Coronary and Myocardial Angiography

Angiographic Assessment of Both Epicardial and Myocardial Perfusion

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Angiographic assessment of epicardial coronary artery blood flow has played a pivotal role in our understanding of the “time-dependent open artery hypothesis” and in the evaluation of reperfusion strategies over the past 2 decades. It has become increasingly apparent, however, that clinical outcomes are not only associated with angiographic flow in the epicardial artery, but also with angiographic flow in the myocardium. To this end, the goal of reperfusion therapies has shifted to include reperfusion downstream at the level of capillary bed, and it might be more appropriate that the hypothesis now be termed “the time dependent open artery and open microvascular hypothesis.” The goal of this article is to review angiographic methods used to evaluate myocardial ischemia and infarction and to discuss the insights into the pathophysiology of acute coronary syndromes provided by these angiographic indexes of coronary artery blood flow and myocardial perfusion.

**TIMI Flow Grades (TFGs)**

For nearly 2 decades now, the Thrombolysis In Myocardial Infarction (TIMI) flow grade classification scheme has been successfully used to assess coronary blood flow in acute coronary syndromes (Table). It has been a valuable tool to compare angiographic outcomes following reperfusion, and the association of the TFGs with clinical outcomes (including mortality) has been well documented. The relationship between TFG and mortality does satisfy what some consider to be 3 criteria required to validate a surrogate end point for mortality, as follows: (1) There is an association between TIMI grade 3 flow and mortality, (2) an agent such as recombinant tissue plasminogen activator improves TIMI grade 3 flow by 22% over another agent such as streptokinase, and (3) the agent tissue plasminogen activator improves mortality 1.1% over streptokinase.

On the basis of this relationship between TIMI flow and mortality observed in the GUSTO I (Global Utilization of Streptokinase and Tissue Plasminogen activator [alteplase] for Occluded coronary arteries) angiographic trial, it was anticipated that an additional 20% improvement in TIMI 3 flow would be required to further improve mortality by another 1%. The disparity between the early results of trials evaluating combination therapy [low-dose fibrinolysis combined with full-dose glycoprotein IIb IIIa (GP IIbIIIa) inhibition] and the lack of mortality benefit in subsequent large-scale clinical trials has raised questions regarding the relationship between improvements in TIMI grade 3 flow and clinical outcomes. Despite the initial optimism of early dose-escalation trials involving low-dose fibrinolytic combined with full-dose GP IIbIIIa inhibition, later results in blinded parallel dose-confirmation phases yielded a modest improvement in TIMI grade 3 flow at 60 minutes and an even more modest 4% improvement at 90 minutes among 948 patients studied (Figure 1). On the basis of approximately a 6% improvement in early TIMI grade 3 flow, a 0.3% improvement in mortality in a large-scale mortality trial might be expected (6% observed TFG 3 improvement/20% required TFG 3 improvement for 1% mortality reduction =0.3%). The results of GUSTO V are in keeping with this modest estimated reduction with mortality rates of 5.9% and 5.6% (n=16,588, P=NS).

Care must be taken when extrapolating the results of angiographic patency studies to estimate potential clinical benefits. For example, the association of TIMI grade 3 flow with mortality is confounded by the fact that the majority of TIMI grade 2 flow is observed in the left anterior descending artery (LAD) territory, whereas the majority of TIMI grade 3 flow is observed in the right coronary artery (RCA). Thus, improved outcomes among patients with TIMI grade 3 flow are explained at least in part by the fact that inferior myocardial infarction (MI) location is associated with a lower mortality rate. Use of a more precise angiographic measure such as the TIMI frame count does support the notion that improved epicardial flow is associated with improved clinical outcomes; however, the magnitude of the clinical improvement associated with TIMI grade 3 flow may have been overestimated and may be nonlinear. Greater clinical benefits may be observed if a closed artery (TIMI grade 0/1 flow) is opened (TIMI grade 2 flow) compared with the magnitude of improvement that might occur if an artery with TIMI grade 2 flow is converted to TIMI grade 3 flow. As more arteries with TIMI grade 2 flow are treated with adjunctive percutaneous coronary intervention (PCI), the prognosis associated with this flow grade may improve. Indeed, 2-year mortality in more recent analyses indicates that the survival advantage of TIMI grade 3 flow over TIMI grade 2 flow at 2 years may not be as great as it once was in the era before aggressive utilization of rescue and adjunctive (PCI).
Indeed, one of the benefits of rescue and adjunctive PCI following fibrinolytic administration may be to reduce reocclusion. The benefit of achieving early patency is greatly reduced if it is not sustained as a result of reocclusion. Reinfarction is associated with a doubling in mortality, which is due to an early divergence in mortality by 30 days.\textsuperscript{15,16} Although early randomized trials failed to demonstrate a benefit in the performance of conventional angioplasty soon after fibrinolytic administration, these trials preceded the use of stents, thienopyridines, platelet GP IIb/IIIa inhibitors, and the monitoring of activated clotting times. Among 20,101 patients enrolled in recent TIMI trials, performance of PCI during the index hospitalization was associated with a lower rate of in-hospital recurrent MI (1.6\% versus 4.5\%, \(P<0.001\)) and lower 2-year mortality (5.6\% versus 11.6\%, \(P<0.001\)).\textsuperscript{16} The rates of PCI were low (\(<10\%) in GUSTO V, and this may account for the modest benefits observed among patients treated with combination therapy. Somewhat paradoxically,
although one of the benefits of combination therapy was touted to be a reduction in “urgent revascularization,” if such revascularization is actually beneficial by virtue of stabilizing the lesion and reducing the risk of reocclusion, then clinical outcomes may in fact be worse if a more successful pharmacological strategy lowers the rate of urgent revascularization.17 It should also be noted that while these angiographic measures are indexes of flow, and although they are associated with the risk of reocclusion,18 other nonangiographic processes may also underlie the pathophysiology of reocclusion as well as other clinical outcomes.

Although poorer TFGs and poorer clinical outcomes are clearly associated, the directionality of any causal relationship between the two has not been unequivocally demonstrated. For instance, it is not clear whether slower blood flow causes larger MIs or alternatively whether larger MIs cause slower blood flow as a result of greater edema or microvascular disruption in the myocardium. Furthermore, cessation of coronary blood flow in acute MI does not explain all deaths, as there are other pathophysiological mechanisms by which patients may die in acute MI, such as intracranial hemorrhage. Finally, as discussed below, successful restoration of epicardial patency in the absence of successful myocardial perfusion may not confer large clinical benefits.

The Corrected TIMI Frame Count: A Measure of the Time for Dye to Go Down the Artery

Although the TFG classification scheme has been a valuable tool for comparing the efficacy of reperfusion strategies and in the identification of patients at higher risk for adverse outcomes in acute coronary syndromes, there are limitations to this classification scheme.7 To overcome these limitations, one of us (C.M.G.) developed a new, more objective and precise index of coronary blood flow called the corrected TIMI frame count (CTFC), in which the number of cine-

frames required for dye to reach standardized distal landmarks are counted; this is essentially a measure of the time for dye to go down the artery.7,8,10

In the first frame used for TIMI frame counting, a column of dye touches both borders of the coronary artery and moves forward (Figure 2).7 In the last frame, dye begins to enter (but does not necessarily fill) a standard distal landmark in the artery (Figure 2). These standard distal landmarks are as follows: in the RCA, the first branch of the posterolateral artery in the RCA (third column, bottom panel); in the circumflex system, the most distal branch of the obtuse marginal branch that includes the culprit lesion in the dye path (third column, middle panel); and in the LAD the distal bifurcation, which is also known as the moustache, pitchfork, or whale’s tail (third column, bottom panel). Adapted from Gibson et al.7

In contrast to the conventional TFG classification scheme, the CTFC is quantitative rather than qualitative, it is objective rather than subjective, it is a continuous rather than a categorical variable, and it is reproducible.7 Using the CTFC, coronary blood flow is unimodally distributed as a continuous variable.7 Thus, any division of flow into normal and abnormal categories is somewhat arbitrary. It should be noted that if an epicardial artery is occluded, then a frame count of 100 is imputed. This value of 100 lies near the 99th percentile for frame counts among open arteries in the ST-elevation MI (STEMI) setting. If values are imputed for closed arteries, then nonparametric analyses (eg, Wilcoxon rank sum tests) should be utilized because the dataset often follows a nonparametric distribution.

In multiple studies, the CTFC has been shown to be quite reproducible with a 1- to 2-frame difference between observ-
ers,20–32 Similarly, 2 experienced angiographic core laboratories (GUSTO and TIMI) have analyzed the same films from a fibrinolytic trial with discrepancies in 21% of TFG readings (41/194, Kappa=0.76); however, excellent concordance in trial results were seen using the CTFC (overall median difference=0 frames).32 The CTFC is also accurate in that it is highly correlated with Doppler velocity wire measure of coronary flow reserve, distal velocity, average peak velocity, and volumetric flow,21–23 as well as fractional flow reserve ($r=0.85$).24

**Technical Factors Influencing the CTFC**

Normally 21 frames are required for dye to traverse the human coronary artery.7 Despite differences in the length of the coronary arteries, the force of injections, the diameter of the arteries, heart rates, cardiac output, and catheter engagement, there is only a 3.1-frame standard deviation among patients with normal flow, and the 95% confidence interval for normal flow extends from >14 frames to <28 frames.7 Faster than normal or hyperemic flow is therefore defined as a CTFC <14 frames and constitutes what we now term “TIMI grade 4 flow.”77 Although the CTFC is not used to determine the TFGs, in a retrospective analysis, the TIMI Angiographic Core Laboratory tended to classify flow as TIMI grade 2 flow if the CTFC was >40 (≈1.3 seconds).7

A variety of technical and physiological variables impact the CTFC.20,33–36 Use of a power injector to change the force of injection (cc/sec) from the 10th to the 90th percentile of human injection rates lowers the CTFC by only 2 frames,33 nitrate administration significantly increases the CTFC by ≈6 frames (P<0.001),20 dye injection at the beginning of diastole significantly decreases the CTFC by 3 to 6 frames,20 and increasing the heart rate by 20 bpm significantly decreases the CTFC by ≈5 frames (P<0.001).20 It is for this reason that administration of nitrates is often standardized in trials assessing the CTFC as an end point. The mechanical force of injection alone in a closed artery following fibrinolytic therapy will open ≈10% of closed arteries,34 but the dye type used for injection is not associated with changes in the CTFC.35

**Relation of the CTFC to Clinical Outcomes**

The CTFC following fibrinolytic administration is related to a variety of clinical outcomes.7,8,10,25–30 Flow in the infarct-related artery in survivors is significantly faster than in patients who die (49.5 versus 69.6 frames; P=0.0003).8 Mortality increases by 0.7% for every 10-frame rise in CTFC (P<0.001).8 None of the patients in the TIMI studies who have had a CTFC <14 (hyperemic or TIMI grade 4 flow) died by 30 days.8 Likewise, in patients with unstable angina (UA) or non–ST-elevation MI (NSTEMI), the post-PCI culprit flow among survivors is significantly faster than among those patients who died (CTFCs 20.4 versus 33.4 frames, $P=0.017$).37 Again, none of the 376 patients with a CTFC <14 following PCI died, underscoring the fact that, even within the subgroup of patients with “normal flow,” there may be further subgroups with even better flow and even better mortality.37 Multiple studies have now documented an association between the CTFC and clinical outcomes among patients treated with primary PCI also, and the CTFC has demonstrated greater sensitivity in detecting improvements in epicardial flow compared with the use of TIMI grade 3 flow among patients treated with new device interventions.38–42

With respect to other end points, the CTFC has also been related to a lower rate of restenosis, even when postprocedure diameters were corrected for.37 Thus, not only is “bigger better,” but “faster is better” also.37 Stankovic et al22 have built on these observations further by dividing the CTFC by the minimum lumen diameter to demonstrate that this measure, which integrates both anatomic and functional flow data, is the strongest predictor of restenosis in their study, and we have demonstrated that the ratio is associated with post-PCI death or MI.37 Slower CTFCs on surveillance angiography are also associated with higher rates of transplant rejection.43

**Insights Into the Pathophysiology of STEMI and UA/NSTEMI Based on the CTFC**

Until recently, it was assumed that flow in nonculprit arteries in the setting of acute coronary syndromes was “normal.” However, the CTFC in uninvolved arteries in acute STEMI (30.5 frames) is in fact 40% slower than normal (21 frames, $P<0.001$).44–46 In the setting of STEMI, adjunctive and rescue PCI restores flow in culprit vessels that is nearly identical to that of nonculprit arteries in the setting of acute MI (30.5 versus 30.5 frames, p=NS),46 but this flow remains slower than normal. PCI of the culprit lesion is also associated with improvements in the nonculprit artery after the intervention in both the STEMI and UA/NSTEMI settings.44,45 If abnormal flow was present in the nonculprit artery at baseline (ie, CTFC >28), then the improvements in nonculprit flow were more dramatic (10 frames).44 It is notable that slower flow throughout all 3 arteries in STEMI is associated with a higher risk of adverse outcomes,44 poorer wall motion in remote territories,44 poorer tissue perfusion on digital subtraction angiography (DSA),45 and a greater magnitude of ST depression in remote territories such as the anterior precordium in inferior MI.47 It could be speculated that the poorer flow in nonculprit arteries may be the result of more extensive necrosis in shared microvasculature, or a result of vasoconstriction mediated through either a local neurohumorl or paracrine mechanism. Indeed, Gregorini et al48 have demonstrated over the past 2 decades that a widespread range of vasoconstrictors including thromboxane A2, serotonin, endothelin, oxygen-derived free radicals, and thrombin are all released in the setting of vessel injury and thrombosis.

It has long been assumed that the residual stenosis in the setting of STEMI is largely responsible for the delay in flow. However, despite a 13% residual diameter stenosis and the relief of intraluminal obstruction that would be anticipated after stent placement, flow remains persistently delayed to 26 frames poststenot, and likewise 34% of stented vessels remain
with abnormal flow with a CTFC ≥28 (the 95th percentile of the upper limit of normal). This persistent delay is unlikely to be due to either the residual stenosis or intraluminal obstruction and points to the fact that there are other determinants of delayed epicardial blood flow. Multiple determinants of coronary blood flow at 90 minutes after fibrinolysis have been identified, and a map of their multiple interrelationships or colinearities observed in an expanded dataset is shown in Figure 3. All variables on the chart are related to impaired epicardial blood flow following fibrinolytic administration, and overlap of the Venn diagram among variables indicates which variables are related to each other. While the presence of pulsatile flow (systolic flow reversal), and a greater percentage of the vessel to the stenosis is associated with slower flow in the epicardial artery, these 2 variables are likewise associated with LAD infarct artery location. Likewise, poorer tissue perfusion (TMPG), tighter percent diameter stenoses, and a shorter duration of patency by 90 minutes (delayed opening in response to a fibrinolytic agent) are all associated with a longer duration of symptoms before arrival to the hospital.

Obviously, the residual percent stenosis plays a critical role, with the average 70% stenosis increasing the CTFC by ~17 frames. The presence of residual thrombus adds ~4 frames. There are also unanticipated contributors to delayed flow such as the timing of reperfusion; ie, those patients who were patent at 60 minutes had 15 frames faster flow than those patients who achieved flow between 60 and 90 minutes. LAD infarcts had slower flow by 8 frames than infarcts in other locations. It could be speculated that left system infarcts have slower flow as a result of the fact that they have lesions that are located more proximally, they subdent the thicker left ventricular wall, and there is higher wall stress in the left ventricle than in the right ventricle.

Myocardial Angiography: The TIMI Myocardial Perfusion Grade (TMPG)

Restoration of epicardial flow does not necessarily lead to restoration of tissue level or microvascular perfusion, as elegantly documented by Ito et al on myocardial contrast echocardiography. Perfusion of the myocardium can also be assessed using the angiogram. In the TMPG system, TMPG 0 represents minimal or no myocardial blush; in TMPG 1, dye stains the myocardium, and this stain persists on the next injection; in TMPG 2, dye enters the myocardium but washes out slowly so that dye is strongly persistent at the end of the injection; and in TMPG 3, there is normal entrance and exit of dye in the myocardium (Table). Another method of assessing myocardial perfusion on the angiogram is the myocardial blush grade (MBG) developed by van’t Hof et al. A grade of 0 (no blush) and a grade of 3 (normal blush) are the same in the TMPG and MBG systems. An MBG grade 1 or 2 represents diminished intensity in the myocardium and corresponds to a value of 0.5 in the expanded TMPG grading system. A TMPG of 1 or a stain in the TIMI system is subsumed within the value of a 0 in the MBG system. Lepper et al have demonstrated that angiographic and echocardiographic myocardial perfusion are closely related, and among patients undergoing primary PCI for acute MI, impaired MBG was the best multivariate predictor of nonreperfusion on myocardial contrast echocardiography.

Independent of flow in the epicardial artery and other covariates such as age, blood pressure, and pulse, the TMPG has been shown to be multivariate predictors of mortality in acute STEMI at 2 years. The TMPG permits risk stratification even within epicardial TIMI grade 3 flow. Despite achieving epicardial patency with normal TIMI grade 3 flow, those patients whose microvasculature fails to open (TMPG 0/1) have a 7-fold increase in mortality compared with those patients with both TIMI grade 3 flow in the epicardial artery. Achievement of both TIMI grade 3 flow in both the artery and the myocardium is associated with a mortality under 1% (Figure 4). Likewise, in the setting of primary PCI, both van’t Hof et al and Haager et al have demonstrated an association between impaired myocardial perfusion and early and late mortality. These improvements in early and late mortality may be mediated by improvements in myocardial salvage. As Dibra et al have...
demonstrated, restoration of TMPG 2/3 is associated with a higher salvage index (0.49±0.42 versus 0.34±0.49, \( P=0.01 \)) and a smaller final infarct size (15.4±15.5% versus 22.1±16.2% of the left ventricle, \( P=0.001 \)). Indeed, second only to stent placement, restoration of TMPG 2/3 was the next most powerful independent determinant of the myocardial salvage index, and was more closely associated with higher salvage indexes than the TFGs.\(^{60}\)

The association between a prolonged duration of symptoms before treatment in ST-elevation MI with poorer clinical and angiographic outcomes has led to the phrase “time is myocardium.” Indeed, the angiographic data do now provide mechanistic data to support this common notion. The association between increased time from symptom onset to treatment, worsened myocardial perfusion, and increased mortality rate has been demonstrated both in patients treated with fibrinolytic therapy\(^{61}\) and in those treated with primary angioplasty.\(^{62,63}\) Impaired myocardial perfusion on the angiogram has in turn been associated with greater left ventricular end-diastolic pressure\(^{64}\) and the presence of overt congestive heart failure on presentation.\(^{65}\) Among patients presenting with cardiogenic shock, a restoration of normal myocardial perfusion is associated with improved survival.\(^{66}\)

There are data associating abnormal myocardial perfusion on the angiogram with slower Doppler velocity measurements in the epicardial artery.\(^{67}\) Does abnormal myocardial perfusion slow epicardial flow, or, alternatively, does abnormal epicardial flow impair myocardial perfusion? Although there is likely a bidirectional nature to any causal relationship between the two, after restoration of full epicardial patency (eg, after the scaffolding of the lesion by intracoronary stent placement), it is likely that impaired myocardial perfusion may play a major role in reducing antegrade flow in the epicardial artery. A variety of drugs are available to treat abnormal myocardial perfusion, but aside from adenosine, their association with improved clinical outcomes remains largely untested.\(^{68}\)

**Association of Electrocardiographic Findings With Angiographic Findings in STEMI**

The ECG (ST resolution) and the angiogram provide insight into myocardial perfusion. It is notable that both the ST segment resolution and the TMPG provide independent prognostic information with respect to SPECT infarct size.\(^{11}\) Likewise, with respect to clinical outcomes, 2 additional studies have now documented the complementary prognostic information provided by the ECG (degree of ST resolution) and the angiographic blush, with failure to achieve ST resolution and a closed myocardium on angiography following primary PCI carrying a particularly poor prognosis.\(^{59,69}\) These data suggest a potential electromechanical dissociation between microvascular blood flow and myocardial function. Whereas the angiogram may reflect mechanical patency of the microvasculature and the integrity of the endothelium, the electrocardiogram may reflect the functional status of the supplied myocardium.\(^{11}\) Measures of both processes appear to be independent and complementary in their prognostic significance. Finally, restoration of normal (TMPG 3) myocardial perfusion is not only associated with complete ST resolution, but it is also associated with earlier ST resolution on continuous ST-segment monitoring.\(^{70}\)

Similar to what has been observed in the STEMI setting, in the setting of UA/NSTEMI, independent of epicardial blood flow, pre-PCI TMPG 0/1 flow is associated with troponin T and I elevations, and if TMPG 0/1 persists after PCI, the risk of death or MI at 6 months is increased.\(^{71}\) With respect to the other marker of myonecrosis, creatine kinase (CK)–MB, TMPG 0/1/2 perfusion following PCI in the setting of UA/NSTEMI is associated with a nearly 10-fold rise in the risk of CK-MB elevations (41% versus 4%, \( P=0.002 \)), as well as a higher risk of adverse clinical outcomes at 1 year (32% versus 4%, \( P=0.01 \)).\(^{72}\) Taken together, these findings suggest a pathophysiological link between impaired tissue level perfusion, the release of cardiac markers (both before and after PCI), and adverse clinical outcomes in a variety of settings.
To quantitatively characterize the kinetics of dye entering the myocardium using the angiogram, DSA has been utilized. DSA is performed at end diastole by aligning cineframe images before dye fills the myocardium with those at the peak of myocardial filling to subtract spine, ribs, diaphragm, and epicardial artery (Figure 5). A representative region of the myocardium is sampled that is free of overlap by epicardial arterial branches to determine the increase in the Gray-scale brightness of the myocardium when it first reached its peak intensity. The circumference of the myocardial blush is measured using a handheld planimeter. The number of frames required for the myocardium to first reach its peak brightness is converted into time (seconds) by dividing the frame count by 30. In this way, the rate of rise in brightness (Gy/sec) and the rate of growth of blush (cm/sec) can be calculated.

Integrating the Assessment of Epicardial and Myocardial Perfusion

There is the need for a simple, broadly applicable angiographic metric that takes into account indices of epicardial and myocardial perfusion both before and after PCI to arrive at a single perfusion grade. The Angiographic Perfusion Score (APS) is the sum of the TFG (0 to 3) added to the TMPG (0 to 3) before and after PCI (total possible grade of 0 to 12). Failed perfusion can be defined as an APS of 0 to 3; partial perfusion, 4 to 9; and full perfusion, 10 to 12. Among STEMI patients, the APS is associated with larger SPECT infarct sizes and with the incidence of death or MI, as follows: failed, 16.7%; partial, 2.5%; and full, 2.4% (P=0.039). No patient with an APS score of “full” died, whereas mortality was 11.1% among patients with an APS score of “failed” (P=0.03). Such an APS, which combines grades of epicardial and tissue level perfusion before and after PCI or at the end of diagnostic cardiac catheterization to arrive at a single angiographic variable that is associated with infarct size and 30 day death or MI, may prove valuable in clinical risk stratification.

Other Angiographic Findings That Are Associated With Adverse Outcome

Lesion Location

Although epicardial and myocardial flow likely play a predominant role in mediating adverse clinical outcomes, other angiographic variables may provide valuable prognostic information. For instance, a greater proportion of the culprit artery distal to the stenosis is associated with higher mortality, poorer ST-segment resolution, and larger infarct sizes, particularly in the LAD distribution. The planimetered distance from the ostium to the LAD culprit lesion is associated with 30-day death or recurrent MI (odds ratio, 0.79 per centimeter increase in distance down the artery, P=0.01).

Pulsatile Flow

Reversal of systolic flow on Doppler velocity wire recordings has been associated with impaired tissue perfusion on myo-
cardiac contrast echocardiography in the STEMI setting. On the coronary angiogram, pulsatile flow (systolic flow reversal with cessation of antegrade contrast-dye motion or frank reversal of contrast-dye motion during systole) is likewise associated with higher CTFCs (slower epicardial flow), impaired TMPGs, less complete (≥70%) ST-segment resolution (23.5% versus 58.9%, P = 0.008), and a higher risk of death or reinfarction at 30 days (10.3% versus 5.0%, P = 0.019) independent of the velocity of antegrade flow in the epicardial artery.79 This simple and easily identifiable angiographic flow pattern may also be useful in clinical risk stratification.

Lesion Complexity
Worse angiographic lesion complexity has long been associated with reduced PCI success rates.80–84 In the setting of STEMI, greater lesion complexity is also associated with poorer epicardial and myocardial perfusion, both before and after PCI. In a multivariate model, Type C lesion complexity remains associated with increased 30-day mortality (odds ratio, 2.83; 95% CI, 1.44 to 5.57, P = 0.003) even after controlling for 60-minute TFGs, LAD infarct location, age, performance of PCI, and pulse and systolic blood pressure on admission location.85

Future Directions
The integration of newer angiographic parameters such as the TMPG into clinical practice has now begun. Other angiographic parameters, which at this time are predominantly research tools, will require software integration into the imaging chain to facilitate their real-time analysis. For example, just as DSA is performed in peripheral arterial interventions, it is now time that DSA be performed during coronary intervention to facilitate the assessment of myocardial perfusion. DSA can be performed offline if the cinerun is divided by the frame count (a measure of time) to estimate absolute velocity (cm/sec) in real time.

For decades, the technique we perform has traditionally been referred to as “coronary angiography.” It is now time to shift our eyes away from the obvious epicardial stenosis to assess perfusion of the microvasculature and perform the emerging technique of “myocardial angiography.” An emerging reality is in plain sight for all those who learn to see.

References


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