INTRODUCTION

Bifurcation stenosis is one of the most complex coronary lesions requiring endovascular treatment because the lumen of both the main vessel and the side branch needs to be restored. The best approach for the management of a bifurcation to achieve optimal procedural outcomes and, more importantly, long-term success with low restenosis rates and low major adverse clinical event (MACE) rates is still debated.

(Berger PB,et al.,2000)

True bifurcation lesions, representing up to 16% of coronary targets for intervention, have been associated with higher peri-procedural complication rates and lower long-term patency rates, In the contemporary interventional era, several approaches have been proposed to treat bifurcation lesions, Coronary stents improve the immediate angiographic results by reducing lesion recoil and achieving better scaffolding, but stents are associated with increased thrombotic complications and later restenosis in bifurcation lesions.

(Moses JW,et al.,2004)

When compared with nonbifurcation coronary interventions, bifurcation interventions have historically reported a lower rate of procedural success, higher procedural costs, longer hospitalization, and higher clinical and angiographic restenosis, Consequently, the treatment of coronory bifurcation lesions represents a challenging area in interventional cardiology. However, recent advances in stent

design, selective use of a 2-stent technique, acceptance of a suboptimal SB result, and various percutaneous techniques (high-pressure postdilatation, kissing balloon inflation, and intravascular ultrasound) have led to a dramatic increase in the number of patients with bifurcation lesions who are being successfully treated with excellent long-term outcome

(Pan M,et al.,2004)

Balloon angioplasty alone to treat bifurcation lesions has resulted in relatively low angiographic success and high restenosis rates, Although the introduction of bare-metal stents (BMSs) resulted in more predictable results and higher success rates, angiographic restenosis rates still remained high. The introduction of drug-eluting stents (DESs) in clinical practice has altered the treatment perspective when dealing with this type of lesion; however, abrupt side-branch closure with the single-stent strategies, together with the risk of thrombosis and restenosis associated with the complex two-stent techniques.

(Vegna C,et al.,2007)

CLASSIFICATION:

Bifurcation lesions are variable not only in their anatomy (eg, location of plaque, plaque burden, angle between branches, site of bifurcation, size of branches) but also in the dynamic changes in anatomy during treatment (dissections and carina shift). As a result, there are no 2 identical bifurcations, and hence, there is no single strategy to be used on every bifurcation.

Bifurcations vary in plaque burden, the location of plaque, the angle between branches, the diameter of the branches and the bifurcation site. No two bifurcations are identical, and no single strategy exists that can be applied to every bifurcation. Thus, the more important issue in bifurcation PCI is selecting the most appropriate strategy for an individual bifurcation.

(Medina A,et al.,2006)

The most frequently used older bifurcation classification is the Lefevre classification , However, Lefevre and other older classifications of coronary bifurcation lesions require significant efforts of memorization, The Medina classification is a simplified and universal classification of bifurcation lesions, is straightforward and does not need to be memorized, even though it provides all the information contained in the others.

Coronary bifurcations have been previously classified according to both the angulation between the MV and the SB and the location of the plaque burden. Depending on the degree of SB angulation, a bifurcation lesion can be classified as (1) “Y-angulation” (when the angulation is <70°; access to the SB is usually less difficult but plaque shifting is more pronounced, and precise stent placement in the ostium is more difficult) and (2) “T-angulation” (when the SB angulation is more than 70°; access to the SB is usually more difficult but plaque shifting is often minimal, and precise stent placement in ostium is more straightforward).

There have been multiple classifications proposed to morphologically distinguish bifurcation coronary lesions over the past several years, primarily based on the presence of disease in the main branch alone, side branch alone, or both, Each classification scheme differs only slightly in how it describes the presence of disease in the main branch proximal and/or distal to the level of the carina, as well as disease in the side branch ostium.

It has been widely held that certain lesion characteristics may predict treatment success using currently accepted techniques and DES platforms. Despite this assertion, none of these widely used classification schemes, based solely on anatomic distribution of disease, has been proven to be sufficiently predictive of procedural success.

(Movahed MR,et al.,2006)

Any successful treatment strategy for bifurcation lesions must factor in a wide variety of anatomic considerations. While the distribution of disease in the main and side branch vessels is critical, so to are issues of side branch angulation, extent of lesion calcification and fibrous tissue buildup, as well as vessel diameter. If one likens the heterogeneity of bifurcation coronary lesions to that seen with fingerprints, it becomes clearer why the one-size-fits-all approach is not appropriate for bifurcation revascularization techniques or for dedicated devices; no single strategy has been shown to suffice.

There are 2 classification patterns commonly used to describe bifurcation plaque distribution: the Duke classification and the Medina classification Both of these classifications underestimate plaque distribution and plaque burden when compared with intravascular ultrasound and do not take into account the fate of the SB on dilatation of the MV.

A new, simple, practical, and prognostic classification of bifurcation lesions has been suggested by Movahed that takes into account the size of proximal MV , which is very important while considering 1- or 2-stent techniques.

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

A

Type I

True bifurcation lesion

Type

One asymmet where branch is

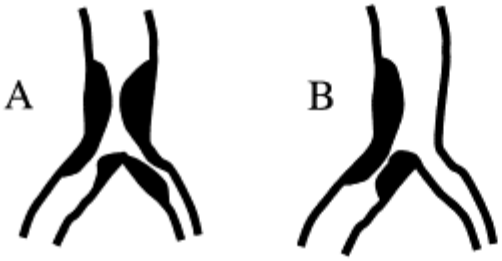
Type IV

Lesion in the parent vessel either before or after the take of a side branch that may or may not have additional osti disease

B

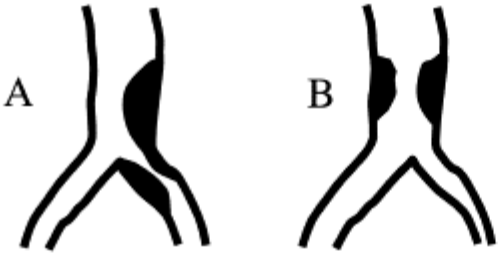
Type I

Parent vessel stenosis proximal and distal to bifurcation



Type II

Parent vessel stenosis proximal to bifurcation



Type III

Parent vessel

Type 1

Lesions located in the main proximal and distal, and the side branch

Type 2

Lesions located only in the branch, proximal and distal, the ostium of side branch

Type 3

Lesions located in the main proximal to the bifurcation

Type 4

Fig. 1 Duke classification of bifurcation lesions based on the location of the obstructive plaque.

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

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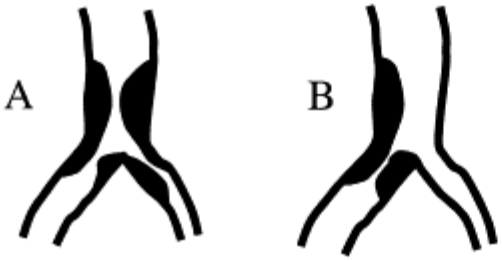
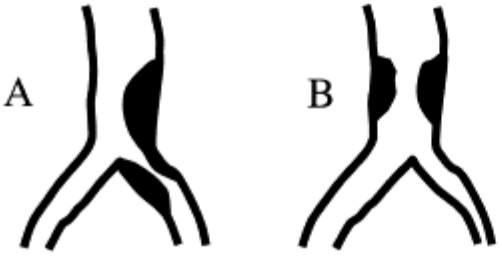


Type II

Parent vessel stenosis proximal to bifurcation



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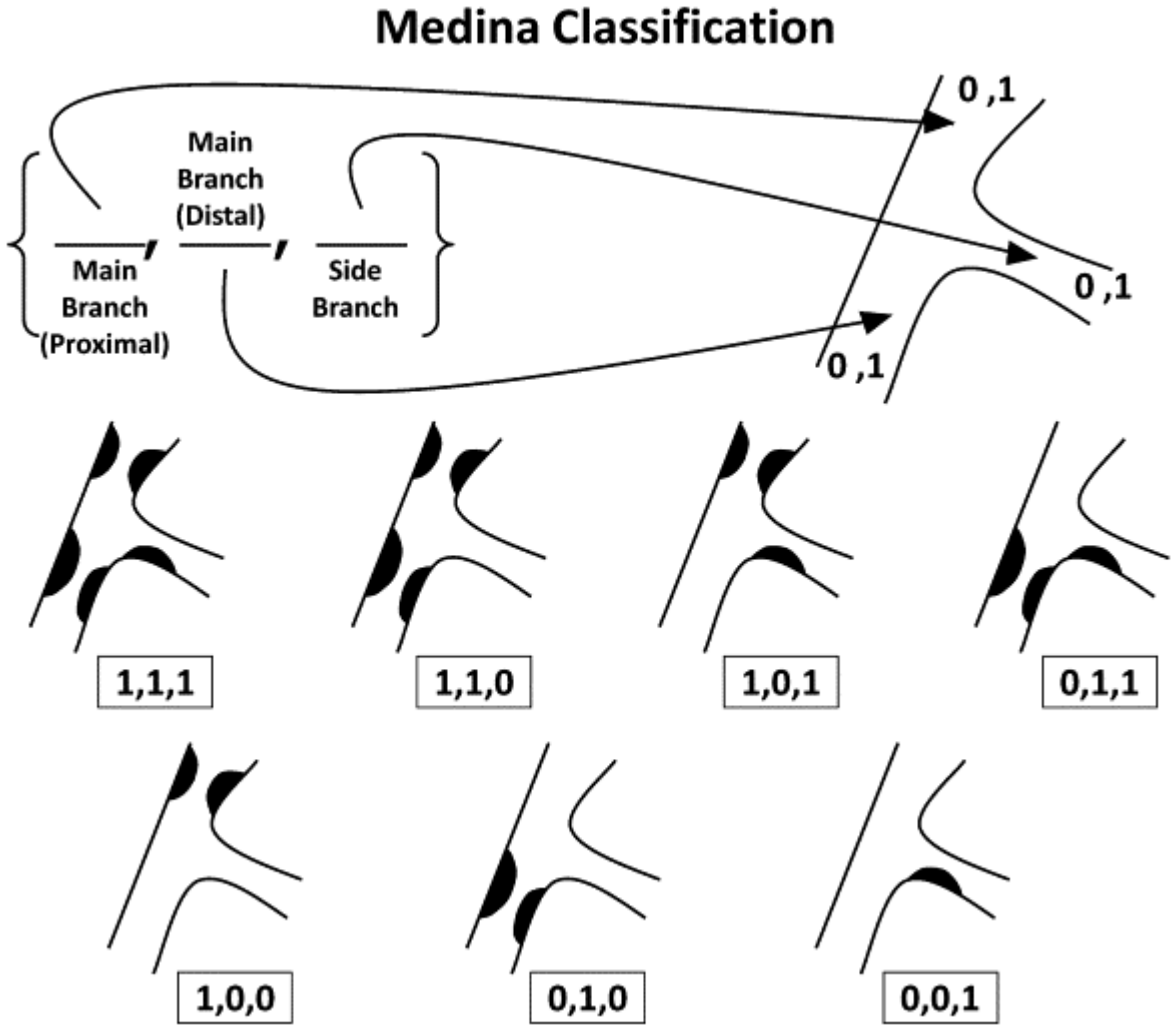


Medina classification of bifurcation lesions based on the location of the obstructive plaque. Number 1 is assigned to the location of plaque.

* Mohaved classification incorporating location of the obstructive plaque, vessel size, and angulation, with optional suffix for further details.

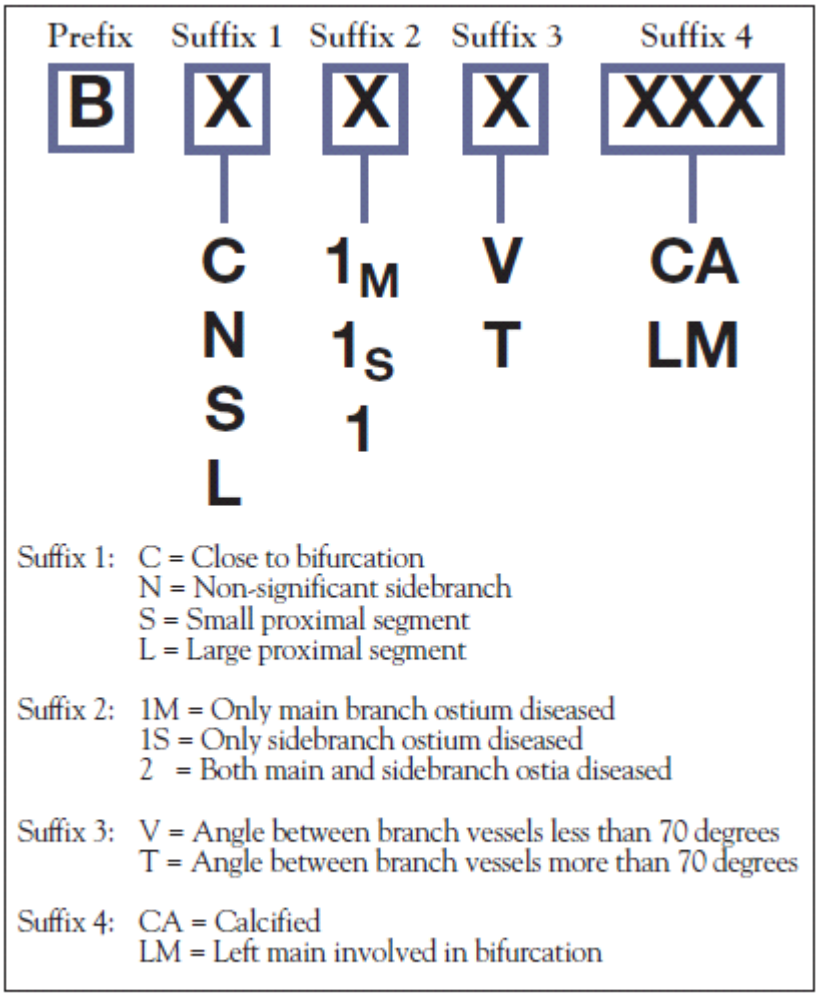
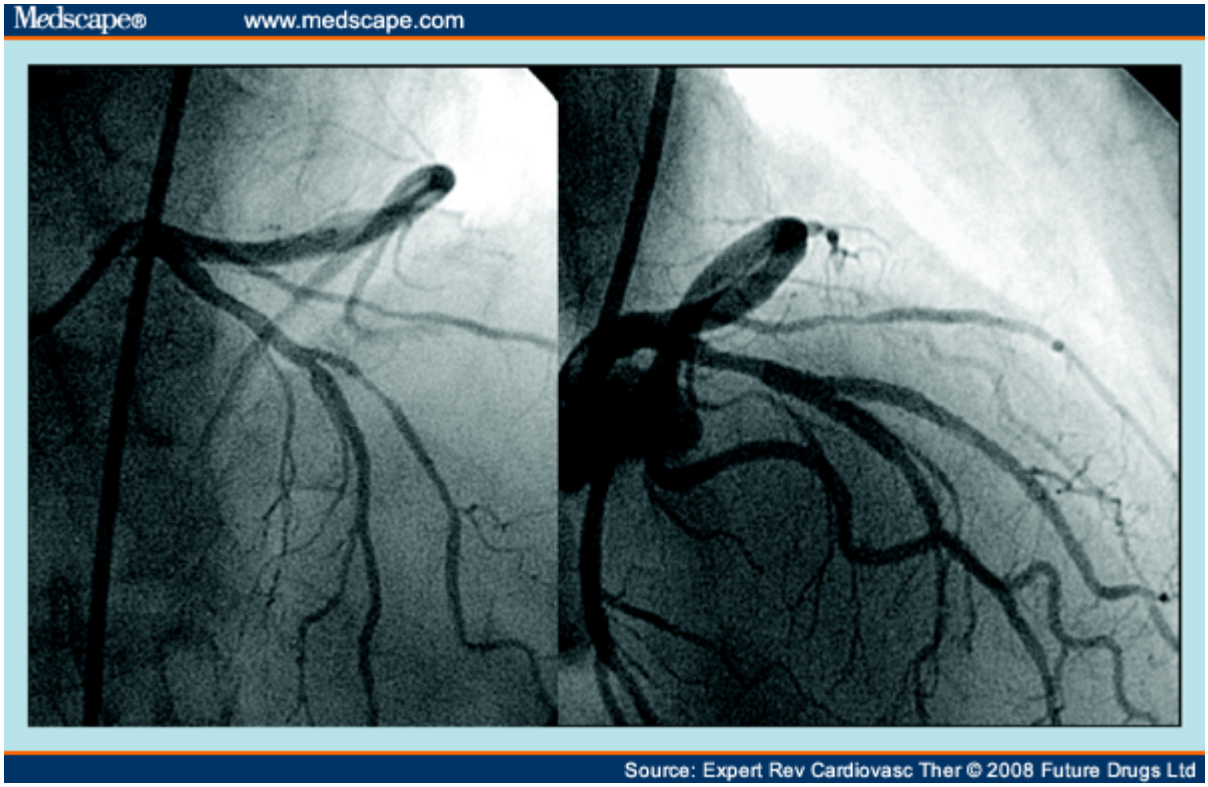
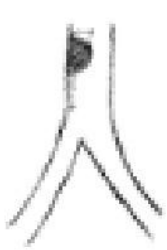


Figure 2. *Movahed's classification. Originally published in the Journal of Invasive Cardiology 2006;18:199–204.*

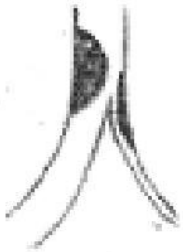


An example of a BL2V lesion. A bifurcation lesion with a large proximal segment and involvement of both ostia (2) with an angle of less than 70° (V) between the branches that was successfully treated using the kissing stent technique

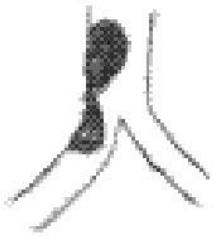
BC



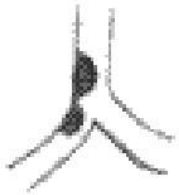
BN



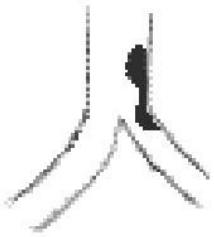
$BL1_MV$



$BS1_MV$

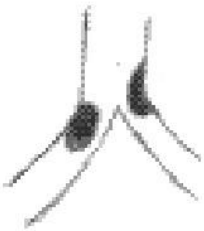


$BL1_SV$

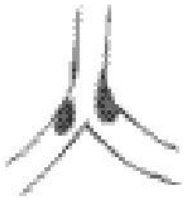


$BS1_SV$

BL2V



BS2V



$BL1_MT$

$BS1_MT$

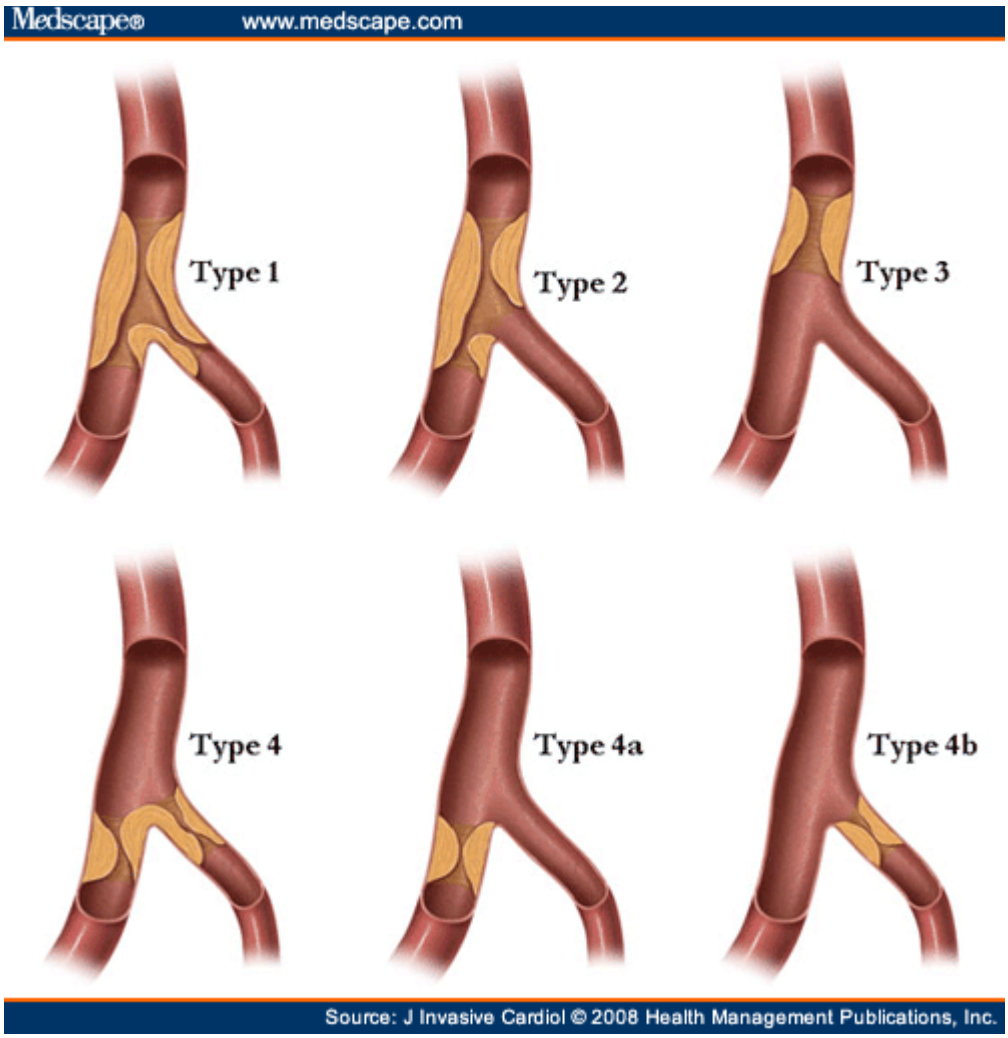
$BL1_ST$

$BS1_ST$

While certain lesion characteristics are associated with better revascularization results, the nonuniformity of bifurcation lesions has made it impossible to reliably classify lesion types in any meaningful way with regard to expected outcomes. The ACC/AHA Lesion Classification System, commonly used for describing CAD, does not apply of several bifurcation classifications published in the literature.

The Lefevre system is widely recognized. This system focuses primarily on describing the basic anatomic patterns of plaque distribution , The categories described, however, have no meaningful correlation with outcomes, Most disease patterns described by Lefevre are not "true bifurcation" lesions (Lefevre Type 1) at the time of diagnostic angiography, but instead may degenerate into bifurcation disease once instrumentation of the vessel occurs. This phenomenon of lesion architecture changing during revascularization procedures has been encountered by most interventionalists. Plaque redistribution at the carina of the so called "pseudobifurcation" lesion (Lefevre Types 2, 3 and 4) can occur, and resultant reconfigurations may then require the application of bifurcation therapies.

(Lefevre T,et al.,2000)



The Lefevre classification of bifurcation coronary artery disease. Type 1: disease involving the main branch (both proximal and distal to the carina) as well as at the side branch ostium; Type 2: disease confined to the proximal and distal main branch, but not involving the side branch; Type 3: disease located only in the main branch proximal to the vessel carina; Type 4: disease confined to the ostium of each branch distal to the carina (4a main branch and 4b side branch) without disease proximal to or at the level of the carina).

(Lefevre T, Louvard Y,et al.,2000)