distribution

Syntax classification

A New Proposed Simplified Classification of Coronary Artery Bifurcation Lesions and Bifurcation Interventional Techniques

Mohammad-Reza Movahed, MD, PhD and Curtiss T. Stinis, MD

ABSTRACT: Current classification systems of coronary bifurcation lesions are confusing and difficult to memorize. As coronary revascularization techniques become increasingly complex, it is important to establish a universal classification system. This manuscript proposes a simplified classification system that uses a combination of letters and numbers to provide a clinically relevant anatomic description of a given coronary artery bifurcation lesion. This classification consists of the prefix B (for Bifurcation lesion), followed by the addition of 4 separate suffixes. The first suffix consists of one of the letters C, N, S, or L. C = Close to the bifurcation: the lesion is close to a bifurcation, but the distance from the carina is more than the width of the plaque protruding into the lumen; N = Bifurcation lesion with one branch being Non-significant; nonsignificant being defined as less than 2.0 mm vessel diameter; S = Small proximal segment; or L = Large proximal segment: large defined as more than two-thirds of the sum of the diameters of both branch vessels. The second suffix describes the number of diseased ostia. 1M = only the Main vessel ostium is involved; 1S = only the Side branch ostium is involved; or 2 = both ostia are involved. The third suffix classifies the angle between the bifurcation vessels and uses the letters V or T; V = the angle between the two branches is less than 70 degrees, T = angle more than 70 degrees. The fourth suffixes are optional: CA for calcified, LM for left main involvement.

J INVASIVE CARDIOL 2006;18:199–204

Key words: coronary bifurcation, PCI, percutaneous coronary intervention, classification, stenting

Table 1. Classification system nomenclature.

<table>
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<tr>
<th>Prefix</th>
<th>Suffix 1</th>
<th>Suffix 2</th>
<th>Suffix 3</th>
<th>Suffix 4</th>
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<tr>
<td>B</td>
<td>C</td>
<td>1M</td>
<td>V</td>
<td>CA</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>1S</td>
<td>T</td>
<td>LM</td>
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<tr>
<td></td>
<td>S</td>
<td>2</td>
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<td></td>
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<tr>
<td></td>
<td>L</td>
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<td></td>
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</tr>
</tbody>
</table>

Suffix 1:  
- C = Close to bifurcation
- N = Non-significant sidebranch
- S = Small proximal segment
- L = Large proximal segment

Suffix 2:  
- M = Only main branch ostium diseased
- S = Only sidebranch ostium diseased
- 2 = Both main and sidebranch ostia diseased

Suffix 3:  
- V = Angle between branch vessels less than 70 degrees
- T = Angle between branch vessels more than 70 degrees

Suffix 4:  
- CA = Calcified
- LM = Left main involved in bifurcation
A New Proposed Simplified Classification of Coronary Artery Bifurcation Lesions and Bifurcation Interventional Techniques

<table>
<thead>
<tr>
<th>MOVAHED</th>
<th>DUKE</th>
<th>SAFIAN</th>
<th>LEFEVRE</th>
<th>SANBORN</th>
</tr>
</thead>
<tbody>
<tr>
<td>BL1&lt;sub&gt;M&lt;/sub&gt;T, BL1&lt;sub&gt;M&lt;/sub&gt;V</td>
<td>Type B and C</td>
<td>Type IB and IIIB</td>
<td>Type 2 and 4a</td>
<td>Type II</td>
</tr>
<tr>
<td>BS1&lt;sub&gt;M&lt;/sub&gt;T, BS1&lt;sub&gt;M&lt;/sub&gt;V</td>
<td>Type D</td>
<td>Type IA and IIIA</td>
<td>Type 1 and 4</td>
<td>Type I and III</td>
</tr>
<tr>
<td>BS2V, BL2V, BL2T, BS2T, BC</td>
<td>Type A</td>
<td>Type IIB</td>
<td>Type 3</td>
<td>Type IV</td>
</tr>
<tr>
<td>BL1&lt;sub&gt;S&lt;/sub&gt;T, BL1&lt;sub&gt;S&lt;/sub&gt;V</td>
<td>Type E and F</td>
<td>Type IIA and IV</td>
<td>Type 4b</td>
<td></td>
</tr>
<tr>
<td>BS1&lt;sub&gt;S&lt;/sub&gt;T, BS1&lt;sub&gt;S&lt;/sub&gt;V</td>
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A New Proposed Simplified Classification of Coronary Artery Bifurcation Lesions and Bifurcation Interventional Techniques

Prefix: B + The Following Applicable Suffixes

Suffix 1:
- C = Close to a bifurcation: Lesions that are in the vicinity of a bifurcation but in which the distance from the carina is more than the width of the nearest plaque edge which protrudes into the vessel lumen. No further suffixes are required.
- N = Nonsignificant bifurcation lesions: One of the bifurcation vessels is less than 2.0 mm in diameter or clinically appears to be unimportant. No further suffixes are required.
- S = Small proximal segment: The vessel diameter proximal to the bifurcation is less than 2/3 of the sum of the diameters of both branch vessels.
- L = Large proximal segment: The vessel diameter proximal to the bifurcation is equal to or more than 2/3 of the sum of the diameters of both branch vessels.

Suffix 2:
- 1 = Only the parent/main vessel ostium is diseased
- 2 = Only the daughter/side branch ostium is diseased
- 3 = Both ostia are diseased

Suffix 3:
- V = Looks like a ‘V’: Angle between both branches is < than 70°
- T = Looks like a ‘T’: Angle between both branches is > 70°

Suffix 4:
- Note: Adding this suffix is optional. This additional information may be important for research purposes or when comparing clinical data.
- LM = Left Main: The left main is involved
- CA = Significant Calcification

Movahed, J Invas Cardiol 2006;18:199–204
Major Limitations of Randomized Clinical Trials Involving Coronary Artery Bifurcation Interventions: Time for Redesigning Clinical Trials by Involving Only True Bifurcation Lesions and Using Appropriate Bifurcation Classification

Suggested algorithmic approach for randomized clinical trials involving coronary bifurcation intervention

Movahed. Journal of Interventional Cardiology 2011
Medina Classification

Medina et al. Rev. Esp. Cardiol 2006; 59(2): 183-4
Name the bifurcation

Why?:  - for Medina classification (which branch is the SB ?)
       - for stenting technique definition
       - for intention to treat analysis
What is our Medina classification?

- Simple
- Easy to remember
- Research classification
- Incomplete (angle, SB lesion length, Ca++ …)
- Can be completed by quantification
- Visual / base on quantification
- IVUS Medina, OCT Medina, FFR Medina …
ORIGINAL ARTICLE

Evaluation of a Strategy for Treating Bifurcated Lesions by Single or Double Stenting Based on the Medina Classification

Daniel Todaro, Francesco Burzotta, Carlo Trani, Salvatore Brugaletta, Maria De Vita, Giovanni P. Talarico, Maura Giammarinaro, Italo Porto, Antonio Maria Leone, Giampaolo Niccoli, Rocco Mongiardo, Mario Attilio Mazzari, Giovanni Schiavoni, and Filippo Crea

Instituto de Cardiología, Universidad Católica, Roma, Italy
Evaluation of a Strategy for Treating Bifurcated Lesions by Single or Double Stenting Based on the Medina Classification

- Bifurcation lesions n=120
  - Medina classification
    - Medina 1,1,1 n=25
      - Double stent as intended treatment n=25
        - Medina 1,1,1: n=17 + Other Medina types: n=3
          - Lesions treated with double stent n=20
    - Other Medina types n=95
      - Single stent as intended treatment n=95
        - Other Medina types: n=92 + Medina 1,1,1: n=8
          - Lesions treated with a single stent n=100

## Evaluation of a Strategy for Treating Bifurcated Lesions by Single or Double Stenting Based on the Medina Classification

<table>
<thead>
<tr>
<th></th>
<th>All Patients</th>
<th>Other Medina</th>
<th>Medina 1,1,1</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>In-hospital MACE, % (n)</td>
<td>2.5 (3)</td>
<td>2.1 (2)</td>
<td>4 (1)</td>
<td>.6</td>
</tr>
<tr>
<td>Cardiac death, % (n)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>NA</td>
</tr>
<tr>
<td>AMI, % (n)</td>
<td>2.5 (3)</td>
<td>2.1 (2)</td>
<td>4 (1)</td>
<td>.6</td>
</tr>
<tr>
<td>Q wave, % (n)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>NA</td>
</tr>
<tr>
<td>Non-Q wave, % (n)</td>
<td>2.5 (3)</td>
<td>2.1 (2)</td>
<td>4 (1)</td>
<td>.6</td>
</tr>
<tr>
<td>TLR, % (n)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>NA</td>
</tr>
<tr>
<td>TLR (Re-PCI), % (n)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>NA</td>
</tr>
<tr>
<td>TLR (CABG), % (n)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>NA</td>
</tr>
<tr>
<td>Cumulative 12-month MACE, % (n)</td>
<td>11.7 (14)</td>
<td>12.6 (12)</td>
<td>8 (2)</td>
<td>.4</td>
</tr>
<tr>
<td>Cardiac death, % (n)</td>
<td>0.8 (1)</td>
<td>1.1 (1)</td>
<td>0 (0)</td>
<td>.6</td>
</tr>
<tr>
<td>AMI, % (n)</td>
<td>1.7 (2)</td>
<td>2.1 (2)</td>
<td>0 (0)</td>
<td>.1</td>
</tr>
<tr>
<td>Q wave, % (n)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>NA</td>
</tr>
<tr>
<td>Non-Q wave, % (n)</td>
<td>1.7 (2)</td>
<td>2.1 (2)</td>
<td>0 (0)</td>
<td>.5</td>
</tr>
<tr>
<td>TLR, % (n)</td>
<td>12.5 (15) a</td>
<td>13.7 (13) a</td>
<td>8 (2) b</td>
<td>.5</td>
</tr>
<tr>
<td>TLR (Re-PCI), % (n)</td>
<td>11.7 (14)</td>
<td>12.6 (12)</td>
<td>8 (2) b</td>
<td>.5</td>
</tr>
<tr>
<td>TLR (CABG), % (n)</td>
<td>0.8 (1)</td>
<td>1 (1)</td>
<td>0 (0)</td>
<td>.6</td>
</tr>
<tr>
<td>Stent thrombosis, % (n)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>NA</td>
</tr>
</tbody>
</table>

AMI indicates acute myocardial infarction; CABG, coronary artery bypass graft; MACE, major adverse cardiac events; NA, not available; PCI, percutaneous coronary intervention; TLR, target lesion revascularization.

*aIn one case, the same patient underwent both percutaneous and surgical TLR.

*bCrush technique in both cases.
Quantification of a coronary bifurcation stenosis
Structure-function scaling laws of vascular trees

Murray’s law
\[ D_1^{3*} = D_2^{3*} + D_3^{3*} \]

Finet’s formula
\[ D_1 = (D_2 + D_3) \times 0.678 \]

* 2.3 (Huo-Kassab)

Adapted from G. Kassab
Traditional QCA for coronary bifurcation quantification: reference, MLD, %
**Interventional Rounds**

Quantitative Angiographic Methods for Bifurcation Lesions: A Consensus Statement from the European Bifurcation Group

Alexandra Lansky, MD, Joan Tuinenburg, MSC, Marco Costa, MD, Micheal Maeng, MD, Gerhard Koning, MSC, Jeffrey Popma, MD, Ecatarina Cristea, MD, Laurence Gavit, MD, Ricardo Costa, MD, Andrei Rares, MD, Gerritt-Ann Van Es, PhD, Thierry Lefevre, MD, Hans Reiber, PhD, Yves Louvard, MD, and Marie-Claude Morice, MD, on behalf of the European Bifurcation Angiographic Sub-Committee

The treatment of bifurcation lesions is complex and increasingly common. A growing number of dedicated bifurcation devices are under clinical evaluation, but no standardized methodology exists. Specifically, the angiographic analysis of bifurcation lesions is not standardized and current QCA packages are not designed for bifurcation lesions. This consensus statement outlines the limitations of conventional QCA in the bifurcation application, and outlines a new standard approach for the analysis and reporting of the angiographic results of the bifurcation lesion allowing for future trial and device comparisons and mechanistic insight into location and modes of treatment failure.

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Key words: quantitative coronary angiography; diagnostic cardiac catheterization; percutaneous coronary intervention
Dedicated bifurcation QCA
Bifurcation
Quantitative Angiographic Methods for Bifurcation Lesions: A Consensus Statement from the European Bifurcation Group

Subsegment analysis demonstrating the various segments that should be analyzed and reported separately

Lansky, CCVI 73:258–266 (2009)
Classification of bifurcation stenting techniques
Classification of Treatments

A

B

C

D

Hi Tech meeting 1997
Classification of Treatments

A Technique
1
2

B Technique
5

C Technique
7

D Technique
8

«Skirt» Technique

Y. Louvard, Heart 2004; 90:713 –722
Schematic description of interventional bifurcation techniques:

OST = one stent technique;
SBT = stent with balloon technique;
KST = kissing stent technique;
TST = "T" stenting technique;
CRT = crush stenting technique;
CUT = culotte stenting technique

Main prox. first

1st stent
PM stenting

After balloon
Skirt

2 stents
Skirt + DM
Elective T stenting

3 stents
Extended V

Main across side first

1st stent
MB stenting across SB

After balloon
Skirt
MB stenting + SB balloon
MB stenting + kissing

2 stents
Elective T stenting
Internal crush

3 stents
Trouser legs and seat

Double first

1st stent
DM stenting
Provisional SKS

After balloon
SB ostial stenting
SB minicrush
SB crush

2 stents
V stenting
SKS
Syst. T stenting
Minicrush Crush

Side branch first

1st stent
SB ostial stenting

After balloon
SB ostial stenting

2 stents
SB ostial stenting

3 stents
SB ostial stenting
<table>
<thead>
<tr>
<th>M</th>
<th>A</th>
<th>D</th>
<th>S</th>
</tr>
</thead>
<tbody>
<tr>
<td>Main prox. first</td>
<td>Main across side first</td>
<td>Distal first</td>
<td>Side branch first</td>
</tr>
<tr>
<td>1st stent</td>
<td>Inv. MB stenting across SB</td>
<td>Inv. Provisional SKS</td>
<td>DM ostial stenting</td>
</tr>
<tr>
<td>After balloon</td>
<td>MB to SB stenting + DM balloon + kissing</td>
<td>DM minicrush</td>
<td>Inv. Syst. Minicrush Crush</td>
</tr>
<tr>
<td>2 stents</td>
<td>Inv. Elective Internal T stenting</td>
<td>Inv. Culotte</td>
<td>Inv. T Stenting</td>
</tr>
<tr>
<td>3 stents</td>
<td>Inv. Internal T stenting</td>
<td>Inv. TAP</td>
<td></td>
</tr>
</tbody>
</table>
Two-stent strategies for bifurcation lesions: which vessel should be stented first, the main vessel or the side ranch?

Adjusted incidences of cardiac death, MI and stent thrombosis using inverse probability weight (A) and standardized mortality/morbidity ratio weight (B)

MB stenting accross SB first

- MB stenting with protection
- MB stenting with SB balloon

1st stent

- Elective T stenting
- Internal Crush
- Culotte
- « Buchbinder ? »

2nd stent

Proximal MB stenting first

1st stent

- Proximal MB stenting

2nd stent

- Helqvist
- Skirt technique

3rd stent

V stenting

V + proximal stenting

½ SKS

SKS

SB ostial stenting

SB Crush stenting

1st stent

Systematic T stenting

1st and 2nd stent

V stenting

1st and 2nd stent

V + proximal stenting

3rd stent

½ SKS

SKS

Wires


Intention

Inverted
Conclusions

• Better definition of bifurcation stenosis? Possible? Useful?

• Do we need another classification than the Medina’s? No…

• Classification of treatments: how to take into account the complexity of techniques?

• What is missing?