we are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists



122,000

135M



Our authors are among the

TOP 1%





WEB OF SCIENCE

Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us? Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected. For more information visit www.intechopen.com



Chapter 12

Coronary Angiography (IJECCE)

Chiu-Lung Wu and Chi-Wen Juan

Additional information is available at the end of the chapter http://dx.doi.org/10.5772/54080

1. Introduction

The ACC/AHA Task Force on Practice Guidelines herein revises and updates the original "Guidelines for Coronary Angiography," published in 1987 The frequent and still-growing use of coronary angiography, its relatively high costs, its inherent risks and the ongoing evolution of its indications have given this revision urgency and priority.

The expert committee appointed included private practitioners and academicians. Committee members were selected to represent both experts in coronary angiography and senior clinician consultants. Representatives from the family practice and internal medicine professions were also included on the committee [1].

1.1. Definitions

Coronary angiography is defined as the radiographic visualization of the coronary vessels after the injection of radiopaque contrast media. The radiographic images are permanently recorded for future review with either 35 mm cine film or digital recording. Percutaneous or cutdown techniques, usually from the femoral or brachial artery, are used for insertion of special intravascular catheters. Coronary angiography further requires selective cannulation of the ostium of the left and right coronary arteries and, if present, each saphenous vein graft or internal mammary artery graft to obtain optimal selective contrast injection and imaging. Numerous specialized catheters have been designed for this purpose. Physicians performing these procedures must be technically proficient in all aspects of the procedure and have a complete understanding of the clinical indications and risks of the procedure and of coronary anatomy, physiology and pathology. It is also important that these physicians understand the fundamentals of optimal radiographic imaging and radiation safety. Coronary angiography is usually performed as part of cardiac catheterization, which may also involve angiography of other vessels or cardiac chambers, and hemodynamic assess-



© 2013 Wu and Juan; licensee InTech. This is an open access article distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/3.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

ment as needed for a complete invasive diagnostic evaluation of the individual patient's cardiovascular condition[2,3].

1.2. Purpose

The purpose of coronary angiography is to define coronary anatomy and the degree of luminal obstruction of the coronary arteries. Information obtained from the procedure includes identification of the location, length, diameter, and contour of the coronary arteries; the presence and severity of coronary luminal obstruction(s); characterization of the nature of the obstruction (including the presence of atheroma, thrombus, dissection, spasm, or myocardial bridging), and an assessment of blood flow. In addition, the presence and extent of coronary collateral vessels can be assessed.

Coronary angiography remains the standard for assessment of anatomic coronary disease, because no other currently available test can accurately define the extent of coronary luminal obstruction. Because the technique can only provide information about abnormalities that narrow the lumen, it is limited in its ability to accurately define the etiology of the obstruction or detect the presence of nonobstructive atherosclerotic disease. A coronary angiography, which can help diagnose heart conditions, is the most common type of heart catheter procedure. [2,3]

2. Coronary angiography for specific conditions

2.1. General considerations

Coronary atherosclerosis is a slowly progressive process that can be clinically inapparent for long periods of time [78–80]. Coronary disease often becomes clinically evident because of the occurrence of symptoms, such as angina or those associated with MI. Patients with known CAD are those in whom the disease has been documented by either angiography or MI. "Suspected coronary disease" means that a patient's symptoms or other clinical characteristics suggest a high likelihood for significant CAD and its related adverse outcomes but that evidence of CAD has not yet been documented as defined above.

Patients may develop symptoms at one point in time but may become asymptomatic thereafter as the result of a change in the disease or as the result of therapy. For instance, many patients are symptomatic after an uncomplicated MI, as are patients with mild angina, who can be rendered asymptomatic by medications. The severity of clinical presentations and the degree of provocable ischemia on noninvasive testing are the principal factors used in determining the appropriateness of coronary angiography.

2.2. Stable angina

Patients with CAD may become symptomatic in many different ways but most commonly develop angina pectoris. In this document, angina pectoris (or simply angina) means a chest

discomfort due to myocardial ischemia, often described as a transient squeezing, pressurelike precordial discomfort. Angina is generally provoked by physical effort (particularly during the postprandial state), with exposure to cold environment or by emotional stress. The discomfort on effort is relieved by rest, its duration being a matter of minutes. The ease of provocation, frequency and duration of episodes may remain relatively unchanged in individuals for extended time periods, leading to the term "stable angina pectoris."

Recommendations for Coronary Angiography in Patients With Nonspecific Chest Pain Class I

High-risk findings on noninvasive testing. (Level of Evidence: B)

Class IIa: None.

Class IIb:

Patients with recurrent hospitalizations for chest pain who have abnormal (but not high-risk) or equivocal findings on noninvasive testing. (*Level of Evidence: B*)

Class III:

All other patients with nonspecific chest pain. (*Level of Evidence: C*)

2.3. Unstable angina

The acute coronary syndromes include unstable angina, non–Q-wave MI, and acute Q-wave MI. The diagnosis of unstable angina has been complicated by a broad range of presentations that can vary between atypical chest pain and acute MI. An expert panel of clinicians attempted to clarify the definition of unstable angina in the recently published "Clinical Practice Guideline for Unstable Angina"[129,130]. Three possible presentations are described:

- Symptoms of angina at rest (usually prolonged 20 minutes);
- New-onset (<2 months) exertional angina of at least CCS class III in severity;
- Recent (<2 months) acceleration of angina as reflected by an increase in severity of at least one CCS class to at least CCS class III.[4,5]

Variant angina, non–Q-wave MI and recurrent angina24 hours after MI are considered part of the spectrum of unstable angina. However, in this document, non–Q-wave MI is discussed in the section on acute MI. [4,5]

Recommendations for Coronary Angiography in Patients With Postrevascularization Ischemia

Class I

1. Suspected abrupt closure or subacute stent thrombosis after percutaneous revascularization. (*Level of Evidence: B*)

2. Recurrent angina or high-risk criteria on noninvasive evaluation (Table 5) within nine months of percutaneous revascularization. (*Level of Evidence: C*)

Class IIa

- **1.** Recurrent symptomatic ischemia within 12 months of CABG. (Level of Evidence: B)
- **2.** Noninvasive evidence of high-risk criteria occurring at any time postoperatively. (Level of Evidence:B)
- **3.** Recurrent angina inadequately controlled by medical means after revascularization. (Level of Evidence: C)

Class IIb

- **1.** Asymptomatic post-PTCA patient suspected of having restenosis within the first months after angioplasty because of an abnormal noninvasive test but without noninvasive high-risk criteria. (*Level of Evidence: B*)
- **2.** Recurrent angina without high-risk criteria on noninvasive testing occurring >1 year postoperatively. (*Level of Evidence: C*)
- **3.** Asymptomatic postbypass patient in whom a deterioration in serial noninvasive testing has been documented but who is not high risk on noninvasive testing. (*Level of Evidence: C*)

Class III

- **1.** Symptoms in a postbypass patient who is not a candidate for repeat revascularization. *(Level of Evidence: C)*
- **2.** Routine angiography in asymptomatic patients after PTCA or other surgery, unless as part of an approved research protocol. (*Level of Evidence: C*)

Coronary angiography during the initial management of patients in the emergency department

Patients Presenting With Suspected MI and ST- segment Elevation or Bundle-Branch Block Of all patients who ultimately are diagnosed with acute MI, those resenting with ST-segment elevation have been studied most extensively. Patients with ST-segment elevation have a high likelihood of thrombus occluding the infarct-related artery [6,7]. Considerable data exist showing that coronary reperfusion can be accomplished either by intravenous thrombolytic therapy or direct mechanical intervention within the infarct-related artery. Because the benefit obtained is directly linked to the time required to reestablish normal distal blood flow [8–10], rapid triage decisions are mandatory, and delays in instituting reperfusion therapy must be minimized. The "ACC/AHA Guidelines for the Management of Patients with Acute Myocardial Infarction" provide a comprehensive discussion of the indications, contraindications, advantages, and disadvantages of thrombolytic therapy and direct coronary angioplasty [11]. Although it is not the purpose of these guidelines to re-examine in detail the merits of these two reperfusion strategies, this is a rapidly evolving area, and some new information exists.

Recommendations for coronary angiography during the initial management of acute MI (MI suspected and ST-segment elevation or bundle-branch block present)

Coronary angiography coupled with the intent to perform primary PTCA

Class I

- **1.** As an alternative to thrombolytic therapy in patients who can undergo angioplasty of the infarct artery within 12 hours of the onset of symptoms or beyond 12 hours if ischemic symptoms persist.
- 2. In patients who are within 36 hours of an acute ST elevation/Q-wave or new LBBB MI who develop cardiogenic shock, are less than 75 years of age and revascularization can be performed within 18 hours of the onset of shock

Class IIa

1. As a reperfusion strategy in patients who are candidates for reperfusion but who have a contraindication to fibrinolytic therapy, if angioplasty can be performed as outlined above in class I. (Level of Evidence: C)

Class III

- **1.** In patients who are beyond 12 hours from onset of symptoms and who have no evidence of myocardial ischemia. (Level of Evidence: A)
- **2.** In patients who are eligible for thrombolytic therapy and are undergoing primary angioplasty by an unskilled operator in a laboratory that does not have surgical capability. (Level of Evidence: B)

Recommendations for early coronary angiography in the patient with suspected MI (ST-segment elevation or BBB present) who has not undergone primary PTCA

Class I: None.

Class IIa: Cardiogenic shock or persistent hemodynamic instability.(Level of Evidence: B)

Class IIb:

- **1.** Evolving large or anterior infarction after Thrombolytic treatment when it is believed that reperfusion has not occurred and rescue PTCA is planned. (Level of Evidence: B)
- 2. Marginal hemodynamic status but not actual cardiogenic shock.(Level of Evidence: C)

Class III

- **1.** In patients who have received thrombolytic therapy and have no symptoms of ischemia. (*Level of Evidence:A*)
- **2.** Routine use of angiography and subsequent PTCA within 24 hours of administration of thrombolytic agents. (*Level of Evidence: A*)

Recommendations for early coronary angiography in acute MI (MI suspected but no stsegment elevation)

Class I

- **1.** Persistent or recurrent (stuttering) episodes of symptomatic ischemia, spontaneous or induced, with or without associated ECG changes. (Level of Evidence:A)
- **2.** The presence of shock, severe pulmonary congestion, or continuing hypotension. (Level of Evidence: B)

Class II: None.

Class III: None.

Hospital-management phase of acute MI

The hospital-management phase of acute MI can encompass several clinical situations. Some patients with acute MI present too late in their course to be candidates for reperfusion therapy, and in others, the occurrence of infarction may not be appreciated at he time of presentation. These groups skip the acute-treatment phase of MI and enter the hospital-management phase directly. During the hospital management phase, the actions of the clinician are driven by the consequences of the infarction, such as congestive heart failure, hemodynamic instability, recurrent ischemia or arrhythmias. Although it is still convenient to divide patients into those with Q-wave and non–Q-wave infarctions, some indications for coronary angiography are common to all patients with MI regardless of how they have been treated initially and whether or not Q waves ultimately develop.

Recommendations for use of coronary angiography in patients with valvular heart disease Class I

- **1.** Before valve surgery or balloon valvotomy in an adult with chest discomfort, ischemia by noninvasive imaging, or both. (Level of Evidence: B)
- 2. Before valve surgery in an adult free of chest pain but with multiple risk factors for coronary disease. (Level of Evidence: C)
- 3. Infective endocarditis with evidence of coronary embolization. (Level of Evidence: C)

Class IIa

None.

Class IIb

During left-heart catheterization performed for hemodynamic evaluation before aortic or mitral valve surgery in patients without preexisting evidence of coronary disease, multiple CAD risk factors or advanced age. (Level of Evidence: C)

Class III

1. Before cardiac surgery for infective endocarditis when there are no risk factors for coronary disease and no evidence of coronary embolization. (Level of Evidence: C)

- **2.** In asymptomatic patients when cardiac surgery is not being considered. (Level of Evidence: C)
- **3.** Before cardiac surgery when preoperative hemodynamic assessment by catheterization is unnecessary, and there is neither preexisting evidence for coronary disease, nor risk factors for CAD. (Level of Evidence: C)

Congenital heart disease

Although there are no large trials to support its use, coronary angiography is performed in congenital heart disease for two broad categorical indications. The first indication is to assess the hemodynamic impact of congenital coronary lesions (375). The second is to assess the presence of coronary anomalies, which by themselves may be innocent but whose presence, if unrecognized, may lead to coronary injury during the correction of other congenital heart lesions. Congenital anomalies with hemodynamic significance include congenital coronary artery stenosis or atresia, coronary artery fistula [11], anomalous left coronary artery arising from the pulmonary artery [12], and anomalous left coronary artery arising from the right coronary artery or right sinus of Valsalva and passing between the aorta and right ventricular outflow tract [13]. Patients with congenital coronary stenosis may present with angina or unexplained sudden death in childhood, whereas patients whose left coronary passes between the pulmonary artery and aorta often have the same symptoms later in life. Patients with a coronary arteriovenous fistula often present with a continuous murmur or may have unexplained angina or congestive heart failure. Anomalous origin of the left coronary artery from the pulmonary artery should be suspected when there is unexplained MI or heart failure in early childhood. Other coronary anomalies of position or origin may cause no physiologic abnormality by themselves. Some, such as origin of the circumflex artery from the right sinus of Valsalva, are not associated with other congenital anomalies and present only as incidental findings and are significant only because they complicate the performance and interpretation of coronary angiograms.

Recommendations for use of coronary angiography in patients with congenital heart disease

Class I

- **1.** Before surgical correction of congenital heart disease when chest discomfort or noninvasive evidence is suggestive of associated CAD. (Level of Evidence: C)
- **2.** Before surgical correction of suspected congenital coronary anomalies such as congenital coronary artery stenosis, coronary arteriovenous fistula and anomalous origin of left coronary artery. (Level of Evidence: C)
- **3.** Forms of congenital heart disease frequently associated with coronary artery anomalies that may complicate surgical management. (Level of Evidence: C)
- **4.** Unexplained cardiac arrest in a young patient. (Level of Evidence: B)

Class IIa

Before corrective open heart surgery for congenital heart disease in an adult whose risk profile increases the likelihood of coexisting coronary disease. (Level of Evidence: C)

Class IIb

During left-heart catheterization for hemodynamic assessment of congenital heart disease in an adult in whom the risk of coronary disease is not high. (Level of Evidence: C)

Class III

In the routine evaluation of congenital heart disease in asymptomatic patients for whom heart surgery is not planned. (Level of Evidence: C)

Congestive heart failure

1. Systolic dysfunction

Although it was once believed that myocardial ischemia was either short-lived and resulted in little or no muscle dysfunction or resulted in infarction with permanent damage, it is now clear that a middle state may exist in which chronic ischemic nonfunctioning myocardium is present, to which function may return after myocardial revascularizations [15,16]. This intermediate state has been termed "myocardial hibernation." Although most cases of myocardial dysfunction resulting from CAD are probably irreversible when due to infarction and subsequent deleterious ventricular remodeling (ischemic cardiomyopathy) [17], some patients with hibernating myocardium have been shown to experience a doubling of resting ejection fraction with resolution of congestive heart failure after coronary revascularization [18,19]. However, in most cases of hibernation, a more modest improvement in ejection fraction of 5% occurs after revascularization [20].

2. Diastolic dysfunction

Isolated diastolic dysfunction is the cause of heart failure in 10% to 30% of affected patients. This disorder is common in older patients with hypertension and often is suspected because of echocardiographically detected concentric left ventricular hypertrophy, normal systolic function and abnormal transmitral flow velocity patterns [21]. However, in some patients with normal systolic function, the abrupt onset of pulmonary edema raises the suspicion that transient ischemia was the cause of decompensation, because elderly patients with hypertension have, by definition, at least two risk factors for coronary disease. In these patients, who are often too ill to undergo stress testing, coronary angiography may be necessary to establish or rule out the diagnosis of ischemically related diastolic dysfunction and heart failure.

Recommendations for use of coronary angiography in patients with congestive heart failure

Class I

1. Congestive heart failure due to systolic dysfunction with angina or with regional wall motion abnormalities and/or scintigraphic evidence of reversible myocardial ischemia when revascularization is being considered. (Level of Evidence: B)

- 2. Before cardiac transplantation. (Level of Evidence: C)
- **3.** Congestive heart failure secondary to postinfarction ventricular aneurysm or other mechanical complications of MI. (Level of Evidence: C)

Class IIa

- **1.** Systolic dysfunction with unexplained cause despite noninvasive testing. (Level of Evidence: C)
- **2.** Normal systolic function, but episodic heart failure raises suspicion of ischemically mediated left ventricular dysfunction. (Level of Evidence: C)

Class III

Congestive heart failure with previous coronary angiograms showing normal coronary arteries, with no new evidence to suggest ischemic heart disease. (Level of Evidence: C)

1. Aortic dissection

The need for coronary angiography before surgical treatment for aortic dissection remains controversial because there are no large trials to support its use. In young patients with dissection due to Marfan syndrome or in dissection in peripartum females, coronary angiography is unnecessary unless there is suspicion that the dissection has affected one or both coronary ostia. In older patients, in whom dissection is usually related to hypertension, coronary angiography is often necessary, especially if patients are suspected of having coronary disease because of a history of angina or objective evidence of myocardial ischemia. In patients who have no history of coronary disease, the indications for coronary angiography are much less certain. Because of the high incidence of coronary disease in older patients with dissection, some studies have advocated routine coronary angiography [22], whereas others have found increased mortality when angiography is performed [23].

2. Hypertrophic cardiomyopathy

Significant CAD due to atherosclerosis is found in 25% of patients aged >45 years with hypertrophic cardiomyopathy [26]. Because symptoms due to CAD and hypertrophic cardiomyopathy are similar, patients with ischemic symptoms not well controlled with medical therapy may require coronary angiography to resolve the cause of chest pain. Coronary angiography also is indicated in patients with chest discomfort and hypertrophic cardiomyopathy in whom a surgical procedure is planned to correct outflow tract obstruction.

3. Arteritis

Some patients with inflammatory processes affecting the aorta, such as Takayasu arteritis, may have coronary artery involvement requiring coronary artery revascularization. In such patients, coronary angiography is required before the surgical procedure. Kawasaki disease can result in coronary artery aneurysm and coronary artery stenosis producing myocardial ischemia or silent occlusion and may require coronary angiographic assessment [24,25].

4. Chest trauma

Patients who have an acute MI shortly after blunt or penetrating chest trauma may have atherosclerotic CAD, but coronary artery obstruction or damage has been reported in the absence of coronary atherosclerosis [27]. Furthermore, myocardial contusion may simulate acute MI. Infrequently, coronary angiography is indicated in the management of such patients.

Recommendations for use of coronary angiography in other conditions

Class I

- **1.** Diseases affecting the aorta when knowledge of the presence or extent of coronary artery involvement is necessary for management (e.g., aortic dissection or aneurysm with known coronary disease). (Level of Evidence: B)
- **2.** Hypertrophic cardiomyopathy with angina despite medical therapy when knowledge of coronary anatomy might affect therapy. (Level of Evidence: C)
- **3.** Hypertrophic cardiomyopathy with angina when heart surgery is planned. (Level of Evidence: B)

Class IIa

- **1.** High risk for coronary disease when other cardiac surgical procedures are planned (e.g., pericardiectomy or removal of chronic pulmonary emboli). (Level of Evidence: C)
- **2.** Prospective immediate cardiac transplant donors whose risk profile increases the likelihood of coronary disease. (Level of Evidence: B)
- **3.** Asymptomatic patients with Kawasaki disease who have coronary artery aneurysms on echocardiography. (Level of Evidence: B)
- **4.** Before surgery for aortic aneurysm/dissection in patients without known coronary disease.
- **5.** Recent blunt chest trauma and suspicion of acute MI, without evidence of preexisting CAD. (Level of Evidence: C)

3. Special considerations regarding coronary angiography

3.1. Accuracy

Cineangiographic images of coronary arteries have been the principal clinical tool for determining the severity of coronary luminal stenosis. Modern angiographic equipment has a resolution of four to five line pairs per millimeter with a six-inch field of view, the usual image magnification for coronary angiography [28]. Validation studies that use known phantoms show a high correlation between actual size and that measured by quantitative coronary angiography (QCA) (r = 0.95) [29–32]. The resolution of these phantom studies indicates the precision of coronary angiography to be 0.02 to 0.04 mm. Factors that limit resolution in the clinical setting include grainy films from "quantum mottling" and motion artifact that, in a clinical setting, limit resolution to 0.2 mm, far less than that realized from static images of known phantoms. Other factors, such as angulation, overlap of vessels and image tube resolution can also influence accuracy in the clinical setting. Nevertheless, the accuracy of coronary angiography does allow for anatomic detail that is not obtainable by current noninvasive or other invasive technology. Only intravascular ultrasound, which is discussed in Appendix C, has an image resolution greater than that of coronary angiography. However, intravascular ultrasound cannot visualize the entire coronary tree nor define the anatomic course of the coronary vessels. It is also limited by shadowing from heavy calcification and by its inability to image very small vessels or very severe stenosis.

3.2. Digital imaging of coronary angiography

Recent advances in computer storage technology have made feasible digital acquisition, processing and archival storage of angiographic images obtained during cardiac catheterization. Widespread conversion from cineangiographic film to digital archiving and storage is anticipated during the next decade. Analog storage technologies such as super VHS videotape and analog optical disks have inadequate resolution to faithfully record coronary angiography. Digital storage methods are generally adequate but until recently have lacked standardization, which precluded easy exchange of digital angiograms between centers with different equipment. The development of the Digital Imaging and Communication standard (DICOM) for cardiac angiography ensures compatibility between equipment from participating vendors.

In the interventional era, the advantages of digital angiography are important. The image quality provided by digital angiography is better than any common videotape format. Improvements in computer speed and processing capability enable rapid replay of coronary injection sequences, as well as evaluation of the results of each intervention and identification of complications such as intraluminal thrombus and dissection. In many laboratories, the availability of high-quality images during catheterization permits diagnostic and therapeutic catheterization to consist of a single procedure, a capability with significant implications for the cost of interventional procedures. Industry sources now estimate that >75% of existing laboratories are equipped with digital imaging capability.

The ACC Cardiac Catheterization Committee is coordinating efforts to develop and promote a standard for archival storage and exchange of digital cardiac angiography. The committee has joined in this common cause with an industry organization, the National Electrical Manufacturers Association (NEMA), and representatives of the American College of Radiology (ACR). The ACR and NEMA have recently released an interim standard known as Digital Imaging Communication in Medicine (DICOM version 3.0).

The initial efforts of the standards committee have focused on adoption of a file format and physical medium for interchange of digital angiographic studies. To transfer images between medical centers, the sender would generate a DICOM-compatible file for review by the receiver. Recently, this working group has chosen a recordable form of the common CD- ROM, termed CD-R, as the official exchange medium. Nearly all equipment vendors have announced support for this format.

3.3. Reproducibility

In clinical practice, the degree of coronary artery obstruction is commonly expressed as the percent diameter stenosis. This is done by comparing the diameter of the site of greatest narrowing (minimal lumen diameter) to an adjacent segment assumed to be free of disease. In clinical practice, the most common method used to estimate the percent diameter narrowing is subjective visual assessment. Because vasomotor tone can alter the reference diameter, nitroglycerin is frequently administered before angiography to improve the reproducibility of the measurement. Several studies have shown that measurement of the degree and extent of luminal narrowing correlates with symptoms as well as with assessments of coronary flow reserve (CFR) and abnormalities on treadmill exercise testing, perfusion imaging with Tl or sestamibi, stress echocardiography and fast computerized tomography [33–37]. In addition, the percent diameter reduction and the number of stenosis of >50% to 70% correlate with long-term outcome [33–37].

3.4. Limitations

Although coronary angiography is considered the reference standard for anatomic assessment of coronary obstructions, there are limitations to the technique. When luminal narrowings are present on coronary angiography (in the absence of spasm), pathological analyses almost always demonstrate severe atherosclerotic obstruction. Even minor angiographic abnormalities are associated with a poorer long-term outcome than are completely normal appearing angiograms. Coronary angiography has a high predictive value for the presence of CAD when abnormalities are present. However, the converse is not true. A normal coronary angiogram does not exclude atherosclerosis, and in fact, most pathological studies suggest that angiography grossly underestimates the extent and severity of atherosclerosis [38–42]. Several factors contribute to this discrepancy.

First, angiography depicts coronary anatomy from a planar two-dimensional silhouette of the contrast-filled vessel lumen. However, coronary lesions are often geometrically complex, with an eccentric luminal shape such that one angle of view may misrepresent the extent of narrowing [39]. Two orthogonal angiograms should demonstrate more correctly the severity of most lesions, but adequate orthogonal views are frequently unobtainable because the stenosis may be obscured by overlapping side branches, disease at bifurcation sites, diographic foreshortening or tortuosity. This can be especially difficult in the left main coronary artery, where identifying a significant stenosis is of utmost clinical importance [43].

Second, an adaptive phenomenon, coronary remodeling," contributes to the inability of coronary angiography to identify mild atherosclerosis [44]. Remodeling was initially observed on histology as the outward displacement of the external vessel wall in vascular segments with significant atherosclerosis. In the early phases of atherosclerosis, this vessel enlargement "compensates" for luminal encroachment, thereby concealing the atheroma from the angiogram. When the atherosclerotic plaque becomes severe, luminal encroachment becomes evident. Although such mild lesions do not restrict blood flow, clinical studies have demonstrated that these minimal or even unseen angiographic lesions represent an important predisposing cause of acute coronary syndromes, including MI [55].

Third, assessment of luminal diameter narrowing is complicated by the frequent absence of a normal reference segment[56]. Angiography visualizes only the lumen of the vessel and cannot determine if the wall of the reference segment has atherosclerosis [38–42]. In the presence of diffuse reference segment disease, percent stenosis will predictably underestimate the true amount of diameter narrowing.

Finally, in the setting of percutaneous intervention, the assumptions underlying simple projection imaging of the lumen are further impaired. Necropsy studies and intravascular ultrasound demonstrate that most mechanical coronary interventions exaggerate the extent of luminal eccentricity by fracturing or dissecting the atheroma within the lesion [45–49]. The angiographic appearance of the postintervention vessel often consists of an enlarged, although frequently "hazy" lumen [46]. In this setting, the lumen size on angiography may overestimate the vessel cross-sectional area and misrepresent the actual gain in lumen size.

Experimental and clinical studies have shown that when percent stenosis is >50%, the ability to increase blood flow in response to metabolic demands is impaired [50]. This augmentation of coronary blood flow to demand is termed the coronary flow reserve. Determination of CFR requires measurement of blood flow at rest and after induction of reactive hyperemia, usually by administration of a coronary vasodilator. Several methods for measurement of CFR in patients have been developed, including intracoronary Doppler flow probes, digital angiography and quantitative PET [51–54].

Coronary collaterals can provide significant additional blood flow to territories served by stenotic vessels [58]. In general, collaterals are not evident unless resting ischemia is present, such as that which occurs with a stenosis.90%. In many patients, collateral flow merely restores normal resting blood flow but does not provide adequate flow when metabolic demand increases. The presence of collaterals, however, is associated with preservation of myocardial function after MI, reduced myocardial ischemia on noninvasive stress testing, and reduced ischemia during angioplasty [59,60]. Paradoxically, a greater ischemic response on noninvasive functional testing with adenosine than with exercise has been reported in the presence of collaterals, presumably due to an increase in the coronary steal phenomenon [61]. Collateral blood flow can only be semiquantified by angiography [62], and precise assessment of perfusion by angiography is poor. This inability to adequately measure collateral flow is one of the factors that prevent accurate assessment of the functional significance of coronary stenosis by angiography alone [57].

3.5. Contrast agents

For an understanding of the pharmacologic properties and adverse effects of contrast agents, the reader is referred to the 1993 review of the subject by the ACC Cardiovascular Imaging Committee [63] and the 1996 review by Hirshfeld [64].

Except for a less potent anticoagulant effect, nonionic agents are better tolerated and have fewer side effects than ionic agents [63]. Several randomized trials have compared their use during cardiac angiography. Barrett et al. [65] compared a nonionic low-osmolar contrast agent with an ionic high-osmolar contrast agent. Although adverse events were reduced, severe reactions were confined to patients with underlying severe cardiac disease. These authors supported the use of nonionic low-osmolar agents in these high-risk patients. Steinberg et al. [66]

The difference in the incidence of any major contrast reaction is proportional to the New York Heart Association clinical function class, rising from 0.5% for class I patients to 3.6% for class IV patients [68]. Given these observations, it has been suggested that nonionic agents should be reserved for patients who are at high risk for adverse reactions and that ionic agents should be used for all other patients [64].

Factors that have been associated with high risk of adverse reactions to contrast media include prior adverse reaction to contrast agents, age >65 years, New York Heart Association functional class IV (or hemodynamic evidence of congestive heart failure), impaired renal function (creatinine >2.0 mg/dL), acute coronary syndromes (unstable angina or acute MI) and severe valvular disease (aortic valve area <0.7 cm² or mitral valve area <1.25 cm²) [64]. It is recommended that the individual practitioner appropriately assess the cost and benefit relationship when selecting contrast agents in any individual patient and that a strategy of reserving nonionic agents for patients who are at high risk of adverse reactions is prudent and cost-effective.[69]

ACC/AHA classifications of class I, II, and III. These classes summarize the indications for coronary angiography as follows:

Class I: Conditions for which there is evidence and/or general agreement that this procedure is useful and effective.

Class II: Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of performing the procedure. *Class IIa:* Weight of evidence/opinion is in favor of usefulness/ efficacy. *Class IIb:* Usefulness/efficacy is less well established by evidence/opinion. *Class III:* Conditions for which there is evidence and/or general agreement that the procedure is not useful/effective and in some cases may be harmful.[70,71]

Coronary angiography indications

- Unstable angina or Chest pain [uncontrolled with medications or after a heart attack]
- Heart attack
- Aortic Stenosis
- Before a bypass surgery
- Abnormal treadmill test results
- Determine the extent of coronary artery disease

- Disease of the heart valve causing symptoms (syncope, shortness of breath)
- To monitor rejection in heart transplant patients
- Syncope or loss of consciousness in patients with aortic valve disease
- Pain in the Jaw, Neck or Arm

Risks

- Generally the risk of serious complications ranges from 1 in 1,000 to 1 in 500. Risks of the procedure include the following :
- Stroke
- Heart attack
- Irregular heart beats
- Low blood pressure
- Injury to the coronary artery
- Allergic reaction to contrast dye[3]

Rare risks and complications include:

- Need for emergency heart surgery or angioplasty.
- A stroke.
- Heart attack.
- Surgical repair of the groin/arm puncture site or blood vessel.
- Abnormal heart rhythm that continues for a long time. This may need an electric shock to correct.
- An allergic reaction to the x-ray dye.[2]

Other, less common complications include:

- Arrhythmias. These irregular heartbeats often go away on their own. However, your doctor may recommend treatment if they persist.
- Kidney damage caused by the dye that's used during the test.
- Blood clots that can trigger a stroke, heart stroke, or other serious problems.
- Low blood pressure.[2]

Coronary angiography contraindications

- Fever
- Kidney failure or dysfunction
- Problems with blood coagulation (Coagulopathy)

- Active systemic infection
- Uncontrolled Blood Pressure (Hypertension)
- Allergy to contrast (dye) medium
- Transient Ischemic attack
- Severe anemia
- Electrolyte imbalance
- Uncontrolled rhythm disturbances (arrhythmias)
- Uncompensated heart failure[4]

Author details

Chiu-Lung Wu¹ and Chi-Wen Juan^{1,2}

*Address all correspondence to: juanchiwen@yahoo.com.tw

1 Department of Emergency Medicine, Kuang Tien General Hospital, Sha-Lu, Taichung, Taiwan, R.O.C.

2 Department of Nursing, Hungkuang University, Taichung, Taiwan, R.O.C.

References

- [1] Ross J Jr, Brandenburg RO, Dinsmore RE, et al. ACC/AHA Guidelines for coronary angiography. A report of the American College of Cardiology/American Heart Association Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures (Subcommittee on Coronary Angiography). J Am Coll Cardiol 1987;10:935– 50.
- [2] Marcus ML, Schelbert HR, Skorton DJ, et al, editors. *Cardiac Imaging: A Companion to Braunwald's Heart Disease*. Philadelphia, Pa: WB Saunders, 1991.
- [3] Grossman WB, Baim DS, editors. *Cardiac Catheterization, Angiography and Intervention.* Philadelphia, Pa: Lea & Febiger, 1991.
- [4] Braunwald E, Mark DB, Jones RH, et al. Clinical Practice Guideline Number 10: Unstable Angina: Diagnosis and Management. 86th ed. Rockville, Md: US Dept of Health and Human Services, Agency for Health Care Policy and Research, 1994. AHCPR publication 94-0602.

- [5] Braunwald E, Jones RH, Mark DB, et al. Diagnosing and managing unstable angina: Agency for Health Care Policy and Research.Circulation 1994;90:613–22.
- [6] DeWood MA, Spores J, Notske R, et al. Prevalence of total coronary occlusion during the early hours of transmural myocardial infarction. N Engl J Med 1980;303:897–902.
- [7] de Feyter PJ, van den Brand M, Serruys PW, Wijns W. Early angiography after myocardial infarction: what have we learned? Am Heart J 1985;109:194 –9.
- [8] Lincoff AM, Topol EJ. Illusion of reperfusion: does anyone achieve optimal reperfusion during acute myocardial infarction? Circulation 1993;88:1361–74.
- [9] Lincoff AM, Topol EJ, Califf RM, et al. Significance of a coronary artery with thrombolysis in myocardial infarction grade 2 flow "patency" (outcome in the thrombolysis and angioplasty in myocardial infarction trials): Thrombolysis and Angioplasty in Myocardial Infarction Study Group. Am J Cardiol 1995;75:871–6.
- [10] Simes RJ, Topol EJ, Holmes DR Jr, et al. Link between the angiographic substudy and mortality outcomes in a large randomized trial of myocardial reperfusion: importance of early and complete infarct artery reperfusion. GUSTO-I Investigators. Circulation 1995;91:1923–8.
- [11] Vavuranakis M, Bush CA, Boudoulas H. Coronary artery fistulas in adults: incidence, angiographic characteristics, natural history. Cathet Cardiovasc Diagn 1995;35:116–120.
- [12] Carvalho JS, Redington AN, Oldershaw PJ, Shinebourne EA, Lincoln CR, Gibson DG. Analysis of left ventricular wall movement before and after reimplantation of anomalous left coronary artery in infancy. Br Heart J 1991;65:218 –22.
- [13] Leberthson RR, Dinsmore RE, Bharati S, et al. Aberrant coronary artery origin from the aorta: diagnosis and clinical significance. Circulation 1974;50:774 –9.
- [14] Levin DC, Fellows KE, Abrams HL. Hemodynamically significant primary anomalies of the coronary arteries: angiographic aspects. Circulation 1978;58:25–34.
- [15] Dilsizian V, Bonow RO. Current diagnostic techniques of assessing myocardial viability in patients with hibernating and stunned myocardium. Circulation 1993;87:1– 20.
- [16] Braunwald E, Rutherford JD. Reversible ischemic left ventricular dysfunction: evidence for the "hibernating myocardium." J Am Coll Cardiol 1986;8:1467–70.
- [17] Greenberg B, Quinones MA, Koilpillai C, et al. Effects of long-term enalapril therapy on cardiac structure and function in patients with left ventricular dysfunction: results of the SOLVD echocardiography substudy. Circulation 1995;91:2573–81.
- [18] Akins CW, Pohost GM, Desanctis RW, Block PC. Selection of angina-free patients with severe left ventricular dysfunction for myocardial revascularization. Am J Cardiol 1980;46:695–700.

- [19] Rankin JS, Newman GE, Muhbaier LH, Behar VS, Fedor JM, Sabiston DC Jr. The effects of coronary revascularization on left ventricular function in ischemic heart disease. J Thorac Cardiovasc Surg 1985;90:818–32.
- [20] Dilsizian V, Bonow RO, Cannon RO III, et al. The effect of coronary artery bypass grafting on left ventricular systolic function at rest: evidence for preoperative subclinical myocardial ischemia. Am J Cardiol 1988;61:1248 –54.
- [21] Bonow RO, Udelson JE. Left ventricular diastolic dysfunction as a cause of congestive heart failure: mechanisms and management. AnnIntern Med 1992;117:502–10.
- [22] Creswell LL, Kouchoukos NT, Cox JL, Rosenbloom M. Coronary artery disease in patients with type A aortic dissection. Ann Thorac Surg 1995;59:585–90.
- [23] Rizzo RJ, Aranki SF, Aklog L, et al. Rapid noninvasive diagnosis and surgical repair of acute ascending aortic dissection: improved survival with less angiography. J Thorac Cardiovasc Surg 1994;108:567–74.
- [24] Kato H, Ichinose E, Yoshioka F, et al. Fate of coronary aneurysms in Kawasaki disease: serial coronary angiography and long-term follow-up study. Am J Cardiol 1982;49:1758–66.
- [25] Suzuki A, Kamiya T, Kuwahara N, et al. Coronary arterial lesions of Kawasaki disease: cardiac catheterization findings of 1,100 cases. Pediatr Cardiol 1986;7:3–9.
- [26] Walston A II, Behar VS. Spectrum of coronary artery disease in idiopathic hypertrophic subaortic stenosis. Am J Cardiol 1976;38: 12–6.
- [27] Oren A, Bar-Shlomo B, Stern S. Acute coronary occlusion following blunt injury to the chest in the absence of coronary atherosclerosis. Am Heart J 1976;92:501–5.
- [28] Nissen SE, Gurley GL. Assessment of coronary angioplasty results by intravascular ultrasound. In: Serruys PW, Straus BH, King SB III, editors. Restenosis After Intervention With New Mechanical Devices. Dordrecht, Netherlands: Kluwer 1992:73–96.
- [29] Keane D, Haase J, Slager CJ, et al. Comparative validation of quantitative coronary angiography systems: results and implications from a multicenter study using a standardized approach. Circulation 1995;91:2174–83.
- [30] Reiber JH, Serruys PW, Kooijman CJ, et al. Assessment of short-, medium-, and longterm variations in arterial dimensions from computer-assisted quantitation of coronary cineangiograms. Circulation 1985;71:280–8.
- [31] Reiber JH, van der Zwet PM, Koning G, et al. Accuracy and precision of quantitative digital coronary arteriography: observer-, short-, and medium-term variabilities. Cathet Cardiovasc Diagn 1993;28:187–98.
- [32] Reiber JHC, Reiber JC, Serruys PW, editors. Advances in QuantiQuantitative Coronary Arteriography. Dordrecht, Netherlands: Kluwer,1993:55–132.
- [33] Amanullah AM, Aasa M. Significance of ST segment depression during adenosineinduced coronary hyperemia in angina pectoris and correlation with angiographic,

scintigraphic, hemodynamic, and echocardiographic variables. Int J Cardiol 1995;48:167–76.

- [34] Arnese M, Salustri A, Fioretti PM, et al. Quantitative angiographic measurements of isolated left anterior descending coronary artery stenosis: correlation with exercise echocardiography and technetium- 99m 2-methoxy isobutyl isonitrile single-photon emission computed tomography. J Am Coll Cardiol 1995;25:1486 –91.
- [35] Fallavollita JA, Brody AS, Bunnell IL, Kumar K, Canty JM Jr. Fast computed tomography detection of coronary calcification in the diagnosis of coronary artery disease: comparison with angiography in patients ,50 years old. Circulation 1994;89:285–90.
- [36] Salustri A, Arnese M, Boersma E, et al. Correlation of coronary stenosis by quantitative coronary arteriography with exercise echocardiography. Am J Cardiol 1995;75:287–90.
- [37] Tron C, Kern MJ, Donohue TJ, et al. Comparison of quantitative angiographically derived and measured translesion pressure and flow velocity in coronary artery disease. Am J Cardiol 1995;75:111–7.
- [38] Arnett EN, Isner JM, Redwood DR, et al. Coronary artery narrowing in coronary heart disease: comparison of cineangiographic and necropsy findings. Ann Intern Med 1979;91:350–6.
- [39] Blankenhorn DH, Curry PJ. The accuracy of arteriography and ultrasound imaging for atherosclerosis measurement: a review. Arch Pathol Lab Med 1982;106:483–9.
- [40] Grondin CM, Dyrda I, Pasternac A, Campeau L, Bourassa MG, Lesperance J. Discrepancies between cineangiographic and postmortem findings in patients with coronary artery disease and recent myocardial revascularization. Circulation 1974;49:703– 8.
- [41] Vlodaver Z, Frech R, Van Tassel RA, Edwards JE. Correlation of the antemortem coronary arteriogram and the postmortem specimen. Circulation 1973;47:162–9.
- [42] Roberts WC, Jones AA. Quantitation of coronary arterial narrowing at necropsy in sudden coronary death: analysis of 31 patients and comparison with 25 control subjects. Am J Cardiol 1979;44:39–45.
- [43] Isner JM, Kishel J, Kent KM, Ronan JA Jr, Ross AM, Roberts WC. Accuracy of angiographic determination of left main coronary arterial narrowing: angiographic-histologic correlative analysis in 28 patients. Circulation 1981;63:1056–64.
- [44] Glagov S, Weisenberg E, Zarins CK, Stankunavicius R, Kolettis GJ. Compensatory enlargement of human atherosclerotic coronary arteries. N Engl J Med 1987;316:1371–5.
- [45] Keeley EC, Lange RA, Landau C, Willard JE, Hillis LD. Quantitative assessment of coronary arterial diameter before and after balloon angioplasty of severe stenoses. Am J Cardiol 1995;75:939–40.

- [46] Waller BF. "Crackers, breakers, stretchers, drillers, scrapers, shavers, burners, welders and melters:" the future treatment of atherosclerotic coronary artery disease? A clinical-morphologic assessment. J Am Coll Cardiol 1989;13:969–87.
- [47] Tenaglia AN, Buller CE, Kisslo KB, Stack RS, Davidson CJ. Mechanisms of balloon angioplasty and directional coronary atherectomy as assessed by intracoronary ultrasound. J Am Coll Cardiol 1992;20:685–91.
- [48] Berkalp B, Nissen SE, De Franco AC, et al. Intravascular ultrasound demonstrates marked differences in surface and lumen shape following interventional devices (abstr). Circulation 1994;90:I-58.
- [49] DeFranco AC, Tuzcu EM, Moliterno DJ, et al. Overestimation of lumen size after coronary interventions: implications for andomized trials of new devices (abstr). Circulation 1994;90(pt 2):I-550.
- [50] Gould KL, Lipscomb K, Hamilton GW. Physiologic basis for assessing critical coronary stenosis: instantaneous flow response and regional distribution during coronary hyperemia as measures of coronary flow reserve. Am J Cardiol 1974;33:87–94.
- [51] Kern MJ, Donohue TJ, Aguirre FV, et al. Assessment of angiographically intermediate coronary artery stenosis using the Doppler flowire. Am J Cardiol 1993;71:26–33D.
- [52] Lamm C, Dohnal M, Serruys PW, Emanuelsson H. High-fidelity translesional pressure gradients during percutaneous transluminalcoronary angioplasty: correlation with quantitative coronary angiography. Am Heart J 1993;126:66 –75.
- [53] Nissen SE, Elion JL, Booth DC, Evans J, DeMaria AN. Value and limitations of computer analysis of digital subtraction angiography in the assessment of coronary flow reserve. Circulation 1986;73:562–71.
- [54] Nissen SE. Radiographic principles in cardiac catheterization. In: Roubin GS, Califf RM, O'Neill W, Phillips H, Stack R, editors. Interventional Cardiac Catheterization: Principles and Practice. New York: Churchill Livingstone, 1993:409 –25.
- [55] Little WC, Constantinescu M, Applegate RJ, et al. Can coronary angiography predict the site of a subsequent myocardial infarction in patients with mild-to-moderate coronary artery disease? Circulation 1988;78:1157–66.
- [56] Leung WH, Alderman EL, Lee TC, Stadius ML. Quantitative arteriography of apparently normal coronary segments with nearby or distant disease suggests presence of occult, nonvisualized atherosclerosis. J Am Coll Cardiol 1995;25:311–7.
- [57] Sambuceti G, Parodi O, Giorgetti A, et al. Microvascular dysfunction in collateral-dependent myocardium. J Am Coll Cardiol 1995; 26:615–23.
- [58] Sasayama S. Effect of coronary collateral circulation on myocardial ischemia and ventricular dysfunction. Cardiovasc Drugs Ther 1994; 8:327–34.

- [59] Dacanay S, Kennedy HL, Uretz E, Parrillo JE, Klein LW. Morphological and quantitative angiographic analyses of progression of coronary stenoses: a comparison of Qwave and non-Q-wave myocardial infarction. Circulation 1994;90:1739–46.
- [60] Dellborg M, Emanuelsson H, Swedberg K. Silent myocardial ischemia during coronary angioplasty. Cardiology 1993;82:325–34.
- [61] Akutsu Y, Hara T, Michihata T, et al. Functional role of coronary collaterals with exercise in infarct-related myocardium. Int J Cardiol 1995;51:47–55.
- [62] Nishimura S, Kimball KT, Mahmarian JJ, Verani MS. Angiographic and hemodynamic determinants of myocardial ischemia during adenosine thallium-201 scintigraphy in coronary artery disease. Circulation 1993;87:1211–9.
- [63] Ritchie JL, Nissen SE, Douglas JS Jr, et al. Use of nonionic or low osmolar contrast agents in cardiovascular procedures: American College of Cardiology Cardiovascular Imaging Committee. J Am Coll Cardiol 1993;21:269–73.
- [64] Hirshfeld JW. Radiographic contrast agents. In: Marcus ML, editor. Cardiac Imaging: A Companion to Braunwald's Heart Disease. Philadelphia, PA: WB Saunders, 1996.
- [65] Barrett BJ, Parfrey PS, Vavasour HM, O'Dea F, Kent G, Stone E. A comparison of nonionic, low-osmolality radiocontrast agents with ionic, high-osmolality agents during cardiac catheterization. N Engl J Med 1992;326:431–6.
- [66] Steinberg EP, Moore RD, Powe NR, et al. Safety and cost effectiveness of high-osmolality as compared with low-osmolality contrast material in patients undergoing cardiac angiography. N Engl J Med 1992;326:425–30.
- [67] Jacobson PD, Rosenquist CJ. The introduction of low-osmolar contrast agents in radiology: medical, economic, legal, and public policy issues. JAMA 1988;260:1586–92.
- [68] Hirshfeld JW Jr, Kussmaul WG, DiBattiste PM, Investigators of the Philadelphia Area Contrast Agent Study. Safety of cardiac angiography with conventional ionic contrast agents. Am J Cardiol 1990; 66:355–61.
- [69] Patrick J. Scanlon, David P. Faxon, Anne-Marie Audet, et al. ACC/AHA guidelines for coronary angiography: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Coronary Angiography) developed in collaboration with the Society for Cardiac Angiography and Interventions J. Am. Coll. Cardiol. 1999;33;1756-1824.
- [70] Ryan TJ, Anderson JL, Antman EM, et al. ACC/AHA guidelines for the management of patients with acute myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on the Management of Acute Myocardial Infarction). J Am Coll Cardiol 1996;28:1328–428.
- [71] American Heart Association, ACC/AHA Guidelines for Coronary Angiography, (*Circulation*).1999;99:pp2345-2357.

[72] Simes RJ, Topol EJ, Holmes DR Jr, et al. Link between the angiographic substudy and mortality outcomes in a large randomized trial of myocardial reperfusion: importance of early and complete infarct artery reperfusion. GUSTO-I Investigators. Circulation 1995; 91:1923– 8.



