Coronary dissection- back to the future- finding good in the bad!

Rajkumar Natarajan[a,b] MBBS, MRCP, Natasha Corballis[a,b] MD, MRCP, Ioannis Merinopoulos[a,b] MD, MSc, MRCP, Vassilios S Vassiliou[a,b #] MD, PhD, FESC, FRCP, FACC, Simon C Eccleshall[a #] MD, MRCP

[a] Department of Cardiology, Norfolk and Norwich University Hospital, UK; [b] Norwich Medical School, University of East Anglia, UK

[#] VSV and SCE contributed equally

Address for correspondence

Dr. Rajkumar Natarajan

2.06 Bob Champion Research & Education Building

Norwich Medical School, University of East Anglia

Norwich, NR4 7TJ

Email: r.natarajan@uea.ac.uk, Telephone: +44 (0)1603 59 2534

Abstract

It has been recognised for decades that dissections occur as a mechanism of balloon angioplasty. A successful angioplasty result contains some degree of intimal splitting and disruption which usually heals well. Nonetheless, some dissections are extensive leading to serious ischemic complications. The evolution of therapeutic coronary dissection concept started in the 1970s and seems to be a favourable mechanism for drug delivery in the current era of drug coated balloons. The primary focus of this article will be on studies undertaken to understand the mechanism of balloon angioplasty and morphological changes in the plaque post balloon angioplasty. In the early days of balloon angioplasty, there was an enormous

interest in dissections mainly to prevent acute vessel closure events and to address the importance of their occurrence in relation to vessel restenosis. We will review the historical background, studies defining clinical, angiographic and morphological patterns of dissection spectrum and various currently evolving management strategies.

1. Introduction

Forty seven years ago, Andreas Gruentzig introduced "Percutaneous transluminal coronary angioplasty" (PTCA) as a new approach to treat symptomatic occlusive coronary artery disease[1]. The mechanism of balloon angioplasty (BA) formed the centrepiece around which newer equipment and technology were developed including plain old balloon angioplasty (POBA), bare metal stents (BMS)[2], drug eluting stents (DES)[3] and drug coated balloons (DCB)[4], each addressing specific risks of earlier technologies. Coronary artery dissection is a frequent result of the vessel injury caused by balloon dilatation. IVUS studies have demonstrated its presence in 50-80% of procedures[5,6]. With the return of balloon angioplasty with DCB technology, dissection is now frequently accepted to achieve a 'DCB only' approach. Having stents on the shelf, there is room for aggressive, yet controlled lesion preparation. However, coronary dissections remain the most common reason for bail out stenting in various DCB studies[7–10]. Moreover, the outdated National Heart, Lung, and Blood Institute (NHBLI) angiographic classification is still in use and, of note, has not been validated or updated recently.

During the POBA era, coronary dissection leading to acute or subacute abrupt closure was the most feared complication[11] and attempts were made to identify the clinical and angiographic predictors in order to prevent and manage any such dissections. The purpose of this article is to review the historical background, studies defining clinical, angiographic and

morphological patterns of dissection spectrum and various currently evolving management strategies.

2.Pre stent era 1977-1990

2.1 Historical background of coronary dissections

2.1 Coronary dissection- an inherent risk of BA

History was made on 16th September, 1977 when Andreas Gruentzig performed the first transluminal balloon catheter inflation of a discrete left anterior descending artery (LAD) stenosis in a 38 year old male patient[1] by using a modified non steerable balloon dilation catheter . When the results of this novel innovation were presented at the American Heart Association meeting in November 1977, it was well received and resulted in wider adoption[12]. In March 1979, the Cardiac Diseases Branch of National Heart, Lung, and Blood Institute (NHLBI) began centrally accumulating baseline and follow up data to gain knowledge of the acute and long-term results of PTCA[12] . According to the PTCA Manual of operations, "coronary intimal dissection (intimal tear) was defined as the presence of angiographically evident intimal damage producing either an intraluminal filling defect or extraluminal extravasation of contrast material; coronary dissection was considered a complication of PTCA if it caused major luminal obstruction or was associated with coronary occlusion, myocardial infarction(MI), or deterioration of flow necessitating emergency coronary artery bypass surgery(CABG)"[13].

The 1983 complication report of NHLBI PTCA registry showed an overall rate of 9.4% intimal tear or coronary dissection in the initial 1977-1981 cohort of 1500 patients from 73 participating centres and 31% of such dissections led to major complications of MI, CABG or death[13]. With an unpredictable occurrence, coronary dissection was recognised as the leading

cause for abrupt vessel closure[14] and became the most common indication for emergency surgery[15] which also carried high early morbidity and mortality risks[16]. With substantial technological advances and more refined tools, the success rates and indications for PTCA expanded exponentially even in more complex and high risk patients[12]. Despite improved efficacy, the incidence of complicated dissections remained unchanged in 1985-1986 NHLBI cohort [17] . Meanwhile, concerns were also raised that intimal dissections accelerated early restenosis[18–20].

The credibility of the novel technology faced challenges due to the limited means of managing the acute vessel complications, high rates of restenosis and their associated costs. In recognition of the incidence of unavoidable dissections, research efforts continued for many years to study the pathophysiology, clinical and angiographic risk predictors and to define the precise relationship between intimal dissection and restenosis. Management strategies also evolved from emergency CABG to a variety of repeat angioplasty techniques during early POBA years and subsequently bailout stenting.

2.2 Patho morphology of coronary dissections

2.2.1 Coronary dissections under microscopy

Balloon angioplasty results in dilatation of the vascular lumen with the underlying mechanism being attributed to redistribution and compression of atheromatous plaque by Dotter[21] and Gruentzig[22] but this was never proven. The exact mechanism remained ill-defined with various theories proposed. In 1981, Block et al. described plaque splitting at its thinnest portion in two patients post BA[23]. Waller et al., in 1983, studied early histological changes occurring during 4hrs to 30 days post BA in several patients and the following series of possible mechanisms were reported: intimal tears or cracks or fractures with variable

degrees of localised or extensive medial penetrations, intimal-medial dissections propagating either antegrade or retrograde or both directions and sometimes lifting of the plaque from the deep medial layer[24]. The deep extensive medial dissections may result in a propagating intramural hematoma subsequently occluding the lumen. The extensive intimal-medial dissection plane may lift plaque from media creating a large flap that eventually curls up in the lumen causing abrupt vessel closure. Similar patterns of intimal or medial splitting, plaque fractures and haemorrhage were described in autopsy studies[25,26].

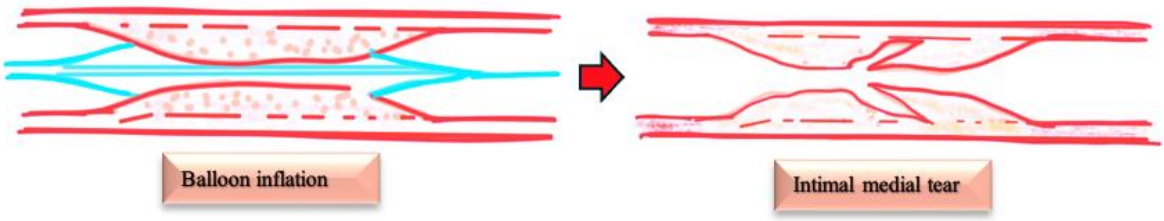


Figure1. Mechanism of balloon angioplasty.

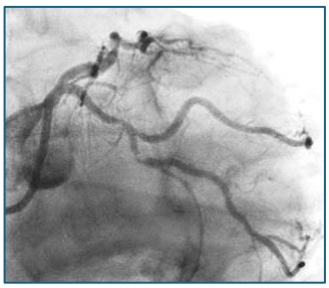
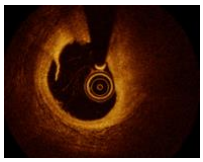
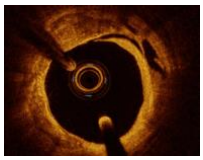
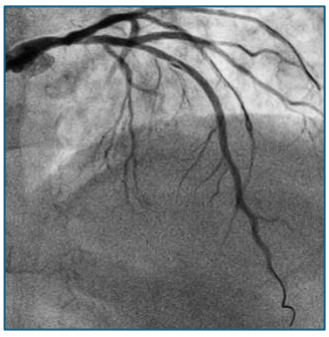
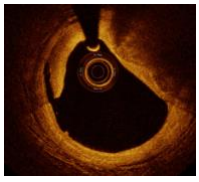
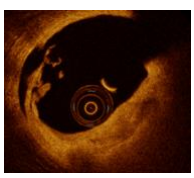
2.2.2 Coronary dissections under angiography

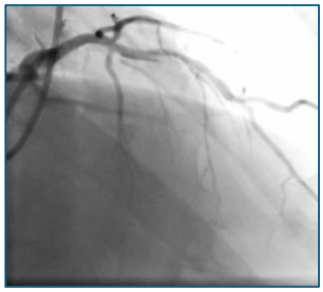
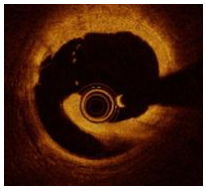
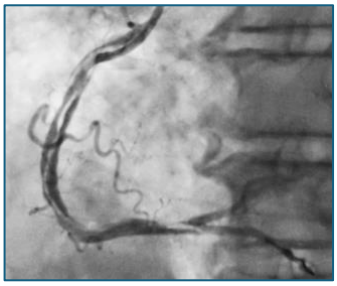
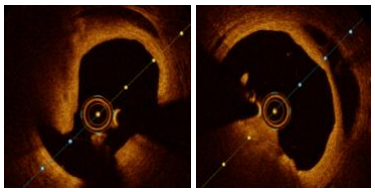
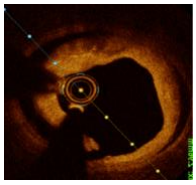
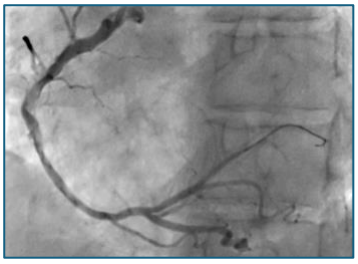
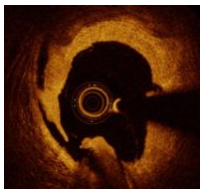
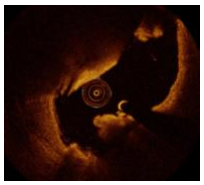
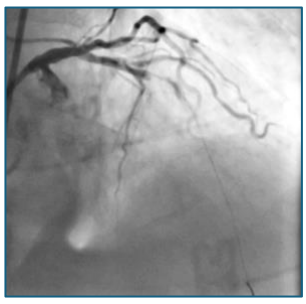
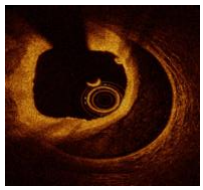
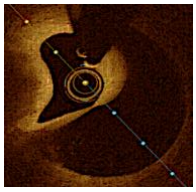
The terms "intimal tears or flaps" and "dissection" were used freely to describe the above spectrum of morphological alterations on angiography but in reality, the result may give diverse angiographic findings often dictated by the lesion characteristics. In order to classify the angiographic appearances following PTCA, Holmes et al., first described four distinct patterns of immediate angiographic changes in 100 patients, namely smooth-walled dilatation, intimal flaps or intramural split or dissection, intraluminal haziness and no change in lesion but there was no anatomical correlation[27]. Dorros et al showed safe healing of intimal dissections following PTCA[28] and on further analysis of early PTCA registry, about two-thirds of the angiographically detected dissections (9.2%) had a benign course[13] post PTCA. It was concluded by the NHBLI PTCA registry that angiographic patterns of intraluminal filling defect, linear luminal density or staining and extravasation of contrast

with good distal flow are called coronary intimal dissection[14]. When a complicated dissection occurs, it is rapidly seen as well-defined long intramural contrast channels, large radiolucent spiral tracts with persistence of contrast material, irregular lumen with contrast hang up, delayed flow, and abrupt closure. Subsequently Dorros and Guiteras Val et al devised the widely used NHLBI angiographic classification of dissections (Type A-F) in 1985 based on the cine-loop fluoroscopy images of the patients from the PTCA registry[17]. Clinically, type A, B and stable type C were classified as uncomplicated whereas type C with suboptimal hemodynamic results, type D and F represent complicated dissection. Type E could be a combination of dissection with thrombus.

This system is useful in providing distinct angiographic categories as shown in table 1, but its application was limited during the late POBA era.

Table 1 – NHLBI classification of types of coronary artery dissections during angioplasty.

Types	Description	Angiographic illustration	Angiographic appearance	OCT appearances
A	Minor radiolucencies within the lumen during contrast injection with no persistence after dye clearance		 LAD in LAO caudal view	  Images of Intimal tears or dissections with intact medial layer
B	Parallel tracts or double lumen separated by a radiolucent area during contrast injection with no persistence after dye clearance		 LAD in PA cranial view	  Images showing intimal medial flaps detached from external elastic lamina in heavily calcified lesions

C	Extraluminal cap with persistence of contrast after dye clearance from the lumen		 LAD in RAO cranial view	 Extensive dissection with detachment of intimal medial flap from adventia forming a false lumen
D	Spiral luminal filling defects		 RCA in LAO view	  Images showing spiral dissection involving the target lesion in a coronary vessel
E	New persistent filling defects		 RCA in LAO view	  Images of deep complicated dissection with thrombus subsequently causing luminal compromise due to intramural hematoma in the second picture.
F	Non A-E types that lead to impaired flow or total occlusion		 LAD in RAO cranial view	  Images showing false lumen with hematoma compressing the true lumen causing abrupt vessel closure.

(LAD- left anterior descending artery; RCA- right coronary artery; LAO- left anterior oblique view; RAO- right anterior oblique view). OCT images in fifth column depict the counterparts

of each NHLBI dissection types and they are not the actual OCT runs of the angiogram images shown in column four.

2.3 Angiographic-morphological correlations

The correlation of angiographic appearances with the morphological patterns of balloon angioplasty mechanisms was well described by Bruce. F. Waller in 1988[29]. He examined histopathological specimens of 76 coronary artery segments containing angioplasty sites from 66 necropsy patients who died within 30 days of PTCA and compared the angiographic description by different PTCA operators with the anatomical findings. Interestingly, the angiographic 'intimal flaps' (43%) and 'intraluminal haziness' (38%) correlated mostly with intimal-medial splits or crack of varying degrees with localised medial dissection. Four extensive medial dissections (9%) were seen in the intimal flap category whereas the haziness pattern had a mix of pure intimal injuries (31%) and laminated thrombus coating (3%). In a 'coronary artery dissection' site, deep intimal-medial tear (figure 2) with an extensive longitudinal medial dissection had occurred. Adventitial extension was seen in 2 patients with 'extravasated contrast' material correlating with confined coronary perforation (confined rupture). In his further works, there were evidence of regression of these intimal flaps with no signs of previous vessel injury histologically[30].

Figure 2: Diagram showing morphological correlates of angiographic appearances of intraluminal flaps and haziness.

2.4 A morphologic-angiographic-clinical nomenclature of dissections

The histopathological studies added to the existing angiographic terms providing an anatomical perspective. The histological definition of dissection was penetration of medial layer. Angiographic coronary artery dissection describes a visible intimal flap (equivalent to intimal-medial tear anatomically) with contrast staining extending beyond the confines of angioplasty lesion with or without clinical symptoms or signs of ischemia[30]. Such intimal flaps could either extend circumferentially in short axis view or propagate in anterograde or retrograde directions longitudinally in long axis plane as shown in figure 3. Using different angiographic projections, an estimate of the biplanar extent is possible. In theory, a dissection involving >50% of short-axis circumference or >1cm anterograde or retrograde of long-axis length was defined as complication of angioplasty whereas anything below those cut-offs are mechanisms. On the contrary, intimal dissection or split described an intimal flap with contrast staining with no evidence of ischemia[30,31].

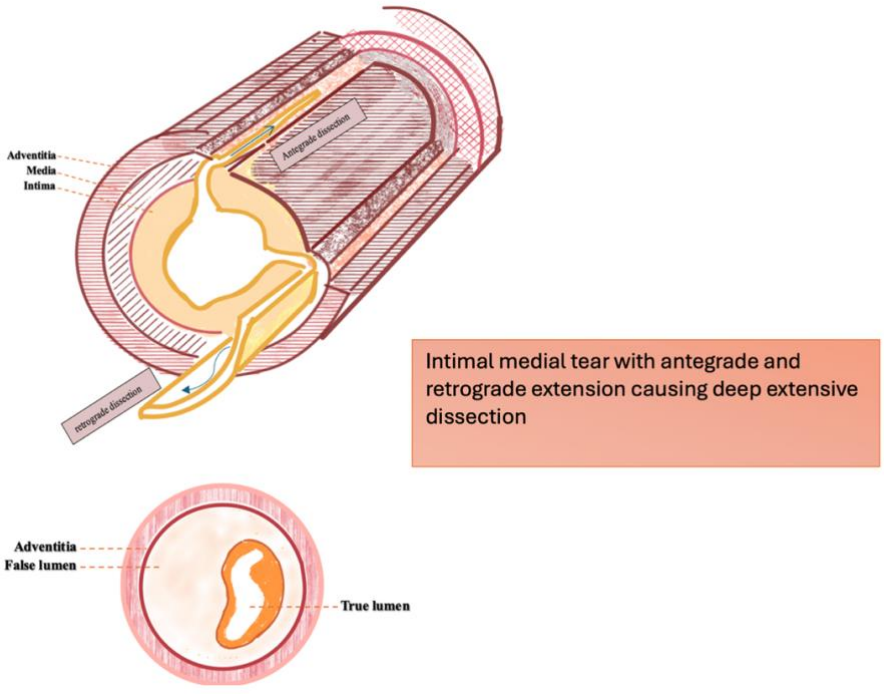


Figure 3: Diagram showing morphological correlation of angiographic appearances of complicated cornary arterial dissection caused by deep extensive medial dissection creating a false lumen and compressing the true lumen.

2.5 Therapeutic dissections – Uncomplicated and safe to leave category!

During the early years of angioplasty, concerns were expressed that intimal tears or dissections in general accelerate early restenosis[18–20]. To address this, in 1985, Leimgruger et al.[32] studied the hemodynamic significance of uncomplicated angiographic coronary intimal dissections by measuring transtenotic pressure gradient following successful PTCA and their relationship with restenosis by using a validated digital electronic caliper method[33] to measure diameter stenosis severity (DS%). A transtenotic pressure gradient was obtained using a guide catheter, guidewire and a balloon catheter. By positioning the guidewire and balloon catheter across the coronary stenosis, a pressure port distal to the balloon segment recorded the distal arterial pressure while the guiding catheter tip at the ostium monitored the proximal arterial pressure. The difference in these phasic pressures yielded the transtenotic pressure gradient by a specialised computer program. Utilising this technology, the authors demonstrated that such dissections did not increase the risk of restenosis and had a beneficial effect of lower restenosis rate if the final transtenotic pressure was ≤15mm Hg. In fact, if the final gradient was greater than 15mm Hg, the rate of restenosis was not significantly different between the groups with and without intimal dissections [35% vs 39%; p= non-significant(NS)]. Matthews et al then showed that patients with dissections during PTCA are unlikely to develop restenosis at one year follow up[34]. In this observational study of 273 patients, 82% of the dissection group did not develop restenosis. Similar retrospective studies during the same year showed a similar relationship between intimal dissection and restenosis[34–39]. The term ‘therapeutic dissection’ was widely used to describe the uncomplicated coronary intimal dissections resulting in

increased cross-sectional area that were less likely to develop restenosis[39,40]. Table 1 summarises the studies in which the relationship between lesions with or without dissections and restenosis was examined.

Table 2 : Summary of studies that examined the relationship between lesions with or without dissections and restenosis

First author	Year	Patients	Angiogram f/u (%)	Dissection (% of lesions)	Restenosis at f/u(%), (with vs without dissections)	P value
Leimgruber et al.[32]	1985	1650	60	25ϕ	19% vs 28% gradient ≤15	<0.05
Matthews et al.[34]	1988	216	30	34	18% vs 23%	NS
Black et al.[41]	1988	384	39	34	29% vs 32%	NS
Quigley et al.[42]	1989	114	88	20	35% vs 31%	NS
Renkin et al.[43]	1990	278	47	33	38% vs 31%	NS
Bourassa et al.[44]	1991	307	80	41	33% VS 36%	NS
Hirshfield et al.[45]	1991	694	73	39	40% vs 39%	NS

Angiogram f/u = percent of patients with angiographic follow up; gradient = final transtenotic pressure gradient in mmHg; ϕ uncomplicated dissections ; NS = not significant, f/u = follow up.

2.6 Complicated dissections- Indeterminate category and 'need to graft' category

As described above, complications occur when an intimal-medial tear produces a flap that could fold or become free in the lumen, collapsing on itself causing intussusception and thus compromising the lumen[29]. Tissue disruption may produce turbulence (shear stress) , stasis and thrombosis leading to suboptimal hemodynamic results[46,47]. If intervened promptly in suitable cases, the flaps can be made to adhere to the vessel wall by further dilatation techniques to

restore distal flow[48]. If the flow is compromised despite rescue strategies, the earliest mode of definitive therapy was emergency surgery as explained below.

In essence, the challenge is the interpretation of dissections by angiography. When intimal damage occurs exposing the thrombogenic plaque layers, the contrast fills these furrows during further injections. Angiography reveals hazy or ill-defined margins of an enlarged lumen with inhomogeneous opacification or a double line or contrast filling defects. Whatever the mechanism may be, not all the dissections are visible and not all visible dissections are complicated. Sometimes a combination of angiographically undetectable dissection, recoil, refractory spasm and intracoronary thrombus can occur. This is a conundrum still faced today where an indeterminate angiographic appearance needs to be classified as either safe or unsafe. Hence, safe dissections may be viewed as a therapeutic mechanisms of balloon angioplasty whilst unsafe dissections represent a complication of dilatation.

3. Existing Classification systems

3.1 NHBLI angiographic classification of dissections-1985

Types A-F, as shown in table 1, represent angiographic appearances of the contrast in relation to their clearance and their effect on distal flow as described in 1985. Neither the circumference nor the length is factored in. It was only in 1991, Huber et al. predicted clinical outcomes of dissections using NHBLI classification retrospectively[38]. Of 691 dissections, 543 were type B that had no higher risk of morbidity and mortality compared to patients with no dissections. A small subgroup of types C-F (n=148) had a statistically significant increase in in-hospital complications against type B. The results are severely limited by low power in the subgroup and unreported inter-and intraobserver variability. In contrast to these results, slightly better clinical outcomes were observed with type B-F dissections in an unblinded

MERCATOR trial by Hermans et al. in 1992[39]. As shown in table 3, there is no significant difference in long term events between the different NHLBI types of dissections. It is difficult to draw any strong conclusion from these studies with conflicting results but the concept of therapeutic dissections (roughly type A-C, some type Es if thrombus clears) strengthened. Nevertheless, the decision to treat a dissection depended on the clinical and haemodynamic parameters that prevailed in relation to the distal perfusion and still applies in current practice.

Table 3 : Summary of studies that examined the relationship between lesions with dissections, NHLBI types and their outcomes.

First author	Year	Patients	NHLBI Dissection types and number of dissections (%)	NHLBI dissection types with acute complications, n,(%)	NHBLI dissection types with Late events, n, (%)
Huber et al.[38]	1991	691	B 543 (78.6%) C 62 (9%) D 33 (4.8%) E 18 (2.6%) F 35 (5.1%)	B 17 (3.1%) C 6 (9.7% D 10(30.3%) E 7 (38.9) F 24 (68.6%)	NR
Hermans et al.[49]	1992	693	247 (32%) NHLBI A 76 (11%) B 136 (19.6%) C 33 (4.8%) D 3 (0.4%) E 3 (0.4%) F 1 (0.1%)	NR	TLR A 12 (15.8%) B 18 (13.2%) C 2 (6.1%) D-F 0
Albertal et al.[49]	2001	256	A-B – 100 C- 32	A-B-- 3(2%) C - 1(2%)	A-B -- 11(11%) C - 4(13%)

Table 3 : Summary of studies that examined the relationship between lesions with dissections, NHLBI types and their outcomes. Acute complications included abrupt closure, Q wave

myocardial infarction, emergency and elective coronary artery bypass. Late events included revascularisation. NHLBI- National Heart, Lung and Blood institute; TLR – target lesion revascularisation; NR = not reported.

In 2001, Albertal et al.[49] showed moderate dissections (as classified in table 4) when left unstented had good outcome with a classification based on clinical relevance.

Table 4 Albertal et al. evaluation of dissections based on clinical parameters
"**mild**" dissections (type A*or B*),
"**moderate**" dissections (type C* without signs or symptoms of ischaemia),
"**severe**" dissections (type C* with symptoms or signs of ischaemia plus types D* to F*).
*Type A-F refers to NHBLI system of classification.

Various forms of classification of dissections were reported in studies throughout the years of POBA and early stent era, mainly based on the operators experience and preference. While some used mild, moderate and severe dissections, few preferred intimal and coronary arterial dissections[41]. Given the limitations with NHBLI angiographic classification, newer classification systems using intravascular ultrasound (IVUS) or angioscopy modalities were proposed to correlate plaque characteristics with dissection risk.

3.2 IVUS patterns of dissections

During the stent era, IVUS was increasingly utilised to show real-time cross-sectional observation of the vessel response to balloon angioplasty and stenting. IVUS gave in-depth assessment of the lesion morphology and the results of balloon angioplasty with IVUS imaging were consistent with Waller's histopathological studies[5]. This helped in deciding the interventional strategies in the event of suboptimal dilatation according to the plaque

composition, calcification and eccentricity. Honye et al.[5] and Gerber et al.[50] simultaneously published their IVUS experiences with dissections suggesting a similar system of classification in 1992. Honye's method of classification failed to have good correlation with angiography detected dissections as 10 of 23 angiographic dissections in his study were not seen on ultrasound. On the other hand, Gerber's patterns of dissections are very detailed, but is complicated and time consuming for an operator with basic IVUS interpretation skills. The application of this technology declined in the field of dissections due to a number of reasons. Firstly, IVUS failed to detect dissection flaps that adhered to the wall when it transversed past them [51] . Secondly, it poorly differentiated the echo free space of thin diseased media from dissection planes. Finally, IVUS detected severity of dissections did not correlate with any pre-interventional lesion characteristics[51]. Figure 4 illustrates IVUS appearances of coronary dissections during PCI.

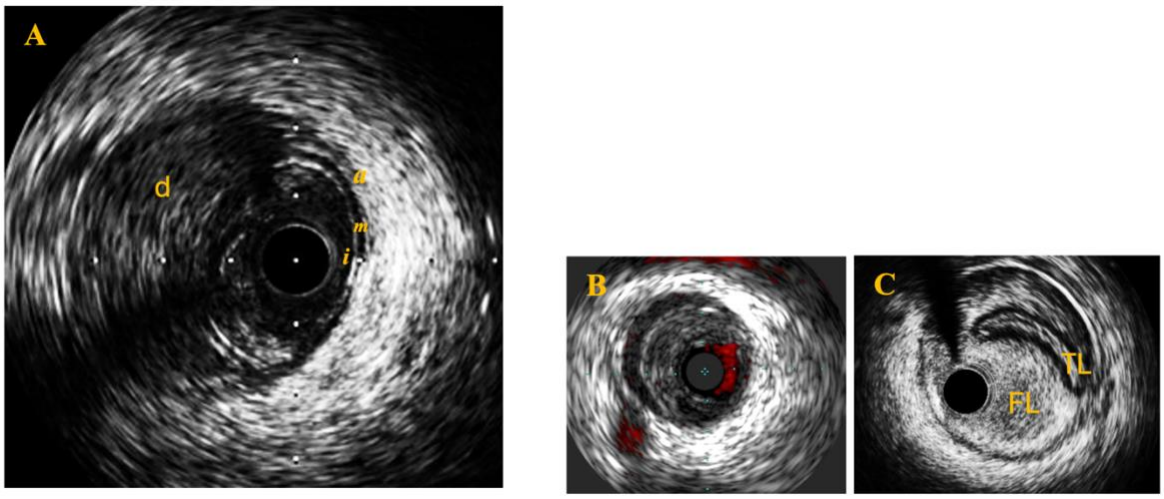


Figure 4: A-C represents IVUS images of coronary dissections during PCI. A- echogenic intramural hematoma (d) seen in the dissection plane. B- Chromoflo IVUS image showing an echo free space representing a false lumen. C- A large false lumen compromising the true lumen during PCI as a result of guide induced dissection and IVUS confirms that the wire is in false lumen; *i – intima; m- media; a- adventitia; d- dissection; TL -true lumen; FL – False lumen.*

3.3 IVUS validation of therapeutic dissection concept

However, IVUS technology continued to improve with high resolution and low-profile catheters. In 2000, Schroeder et al.[52] demonstrated IVUS detected therapeutic dissections (mild and moderate group) did not impact acute or long-term outcome further substantiating the concept of therapeutic dissections. Schroeder's classification method was rather simple and easy to use as follows:

Table 5: Schroeder's IVUS dissection criteria
(a) mild dissection with the presence of a partial tear
(b) medium dissection with a tear through the plaque (50% plaque diameter)
(c) severe dissections with a second channel extending into the media with a clearly identifiable second lumen after contrast dye.

Following this study in 2001, Shigeyama et al. attempted to classify the therapeutic dissections category based on angiography into types A-E in relation to the depth and breadth of dissection and the presence of intimal flap or spiral appearance[53]. But its clinical application is limited as the interest was more in the management of indeterminant group of dissections.

OCT – a better lens for DCB related dissections?

Since 1991, the use of optical coherence tomography has expanded rapidly and is now a preferred modality for precisely imaging coronary luminal architecture, differentiating plaque rupture or erosion, vulnerable plaque identification and dissections[54]. OCT has been considered safe for imaging spontaneous coronary artery dissections (SCAD)[55–58] although clinical risk is reported[59–61]. The superior spatial resolution of OCT identifies intramural hematoma, endothelial tears, or entry sites of dissection[54,62]. Given the low clinical risk, OCT

continues to be indicated in cases of dissection with diagnostic uncertainty[56,58,59]. An OCT-guided DCB strategy is an area of interest in recent DCB studies[63,64], with reconstruction software allowing accurate quantification of dissection depth and volume as seen in figure 5. A recent study called TRANSFORM has shown OCT derived absolute dissection volume has a favourable effect on lumen gain post DCB[65] in Paclitaxel DCB arm compared to Sirolimus arm in small de novo coronary vessels. Furthermore, OCT fused with angiography provides realistic reconstruction of lumen architecture with vessel wall dissections guiding operators to formulate a specialized treatment for the patient subsets with DCB related dissections[66]. This could be the future of DCB technology and further research in this field is underway.

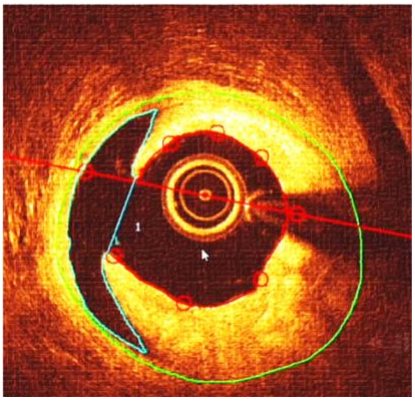


Figure 5: Measurement of dissection area by OCT (QCU-CMS)[65]: a straight line connecting the edges of the flap of dissection isolates the dissection space from the lumen. The volume of the dissection is calculated by Simpson's rule over the entire length of the dissection.

4. Coronary dissections and risk factors

The angiographic predictors of risk of dissection and its sequalae play a vital role in devising management strategies. Here we review the studies identifying clinical and angiographic risk factors and table 6 illustrates such associations.

In NHLBI cohorts, intimal dissections were associated with female gender, RCA lesions, multivessel disease, eccentric and diffuse disease[17]. Complicated angiographic characteristics such as irregular borders, intraluminal lucency, location of stenosis at a bifurcation or a curve were identified as predictors of dissections by Ischinger et al.[11] in 1986. In the same year, Bredlau et al. showed the strongest predictor of a major ischemic complication was procedural appearance of an intimal dissection with 6.5 fold increase in risk of MI, emergency CABG and death [67]. Later, in a hemodynamic study in 1987, Redd et al. first graded the degree of disruption angiographically into intimal (when the luminal or extraluminal contrast staining within the confines of original PTCA lesion) and arterial dissections (when extending beyond the lesion either proximally or distally). The authors studied the relationship between the dynamic behaviour of the transtenotic pressure gradient after each balloon inflations and presence of disruptions with subsequent vessel closure. Patients with a rising trend in transtenotic pressure gradient had higher incidence of arterial dissections (25% vs 7%, OR 4.8, p=0.001)[68] but not the isolated intimal tears alone. On a multivariate analysis of procedural variables, rising transtenotic gradient trend (OR 1.99, p=0.002), lesion length (OR 1.11, p=0.007) and post-PTCA gradient (OR 1.06, p=0.001) were strong predictors of arterial dissections and rising trend in transtenotic pressure was significantly associated with other ischemic complications such as acute closure (OR 2.04, p <0.001) , CABG (OR 1.13, p <0.001), and MI (OR 2.91, p <0.001). Further in 1989 when Black et al.[23] analysed the morphological variables, dissection length , diameter stenosis(DS) of ≥ 25% following dilatation and video-densitometry assessment (VDA) of luminal cross-sectional area(CSA) of <2mm² were found to be the strong correlates of arterial dissection with ischemic complications. Extraluminal contrast cap had a slightly weak correlation compared to the rest of the variables.

Table 6 Factors associated with coronary dissections
Clinical
Age≥62 years[39]
Female gender[13,32]
Acute coronary syndromes[41,69,70]
Low cholesterol<5.7mmol/l[39]**,**
Angiographic
RCA lesion[11,13,17,39]
Multivessel disease[69–71]
Localization at a bifurcation or a curve[11,39]
Length of the lesion[70–72]
Diffuse disease[70]
Eccentric stenosis[11,39,70,72]
Irregular borders[11,41]
Intraluminal lucency* [11,39]**,**
Procedural
Larger balloon assignment(>1.3:1)[70,71]
Higher inflation pressure[39]
Multiple lesion dilatation[70,71]
Multisite dilatation[70,71]
Dilatation at a tortuosity[11,39]

***Intraluminal lucency is a correlate of plaque rupture, ulceration, subintimal haemorrhage, or superimposed or recanalized thrombus; RCA- right coronary artery.**

There was mounting evidence that intimal dissections were therapeutic. PTCA operators became more aware of the safety of uncomplicated intimal dissections and as their experience expanded, the interest in salvaging the indeterminant and complicated dissection group led to a strategy of lesion-specific device therapy to avoid abrupt vessel closure and emergency surgery.

5. Management of coronary dissections – Early and late POBA era

In the early era of POBA, any dissections causing acute coronary occlusion were treated surgically. Despite prompt surgical revascularisation, more than 50% of the patients developed significant MI due to the unavoidable delay of sternotomy (and, of note, vein grafts as opposed to LIMA were used for expediency)[73]. Subsequently in an effort to reverse abrupt vessel closure non-surgically , attempts were made to reopen the occluded vessel by relieving spasm and thrombus pharmacologically with intracoronary vasodilators, thrombolytics and heparin infusions[74,75]. When these measures were exhausted and MI was imminent, intra-aortic balloon pumps were used to limit myocardial injury before vein grafting[75]. Reperfusion catheters were utilised in 1986 to allow optimal bypass grafting[76,77]. However, in the absence of chest pain and ECG changes acutely, even large flow limiting dissections were treated by semi-elective bypass surgery. A variety of PCI management options were also subsequently devised to deal with unsafe dissections.

5.1 Repeat PTCA redilatation technique

Immediate repeat dilatation and successful reopening of the occluded dissection in seven patients was first reported in 1984 by Marquis et al [78]. This became a routine approach in treating dissections complicating abrupt reclosures during or after PTCA in following years. About 50% of patients had successful restoration of antegrade flow, thereby avoiding extensive myocardial damage and emergency surgery[11,79,80].

5.2 Tack-back technique

Further technical improvisation was made by using a standard balloon of same or slightly larger diameter and performing low-pressure inflations at increments of 1 or 2 atmospheres for 60-180 seconds repeatedly to gain patency[48,69,80,81]. In theory, this remodelled the lesion by 'tacking up' the dissected flap and stabilized dissections with high success rates[82]. Successful tack-back phenomenon restores a patent lumen possibly by allowing the tissue flaps to adhere to damaged vessel wall. In an analysis of 109 patients, Lincoff et al. demonstrated that prolonged balloon inflation was found to be an independent correlate of successful resolution of vessel closures (OR 5.11; p=0.001) on multivariate analysis[82].

5.3 Prolonged balloon inflation using auto-perfusion catheters

When repeat balloon angioplasty failed, prolonged balloon inflations were undertaken with the aid of an auto-perfusion catheters[83]. A specialised large profile Stack hemoperfusion catheter was first used in 1988 and maintained distal vessel perfusion through the proximal and distal catheter holes simultaneously facilitating prolonged inflations[84]. The inflation durations were 3 to 30 minutes depending on the tolerance of the patient. It proved very effective in improving outcomes in PTCA refractory dissections[38–42] but its use was limited due to passive inadequate perfusion, unfavourable coronary anatomy (side branch occlusion, small vessel, tortuosity) , poor guidewire access to the distal vessel, difficult delivery and the advent of better techniques[85]. The Ringer perfusion balloon catheter (Ringer PTCA) is a rapid-exchange 0.014" compatible balloon catheter that conforms into a helical cylinder upon inflation and maintains

distal perfusion flow as shown in figure 5. A prospective, multicentre, single-arm clinical study of 60 patients demonstrated that the balloon was well-tolerated in the majority of patients susceptible to procedural ischemia when inflated for 60 seconds or more[90]. The Food and Drug Administration (FDA) has recently approved its use in the United States, and it is mainly indicated in PTCA and bypass grafts[91]. This technology could potentially be utilized in scenarios involving indeterminate dissections that necessitate modification.

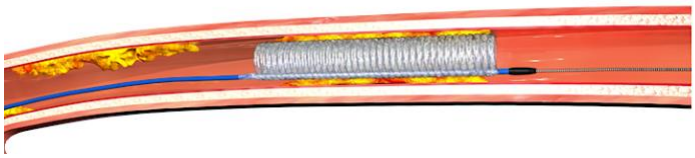


Figure 6 : Ringer PTCA balloon catheter that forms a helical cylinder on inflation and maintains distal perfusion through a large central perfusion lumen[92].

5.4 Controlled inflation technique

Progressive coronary dilation, that is, predilation of the stenosis with a smaller balloon and then maximal dilation with an optimally sized balloon produces less uncontrolled injury and thus reduce the incidence of major complications. This was demonstrated by Banka et al. in a study consisting of 1486 vessels. The success rate with this technique was 98.7% in 1248 partially occluded vessels and 88% in 353 totally occluded vessel. This technique markedly lower incidence of acute closure, major dissection, emergency coronary bypass, and death in dilation of both simple and complex lesions[93].

5.5 Directional coronary atherectomy (DCA) and balloon pyroplasty

Resection of occlusive dissection flaps causing luminal compromise by Atherocath devices (DVI, Devices for Vascular Intervention, California) were reported in few cases with success rates of around 80% during early1990s[94–97]. DCA did not gain much popularity given the greater risks of vessel perforation, inconsistent results and technical difficulties. Sealing of dissection flaps by imparting various forms of thermal energies such as laser[98,99], radio-

frequency[100], microwave[101,102] had been used in the past around 1990s but remained academic owing to restenosis risks and cost.

5.6 Bailout stenting

In 1987, intracoronary stainless-steel stents were described to address abrupt closure and in later years , subsequently reduce restenosis [2] . Sigwart et al. demonstrated the first emergency implantation of the endoluminal Wallstent (Schneider,Inc.) for acute occlusion caused by dissection in 13 patients in 1988[103]. Stents were effective in achieving better angiographic appearances of intimal dissections by securing the flaps and increasing residual lumen diameter[104], Gianturco-Rubin Palmaz-Schatz stents and other varieties of BMS became very popular in handling bailout situations and reducing the incidence of Q-wave MI and emergency CABG[82,105,106]. However, a multitude of thrombotic, bleeding and restenosis risks then ensued with acute and subacute stent thrombosis emerging as a problem[107–109]. When compared with auto-perfusion BA for acute closure in a non-randomised trial, the stent group had a higher subacute reclosure rate and more deaths[110,111]. Emergency CABG was still required when large dissections could not be repaired, or the bailout methods failed or perforation occurred and the conduit choice in emergency settings slowly shifted to left internal artery grafts from saphenous veins[73,112]

Subsequently, stent technology underwent many technical advancements from using heparin coated thick bare mounted rigid coils[113] to the ultrathin drug eluting stents used currently, yet there is persistent risks of restenosis, thrombosis and stent failures[114].

6. DCB era

The concept of drug coated balloon (DCB) angioplasty is ‘device mediated drug delivery’ to a target lesion by using a conventional semi-compliant balloon coated with an antiproliferative

drug[4]. Prior to DCB delivery, the target lesion must be adequately prepared to achieve an acceptable acute lumen gain and identify lesions prone to acute vessel closure and dissections[115]. In the event of flow limiting vessel threatening dissections and >30% residual stenosis after extensive and optimal lesion preparation, bail out stenting (BOS) is recommended[8]. The rates of BOS across major DCB studies[7,8,116–120]are around 5-22% and high grade coronary dissection remains the predominant indication for BOS besides acute vessel recoil. Whilst the DCB expert consensus document recommends BOS for any dissections equivalent or greater than type C NHLBI (National Heart, Lung, and Blood Institute) angiographic classification[8], there are studies that have shown non flow limiting moderate dissections including type C are safe when left alone[49,121].

Universally, severe dissections (Type D, F NHLBI) are treated as a complication requiring stent deployment to prevent periprocedural myocardial infarction (MI). The management of mild to moderate types of dissections (Type A-C) generally varies among interventionalists based on their experience in DCB angioplasty. In the past two decades of drug eluting stent (DES) era, the vast majority of lesions undergoing percutaneous coronary intervention (PCI) are stented. However, the threshold to consider bailout stenting in cases of dissections will become higher with increasing experience with DCB angioplasty when a refined lesion preparation algorithm is applied.

7. Conclusion

Coronary dissections are a stumbling block to widespread adoption of "DCB only" angioplasty and this limitation can be overcome with the lessons from POBA era and a change in outlook towards conservative management of coronary dissections dictated by the clinical situation and patient safety. The re-learning of the avoidance, recognition and management of coronary

dissections will facilitate an increased uptake in this promising new PCI concept of “leave nothing behind” .

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