INTRODUCTION

Coronary artery disease (CAD) remains the leading cause of mortality in most industrialized countries, although age-standardized mortality related to CAD has decreased by more than 40% during the last two decades.[1,2] Half of this decline resulted from prevention and reduction in major risk factors, whereas the other half has been attributed to medical treatment and revascularization.[3] Coronary artery disease is the result of atherosclerosis ,a progressive disorder of the vessel walls, with formation of plaques throughout the arterial system.[4] Vascular inflammation may lead to disruption of the endothelium overlying a plaque and cause subsequent intravascular thrombosis.[5] Symptoms related to atherosclerosis vary depending on the location and degree of stenosis of the vessels and the occurrence, site, and severity of plaque disruption. Coronary atherosclerosis may thus be asymptomatic or cause angina pectoris, myocardial infarction (MI), heart failure, arrhythmias, and sudden death.[4]

Chronic stable angina, the initial manifestation of CAD in approximately 50% of all patients, [6] is usually caused by the obstruction of at least 1 large epicardial coronary artery by atheromatous plaque. Angina is due to the mismatch between myocardial oxygen demand and supply, resulting in myocardial ischemia. Angina pectoris is characterized by sub sternal discomfort, heaviness, or a pressure-like feeling, which may radiate to the jaw, shoulder, back, or arm and which typically lasts several minutes. These symptoms are usually brought on by exertion, emotional stress, cold, or a heavy meal and are relieved by rest or nitroglycerin within minutes. Symptoms can be classified as characteristic of typical angina, atypical angina, or non-cardiac chest pain, depending on whether the chest pain characteristics meet all 3, 2, or less than 2 of the aforementioned criteria, respectively (Diamond classification [7]). The Canadian Cardiovascular Society (CCS)'s grading for angina severity [8] has gained widespread popularity (Table 1).

Anginal "equivalents," such as epigastric discomfort, dyspnea, fatigue, or faintness, may be the dominant symptom in some patients, particularly elderly ones. Coronary artery disease may be asymptomatic or present with such complications as an acute coronary syndrome (unstable angina or MI), congestive heart failure, cardiac arrhythmias, or sudden death.

Table (1): Modified Canadian Cardiovascular Society Grading for Angina Severity [8]

Class I	Angina occurs with strenuous or rapid or prolonged exertion
Class II	Angina occurs with moderate exertion (e.g., walking > 2 blocks on level ground and climbing > 1 flight of ordinary stairs at a normal pace and in normal conditions; walking uphill; or walking or climbing stairs rapidly, in the cold, in the winter, under emotional stress, or during the first few hours after awakening)
Class III	Angina occurs with mild exertion (walking 1 or 2 blocks on level ground and climbing 1 flight of stairs in normal conditions and at normal pace)
Class IV	Angina occurs with any level of exertion and may be present at rest

Physical examination is often unrevealing in patients with stable angina. Nonetheless, examination to check for the presence of such comorbid conditions as hypertension, tobacco stains, chronic lung disease (smoking), xanthelasma (hyperlipidemia), and evidence of non-coronary atherosclerotic disease (decreased peripheral pulses, carotid or renal artery bruits, abdominal aortic aneurysm) is essential because these findings may be important in determining the risks and benefits of a comprehensive treatment strategy and the need for additional investigations.

Cardiac auscultation, particularly during an episode of chest pain, can reveal a third or fourth heart sound due to transient left ventricular (LV) dysfunction or a mitral regurgitation murmur due to papillary muscle dysfunction during myocardial ischemia. Bibasilar rales may be indicative of congestive heart failure.

The importance of estimating the probability of substantial CAD by obtaining a detailed history and performing a risk factor assessment and focused physical examination cannot be overemphasized. Knowing the prevalence of CAD in the population helps the physician estimate pretest probability [9, 10] (Table 2).

Table (2): Pretest Likelihood of Coronary Artery Disease in Symptomatic Patients According to Age and Sex [11]

Age (y)	Nonanginal chest pain		Atypical angina		Typical angina	
	Men	Women	Men	Women	Men	Women
30-39	4	2	34	12	76	26
40-49	13	3	51	22	87	55
50-59	20	7	65	31	93	73
60-69	27	14	72	51	94	86

Data are reported as percentage of patients.

Risk factors, such as smoking, hypertension, diabetes, hyperlipidemia, and a family history of MI before age 60 years, increase the likelihood of CAD.[12,13] resting electrocardiography should be performed in all patients with suspected angina, although findings may be normal in approximately half of patients with stable angina, including those with severe CAD, [14] particularly in the setting of preserved LV function.[15]

Electrocardiographic evidence of ST-T wave changes or LV hypertrophy (even though nonspecific) favor the diagnosis of angina, and prior Q wave MI on electrocardiography is highly suggestive of underlying CAD.[16]

Various conduction disturbances, most frequently left bundle branch block (LBBB) and left anterior fascicular block, may occur in patients with stable angina and are often associated with impairment of LV function and reflect multi vessel disease or previous myocardial damage.

During an episode of angina pectoris, 50% of patients with normal findings on resting electrocardiography develop electrocardiographic abnormalities, with the most

common finding being ST-segment depression. However, ST segment elevation and normalization of previous resting ST-T wave depression or inversion (pseudo normalization) may also develop.

Noninvasive stress tests, although extremely helpful tools, are often underused in the United States and the United Kingdom in patients undergoing percutaneous coronary intervention (PCI) [17, 18]; however, they may perhaps be overused in other situations.[19] These tests are most useful in patients with an intermediate pretest probability of CAD because in such patients the results of the stress test, whether positive or negative, will have the greatest effect on the post test probability (according to Bayesian principles) and consequently on clinical management.

Several noninvasive stress tests are available; sensitivity and specificity of the various tests shown in table 3[11, 20-22]. These data have been obtained from small studies with catheterization laboratory referral biases with a high pretest likelihood of CAD.

Table (3): Sensitivity and Specificity of Noninvasive Stress Test for the Diagnosis of Coronary Artery Disease [11, 20-22]

Noninvasive stress test	Sensitivity	Specificity
Exercise electrocardiography	0.68	0.77
Exercise SPECT	0.87	0.73
Adenosine SPECT	0.89	0.75
Adenosine PET	0.89	0.86
Exercise echocardiography	0.86	0.81
Dobutamine echocardiography	0.82	0.84
Dobutamine magnetic resonance imaging	0.89	0.84
Exercise magnetic resonance imaging	0.84	0.85

PET = positron emission tomography; SPECT= single photon emission computed tomography.

Exercise electrocardiography is a good initial choice in patients who can exercise and who have normal electrocardiographic findings at rest[11]; however, in many other situations an imaging technique is preferred. Imaging studies are recommended for patients whose findings on resting electrocardiography make the relevance of changes with stress (LBBB, ST-segment depression ≥1 mm, ventricular paced rhythm, or Wolff-Parkinson-White syndrome) difficult to assess, for patients who have had previous coronary revascularization, and for patients in whom clinical evaluation and exercise electrocardiography have provided insufficient information to guide management.

A pharmacological imaging test is required if the patient is unable to exercise (due to orthopedic limitations, frailty, deconditioning, symptomatic heart failure, cardiac arrhythmia, acute pulmonary embolus, acute aortic dissection, acute myopericarditis, and possibly acute MI within the previous 48 hours). The choice between stress nuclear

imaging vs stress echocardiography in many cases should depend on the local expertise of the laboratory.

Adenosine or dipyridamole nuclear perfusion imaging is the preferred test for patients with LBBB or ventricular paced rhythm because of increased false-positive findings with exercise or dobutamine echocardiography.

In obese patients or women with large breasts, positron emission tomography stress may be superior to conventional myocardial perfusion imaging because of its ability to perform attenuation correction.

Magnetic resonance imaging (MRI) is an exciting new stress imaging technique that may be used for both adenosine perfusion and dobutamine wall motion imaging; however, it is not widely available.

The American College of Cardiology / American Heart Association guidelines [11] discourage use of noninvasive testing for CAD in asymptomatic patients, except in those with evidence of possible myocardial ischemia on ambulatory electrocardiography or with severe coronary calcification on electron-beam computed tomography (CT). Screening of asymptomatic patients with type 2 diabetes does not reduce MI or death and is not recommended.[23]

Invasive coronary angiography may be indicated for diagnostic purposes in all patients who have survived sudden cardiac death, in patients with a high pretest probability of having left main or 3-vessel disease, and in patients who cannot undergo noninvasive testing.

Other indications include patients with uncertain diagnosis on noninvasive testing, high-risk occupational requirements (pilots) clinically suspected non atherosclerotic causes of ischemia or possible vasospasm with need for provocative testing, multiple hospital admissions, or an overriding patient desire for definitive diagnosis of the presence or absence of obstructive disease.[11]

Risk Stratification of Patients with Chronic CAD

The major clinical and angiographic predictors of survival of patients with CAD are as follows: (1) LV function, (2) anatomic extent and severity of coronary atherosclerosis, (3) severity of ischemia, (4) tempo and severity of angina and/or the presence of recent plaque rupture, and (5) the patient's general health and non-coronary comorbid conditions.

Other non-cardiovascular factors that may be determinants of overall mortality, including ethnicity (south Asian), socioeconomic status, drug adherence, depression, and modification of risk factors, are not addressed in this section but nonetheless may exert a substantial contributory influence on prognosis.

Despite a growing reliance on noninvasive or invasive testing, history and physical examination continue to be helpful in assessing the severity of CAD. Pryor et al [12] identified clinical characteristics typical angina, previous MI, age, sex, duration of symptoms, hypertension, diabetes, hyperlipidemia, smoking, carotid bruit, and chest pain frequency—and formulated a model using these characteristics to accurately estimate the likelihood of severe disease in a patient. An easy to use 5-point cardiac risk score was

developed by Hubbard et al [24] using male sex, typical angina, history or electrocardiographic evidence of MI, diabetes, and use of insulin as risk factors for predicting severe CAD (3-vessel or left main) at different ages (figure 1).

Resting electrocardiography is helpful in risk stratification. The prognosis of patients with normal findings on electrocardiography is usually excellent because normal electrocardiographic findings imply normal LV function.[15]

In contrast, such abnormalities as Q waves, ST-T changes, LV hypertrophy,[25] LBBB, bifascicular block, second and third degree atrioventricular block, atrial fibrillation, and ventricular arrhythmias [26] are associated with a worse prognosis.

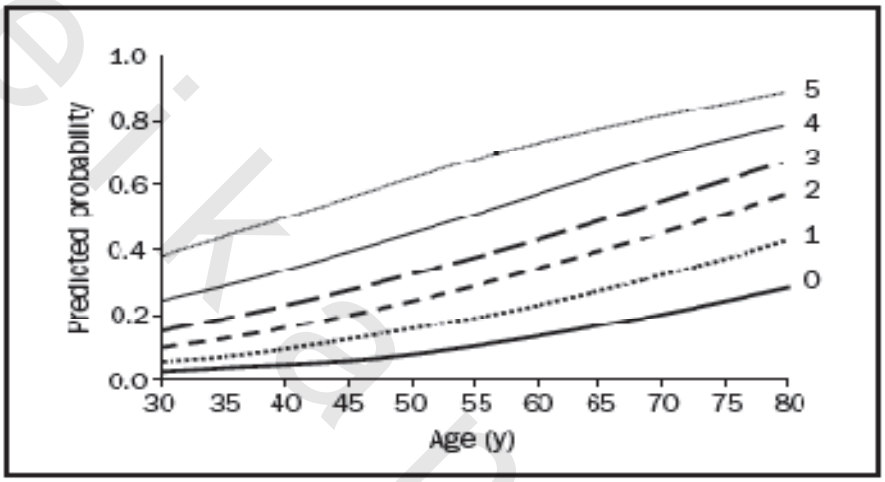

Figure (1): Composite graph estimating the probability of severe coronary artery disease on the basis of a 5 point risk score that awards 1 point for each of the following variables: male sex, typical angina, history or electrocardiographic evidence of myocardial infarction, diabetes, and use of insulin. Each curve shows the probability of severe disease as a function of age of a given risk score.[24]

Left ventricular function is a major predictor of long term survival in patients with CAD [27](figure 2, A) and end-systolic LV volume was found to be the best predictor of survival after MI.[28] Assessment of LV function, usually with echocardiography, is appropriate in patients with symptoms or signs of heart failure, a history of MI, or pathologic Q waves on electrocardiography.

Exercise electrocardiographic testing is recommended as the first choice for all patients with an intermediate or high probability of CAD, except for those who cannot exercise nor have electrocardiographic abnormalities that compromise interpretation or those for whom the information is unlikely to alter management. Risk should also be stratified for patients with known chronic CAD who have a marked change in the severity of cardiac symptoms with exercise electrocardiography. A useful tool for calculating risk is the Duke tread mill score, [29] which incorporates exercise capacity, ST-segment deviation, and angina as major risk determinants.

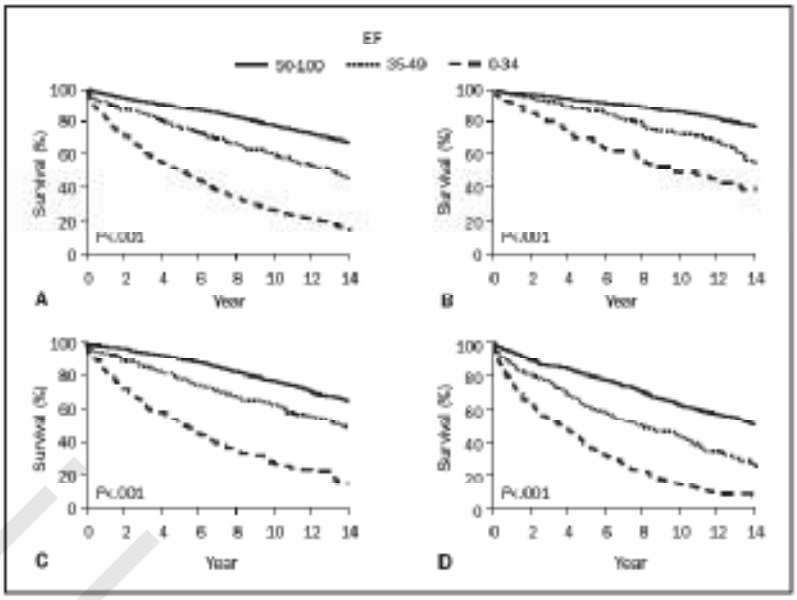

Figure (2): Survival of medical treated patients with coronary artery disease according to ejection fraction (EF) and number of diseased vessels. A patients with 1-, 2- or 3-vessel disease by EF; B patients with 1-vessel disease by EF; C patients with 2-vessel disease by EF; and D patients with 3-vessel disease by EF. [27]

The score is calculated using the following formula:

Exercise Time in Minutes - (5 × the Maximum ST-Segment Deviation in Millimeters) - (4 × the Angina Index [0, no pain; 1, angina; and 2, angina that caused discontinuation of the test]) table 4.

Table (4): Survival According to Risk Groups on The Basis of Duke Treadmill Scores

Risk group	5-y survival (%)	5-y survival free of death or MI (%)
Low(≥ 5)	97	93
Moderate (-10 to 4)	91	86
High (≤ -11)	72	63

Other risk factor determinants include extensive and prolonged ST-segment depression, transient ST-segment elevation, abnormal heart rate recovery, and delayed systolic blood pressure response to exercise [11]. The incremental value of imaging tests as the initial testing modality vs exercise electrocardiography is controversial, [30] but they are the first choice in patients with electrocardiographic abnormalities that preclude interpretation of the exercise tracing or in patients who are taking digoxin. Imaging tests may provide additional information regarding the extent, severity, and location of myocardial jeopardy; an estimate of the extent of irreversible scar tissue; and LV function. Stress imaging studies are also indicated for assessment of the functional implications of coronary lesions in planning PCI.[11] Risk stratification on the basis of the results of noninvasive stress testing is shown in Table 5.

Table (5): Risk Stratification on the Basis of Noninvasive Testing [11]

High risk (>3% annual mortality rate)
Severe resting LV dysfunction (LVEF <35%)
High-risk treadmill score (≤–11)
Severe exercise LV dysfunction (exercise LVEF <35%)
Stress-induced large perfusion defect (particularly if anterior)
Stress-induced multiple perfusion defects of moderate size
Large, fixed perfusion defect with LV dilation or increased lung uptake (thallium-201)
Stress-induced moderate perfusion defect with LV dilation or increased lung uptake (thallium-201)
Echocardiographic wall motion abnormality (involving >2 segments) developing with low dose of dobutamine (≤10 μg/kg/ min) or at a low heart rate (<120 beats/min)
Stress echocardiographic evidence of extensive ischemia
Intermediate risk (1%-3% annual mortality rate)
Mild/moderate resting LV dysfunction (LVEF, 35%-49%)
Intermediate-risk treadmill score (–10 to 4)
Stress-induced moderate perfusion defect without LV dilation or increased lung intake (thallium-201)
Limited stress echocardiographic ischemia with a wall motion abnormality only with higher doses of dobutamine involving ≤2 segments
Low risk (<1% annual mortality rate)
Low-risk treadmill score (≥5)
Normal or small myocardial perfusion defect at rest or with stress
Normal stress echocardiographic wall motion or no change of limited resting wall motion abnormalities during stress

LV= left ventricular; LVEF= LV ejection fraction.

Coronary angiography, which helps stratify risk in patients on the basis of the extent and location of atherosclerosis, is indicated in patients who have high-risk criteria on noninvasive testing, patients who have angina and signs and symptoms of congestive heart failure, patients who have survived sudden cardiac arrest or serious ventricular arrhythmias, and as a first test in patients with CCS class III or IV angina despite medical therapy. Coronary angiography is acceptable for patients with CCS class I or II angina who are intolerant to medication, whose lifestyle is still impaired by these symptoms, who have LV dysfunction, or whose risk status is uncertain after noninvasive testing.

A low threshold for angiographic evaluation is recommended for patients with angina who have undergone previous revascularization and in patients with a history of MI.[31]

The extent and severity of coronary atherosclerotic disease and LV dysfunction identified on cardiac catheterization are the most powerful predictors of long-term outcome [27, 32] (figure2, B-D)

Several prognostic indices have been used to quantify the extent of severity of CAD, but the simplest classification into 1-, 2-, or 3-vessel CAD or left main CAD is the most widely used and is effective.[33] Additional risk stratification is provided by the severity of obstruction and its location, with proximal lesions predicting reduced survival rate (figure 3).[32] Quantifying the extent of coronary disease, including non-obstructive lesions, also adds to risk stratification.[34]

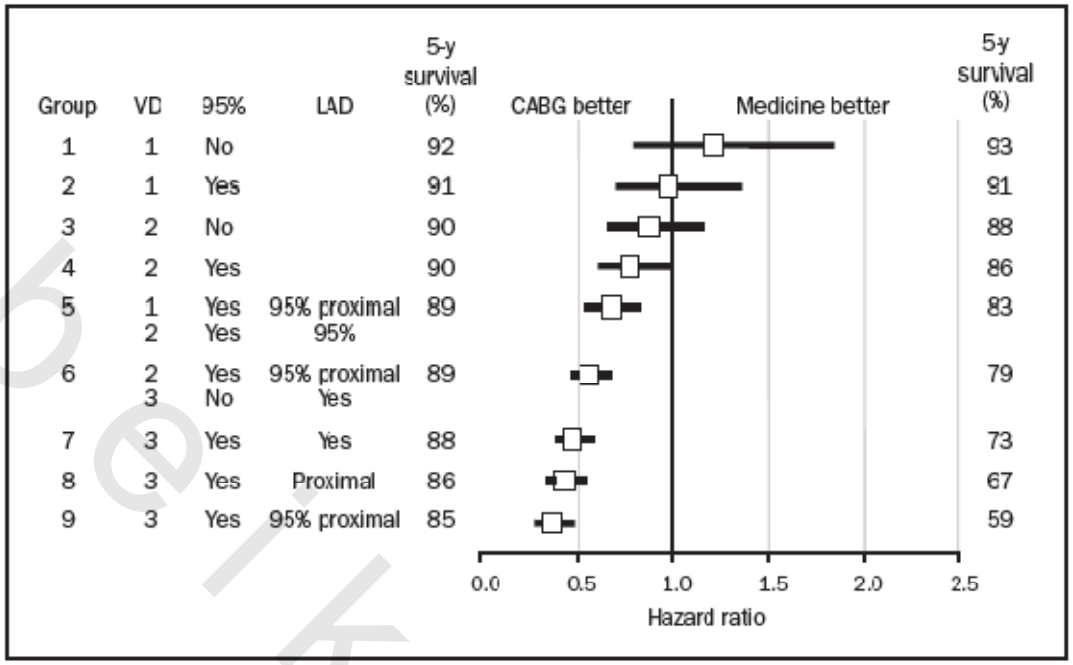

Figure (3): Five year survival rate in patients according to severity and proximity of coronary artery lesions and adjusted hazard ration for coronary artery bypass grafting (CABG) vs medical treatment. 95% = 95% coronary artery stenosis; LAD = left anterior descending artery; VD = number of diseased vessels.[32]

Cardiac CT and MRI

Coronary artery calcium scanning with CT is a screening tool that has no role in patients with established CAD in whom the presence of coronary artery calcification is a given. Furthermore, the specificity of the coronary calcium score for obstructive coronary lesions is low.[11, 35]

Although CT coronary angiography is showing promise for noninvasive detection of obstructive CAD in major epicardial arteries, it is still limited by a high number of false-positive results (up to 50% with severe calcification and coronary stents), specific patient selection (heart rate must be regular and <70 beats/min; patient must hold breath for 15 seconds), and high-dose radiation exposure.[36, 37] Magnetic resonance imaging may be used for stress perfusion or stress wall motion imaging (table 3) as well as noninvasive coronary angiography.[22, 38]

Most heart valve prostheses and vascular stents are compatible with MRI; however, MRI cannot be used in the presence of certain implanted metal objects or medical devices, such as pacemakers or implantable cardioverter- defibrillators.[39] However, electronic rhythm management devices and other cardiovascular devices are being developed that could be compatible with MRI.

Speckle-tracking echocardiography

Speckle-tracking echocardiography (STE) is a new noninvasive ultrasound imaging technique that allows for an objective and quantitative evaluation of global and regional myocardial function, Speckle-tracking echocardiography is based on analysis of the spatial dislocation (referred to as tracking) of speckles (defined as spots generated by the interaction between the ultrasound beam and myocardial fibers) on standard B-mode 2-dimensional images [40].

Before the introduction of this technique, only tagged MRI had enabled an accurate analysis of the deformation components that characterize myocardial dynamics, although tagged MRI may be considered the reference standard in this area of study, its routine use is limited by its high costs, poor availability, relative complexity and time consuming [41].

It is angle-independent technique where random noise is filtered out, while keeping myocardial features temporally stable throughout the cardiac cycle and unique for each myocardial region independent from cardiac translational movements [42].

When tracking a defined region of speckles, a software algorithm follows the change in geometric position of this region, frame by frame,(simultaneously in multiple regions within an image plane) figure 4.

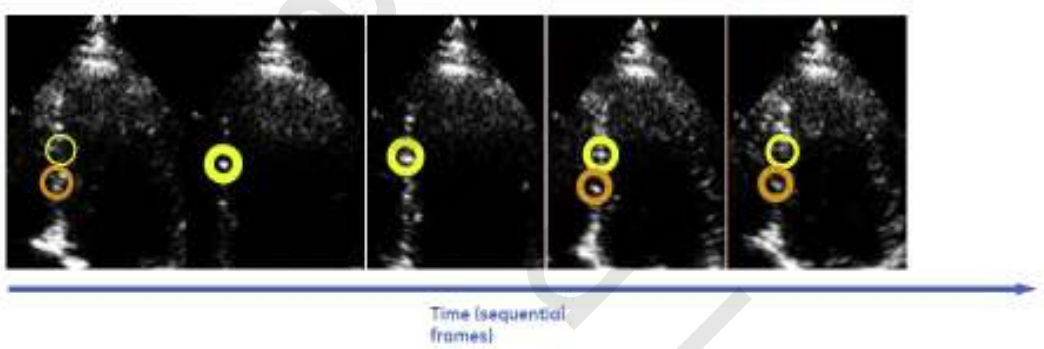

Figure (4): Motion and velocities are analyzed by calculating frame-to-frame changes using "natural acoustic tagging." New features (orange circles) keep coming into the image as old ones (yellow circles) fade away.

The algorithm tracks the wall motion and calculates the percentage of lengthening or shortening in a set of three longitudinal 2D- image planes (apical long, two chamber and four chamber) and displays the results for each plane. It then combines the results of all three planes in a single bull's-eye summary, which presents the analysis for each segment along with a global peak strain value for the left ventricle [43]. A representative example of bull's eye summery is shown in figure 5.

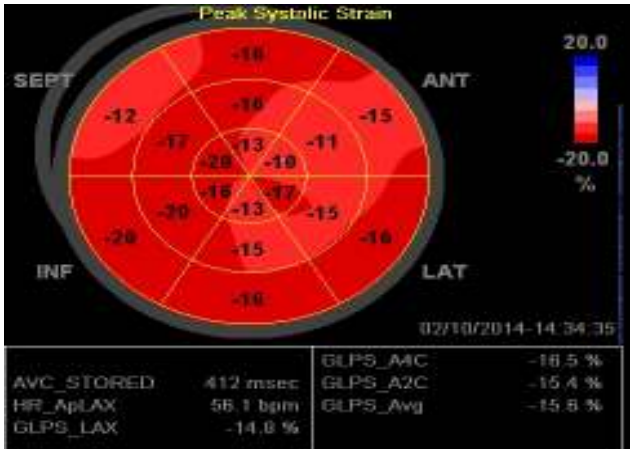

Figure (5): Representative example of the measurement of segmental and global peak systolic longitudinal strain (PSLS) shown on bull's eye display from a patient with three- vessel CAD.

Validation

2D-STE is highly reproducible as its semi-automated nature guarantees good intraobserver and interobserver reproducibility and analysis is affected by only small intraobserver and interobserver variability [44].

It has been validated for the assessment of myocardial deformation against DTI. Since the deformation parameters can be calculated in two dimensions (while TDI derived parameters are one-dimensional), this technique is often referred to as two dimensional strain echocardiography (2DSE).[45]In a direct comparison of TDI and 2DSE with MRI-tagging as a reference, comparable values were found for 2DSE and TDI in normal and dysfunctional myocardial segments. For radial measurements, 2DSE was more reliable than TDI [46]

Strain (ε sys %)

Strain represents the degree of deformation of the analyzed myocardial segment in relation to its initial dimensions. It is expressed a percentage.

The strain equation (ε) is as follows: **ε = L –L0/L0** Where L is the length of the object after deformation and L0 is the basal length of the object. By convention, depending on the direction, a lengthening or thickening deformation is given a positive value, whereas a shortening or thinning deformation is given a negative one [47].

The contractile function of the heart has several components, Speckle tracking is able to analyze each of these components separately. In short axis views you will be able to look at radial and circumferential deformation, while the apical views are used to assess longitudinal function.

During systole, ventricular myocardial fibers shorten with translational movement from the base to the apex; such myocardial deformation represents longitudinal strain and the consequent reduction of the distance between single kernels is represented by negative trend curves. Figure 6

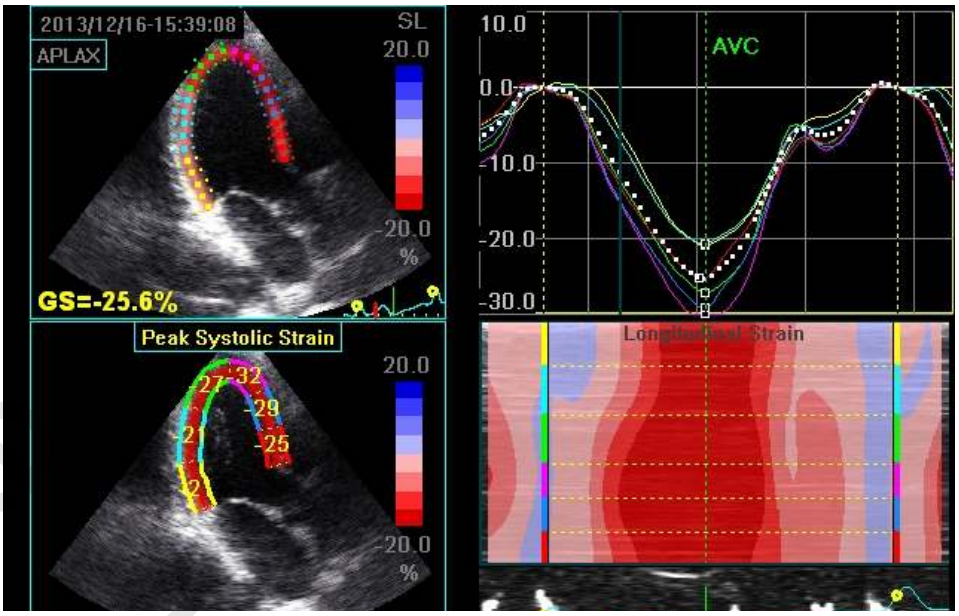

Figure (6): Display of negative staintrend curves from 3 chamber view of a healthy person.

Subendocardial function is driven mostly by longitudinal contraction, and is often impaired before the circumferential or radial component deteriorates.

Thus, longitudinal function serves as an early marker of left ventricular dysfunction. There are numerous disease entities in which strain derived deformation parameters are reduced before the ejection fraction drops. Longitudinal strain allows detection of subclinical left ventricular dysfunction. Strain can be computed during every point and time of the cardiac cycle. The best parameter for systolic function however is the peak systolic strain.[43]

Peak systolic strain is defined as the maximal shortening (at any region of the myocardium) during systole (after the onset of the QRS complex and before aortic valve closure occurs). It is possible to measure strain in individual segments by averaging all segments of the entire ventricle. This value is called global peak systolic strain (GPSS). Usually this is done for longitudinal strain from all apical views. Therefore it is also called global longitudinal peak systolic strain (GLPSS). A meta-analysis of 2597 subjects from 24 studies reported mean normal value of GLS = –19.7%. Normal values for strain are depicted at table 6.

Table (6): Mean left ventricular longitudinal peak systolic segmental strain values calculated from 242 healthy subjects [48]

LV segment 4 ch view	Mean peak systolic longitudunal strain	LV segment (2 ch view)	Mean peak systolic longitudinal strain	LV segment 3ch view	Mean peak systolic longitudinal strain
Basal septal	- 13.7± 4	Basal anterior	- 20.1 ± 4	Basal anteroseptal	-18.3 ± 3.5
Mid septal	-18.7 ± 3	Mid anterior	- 18.8 ± 3.4	Mid anteroseptal	-19.4 ± 3.2
Apical septal	-22.3 ± 4.8	Apical anterior	- 19.4 ± 5.4	Apical anteroseptal	-18.8 ± 5.9
Apical lateral	-19.2 ± 5.4	Apical inferior	-22.5 ± 4.5	Apical posterior	- 17.7 ± 6
Mid lateral	-18.1 ± 3.5	Mid inferior	- 20.4 ± 3.5	Mid posterior	-16.8 ± 5
Basal lateral	-17.8 ± 5	Basal inferior	-17.1 ± 3.9	Basal posterior	-14.6 ± 7.4

Comparability between 2DSE derived and DTI-derived strain

Both TDI and 2DSE derived parameters are accurate quantitative measures of local longitudinal myocardial deformation, and thus will yield comparable values for local deformation and deformation rates in both the LV[49]and the RV [50]. However, variability in the measurements and technical factors make minor differences between the calculated parameters that should be anticipated when comparing values of TDI and 2DSE in a single patient [50].

Most of the observed differences between the 2 techniques are inherent to some limitations. For example, TDI is not possible when the investigated wall is not optimally aligned, or in presence of a stationary artifact, whereas 2DSE is less influenced by these interferences [51].

In contrast to TDI, 2DSE is an angle independent technique as the movement of speckles can be followed in any direction. For the apical views this implies that not only longitudinal, but also transverse parameters can be calculated, which is not possible in TDI recordings. In short axis images both circumferential and radial parameters can be calculated for all myocardial segments [51].

On the other hand, 2DSE has a relative low temporal resolution hindering tracking in presence of high heart rates. This is not a problem with TDI, where frame-rates are higher; up to 250 FPS (frames/sec) [51].

The number of segments which can be analyzed with either technique is comparable, although some studies reported a lower feasibility for 2DSE compared to TDI [52].

Practical differences between the 2 techniques are an additional factor. Training requirements for both image acquisition and post processing are equal. Acquisition time is comparable between the two techniques. However, Post processing time is significantly shorter with speckle tracking with approximately 2 minutes for 2DSE and 11 minutes for TDI analysis of 16 segments from 3 apical views. This mainly results from a more rapid tracking and parameter extraction in 2DSE, which is automated, while TDI is generally perceived as more difficult particularly for post-processing [53].

The reproducibility of 2DSE is generally better in the LV than with TDI, but in the RV, this difference is not seen. 2DSE derived and DTI derived strain parameters are comparable, but STE has advantages with regard to ease of application and analysis of data. However, both techniques are of equal or complementary value in the quantification of myocardial function. [53]

Limitations

Although 2DSE is a useful technique, it has intrinsic limitations of 2D imaging, such as use of foreshortened views that affect accuracy of quantification of individual components of myocardial motion. In addition, the assumption that speckles remain within the 2D imaging plane and can be adequately tracked throughout the cardiac cycle may not always be valid, because of the complex 3D motion of the heart chambers. The inability of 2DSE to measure one of the three components of local displacement vector is an important limitation, which affects the accuracy of the derived indices of local dynamics [54].

Furthermore, considering close dependence of 2DSE on single cardiac cycle for myocardial deformation analysis, it is not possible to conduct strain measurements in patients with non-sinus rhythm[54].

Another limitation of 2DSE is the fact that calculated parameters are averaged over the myocardial segment when using the "results page" of software program, in conditions with small regions of myocardial dysfunction, such as early stages of hypertrophic cardiomyopathy or arrhythmogenic right ventricular dysplasia, the averaging result in normal deformation parameters due to normal deformation properties of the adjacent myocardium.

Potential Pitfalls of 2D STE:

Suboptimal tracking of the endocardial border may be a problem with 2DSE. Another important limitation is its sensitivity to acoustic shadowing or reverberations, which can result in underestimation of true deformation. Therefore, when strain traces appear non-physiologic, signal quality and suboptimal tracking should be considered as potential causes.

Strengths and Weaknesses of 2D STE

Both DTI and 2D STE measure motion against a fixed external point in space (i.e., the transducer). However, 2D STE has an advantage of being able to measure this motion in any direction within the image plane, whereas DTI is limited to the velocity component toward or away from the probe. This property of 2D STE allows measurement of circumferential and radial components regardless of direction of beam[55].

However, 2D STE is not completely angle independent, because ultrasound images normally have better resolution along the ultrasound beam compared with the perpendicular direction. Therefore, in principle,2D STE works better for measurements of motion and deformation in the direction along the ultrasound beam than in other directions [55].

Similar to other 2D imaging techniques, 2D STE relies on good image quality and the assumption that morphologic details can be tracked from one frame to the next (i.e., that they can be identified in consecutive frames), which may not be true when out of plane motion occurs [41].