

# Acute coronary syndromes and diagnostic methods

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## Acute coronary syndrome

Acute coronary syndrome is defined as a clinical presentation often accompanied by acute chest pain or other myocardial damage markers and changes on ECG due to impaired blood flow of heart muscle.

ACS involves arterial thrombotic diseases such as unstable angina pectoris (USAP), non-ST elevation myocardial infarction (NSTEMI) and ST-elevation myocardial infarction (STEMI) [1]. Definition of acute coronary syndrome according to the guidelines by American and European heart societies is shown in table 1 [2].

## Epidemiology

Despite decreased incidence and mortality rates in recent years, coronary heart diseases remain one of the leading causes of death. According to TEKHARF study by the Turkish Cardiology Society, 2 million people have coronary artery disease in Turkey. According to TEKHARF data, cardiovascular diseases are responsible for 45% of all deaths in our country [3]. It is estimated that the number of patients with heart disease will reach 3.4 million if this negative situation, similar to the rates in developed countries, continues [2].

Increasing incidence of NSTEMI and STEMI with age may be related to a triggering factor such as inflammation. NSTEMI is known to be often accompanied by comorbidities such as diabetes mellitus and renal failure.

## Physiopathology

Myocardial ischemic symptoms are mostly resulted from disrupted integrity of vulnerable atherosclerotic plaque [4]. Acute coronary syndrome is caused by occlusive thrombus in more than 90% of cases [5].

Atherosclerosis is a chronic condition progressing up to the occlusion of the vessel, which begins in the intima of the artery [6]. This process influences entire vasculature. Intima and media layers develop plaque. Rupture or erosion of the plaque is required in order to become a thrombus [7]. Acute ischemic events begin as a result of plaque rupture, endothelial damage, inflammation and release of some mediators. Impaired integrity in the arterial wall stop the flow

in the vessel lumen, causing ischemia and damage [8]. Diffuseness of necrosis varies depending on the presence of collateral flow, duration of ischemia and the extent of the involved area.

The thrombus obstructing the vessel lumen is a mixture of clots consisting of platelet rich fibrin and erythrocytes. Although there are several factors regarding time of the occurrence of acute coronary syndrome, decreased fibrin degradation activity due to increased levels of platelet aggregation and plasminogen activator inhibitor plays a role in occurrence of plaque fissure and rupture often early in the morning [9].

USAP, NSTEMI or STEMI occurs based on prevention of blood flow in the vessel lumen by growth of the plaque or degree of the obstruction in the vessel lumen as a result of thrombus occurring due to plaque rupture [10]. Pathological studies in patients with NSTEMI acute coronary syndrome have found myocardial damage areas in the sites supplied by the relevant vascular structure [11].

## Risk factors

There are numerous risk factors in the etiology of coronary artery diseases. Roughly, these risk factors can be divided into two groups as changeable and unchangeable factors. These risk factors are shown in table 2.

**Age:** Being aged over 45 years in men and 55 years in women is accepted as a risk factor for developing atherosclerosis.

**Sex:** Although mortality occurs in older ages among women, the rate of mortality is higher. This might be attributed to that women are more overweight in middle-advanced ages, more often have diabetes and hypertension, more commonly use oral contraceptives, and have higher cholesterol levels compared to men [12,13].

**Family History:** In many studies, patients with a family history of early coronary artery disease have been shown to carry a 1.5 to 1.7 folds higher risk, indicating the importance of family history. Cholesterol levels are increased in families with a history of premature MI and associated mortality. This is an independent risk factor alone [14,15].

**Smoking:** Smoking is an important and changeable risk factor of atherosclerosis. In addition, the mortality rate can be raised even

**Table 1.** ESC/ACC\* definition of acute coronary syndrome

1. Rapid elevation of CK-MB and relatively slow elevation of troponins that are among myocardial damage markers accompanied by at least one of the following factors
• Ischemic symptoms
• Pathologic Q waves on ECG
• ECG findings compatible with myocardial damage
• Intervention to coronary artery
2. Pathological findings of acute coronary syndrome

\* ESC/ACC: European Society of Cardiology / American College of Cardiology

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**Table 2.** Risk Factors

1. Unchangeable factors
• Age
• Sex
• Family history
2. Changeable factors
A. Major
• Hyperlipidemia
• Smoking
• Hipertension
• Diabetes
• Obesity
• Sedentary lifestyle
B. Minor
• Excessive alcohol consumption
• Stress
• A type personality
• Fibrinogen elevation
• Homocysteine elevation

with exposure to cigarette smoke [16]. Mortality rates of those who quit smoking decrease by 36% compared to those who did not quit, regardless of age and gender [17].

Smoking triggers other mechanisms in addition to its negative effects on sympathetic activity of the body and decreased oxygen consumption of the heart muscle. Chronic smoking accelerates atherosclerotic process and increases LDL oxidation. Nitric oxide biosynthesis and endothelium-dependent vasodilatation are impaired following smoking [18,19].

**Hypertension:** Hypertension has an increasing prevalence. A diastolic or systolic elevation in blood pressure has been associated with cardiovascular mortality and morbidity [20]. A decrease of 5-5 mmHg in blood pressure decreases the risk of developing both cerebrovascular disease and coronary artery disease [21]. Therefore, hypertension is considered a factor increasing risk of cardiovascular disease.

**Diabetes Mellitus:** In diabetic patients, functions of endothelial and smooth muscles are impaired, and atherosclerotic load is increased because of the increased platelet adhesion, which is a critical step of atherogenesis, compared to non-diabetic persons [22]. When diabetic patients have been compared with race and age matched persons without diabetes mellitus, future risk of diabetic patients has been found as 2 to 4 times higher [23]. In a study by Gu K et al., 75% of deaths were found to be resulted from coronary artery disease in diabetic patients [24].

**Hyperlipidemia:** The risk of coronary artery disease shows correlation with blood cholesterol levels [25]. Comprehensive clinical trials have shown a decrease in cardiovascular diseases by dropping LDL cholesterol levels with various means and treatment. Therefore, the correlation of atherosclerosis with cholesterol is widely recognized [26].

Understanding of vascular physiology in atherosclerosis has been convincing about the responsibility of LDL cholesterol for the development of atherosclerosis. Mutations of LDL receptor in patients with familial homozygous hypercholesterolemia, cause hypercholesterolemia resulting in atherosclerosis at early periods of life.

**Obesity:** Obesity is epidemiologically associated with an increased vascular risk [27]. However, the pathophysiology of risk effect on vascular structure is still controversial.

**Stress:** Among changeable risk factors, stress increases vascular risk. It can worsen myocardial ischemia by increasing oxygen consumption

through adrenergic stimulation. Stress can cause vasoconstriction, decreasing oxygen supply [28].

Because clinic depression increase the prevalence of smoking, sedentary life and hypertension, its effect continues even if the other risk factors are resolved [29].

**Sedentary Life:** Exercise, which moderately affect LDL and total cholesterol levels, provide a steady increase in HDL and a decrease in triglyceride levels. In addition, as regular exercise cause a decrease in blood pressure, it reduces the risk to a level comparable with many drugs [30].

Regular exercise decreases risk factors that can cause atherosclerosis. It reduces the level of C reactive protein and regulates its endothelial function. Exercise regulates the variables that control plasma viscosity such as plasminogens and fibrins, showing a positive effect in regulation of hemostatic parameters [31].

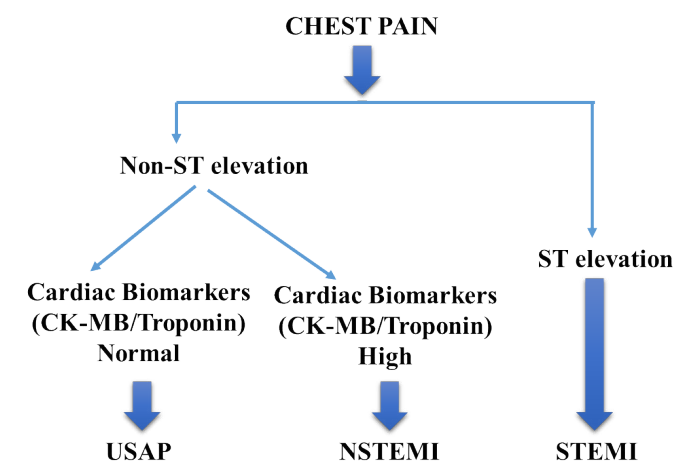
**Alcohol:** It is known that high levels of alcohol consumption disrupt myocardial contractility and even may cause cardiomyopathy. Although HDL levels are higher in alcohol abusers; studies have shown that the risk is increased due to raised blood pressure in people with moderate-to-high alcohol consumption [32].

## Types of acute coronary syndromes

Acute coronary syndromes are divided into two groups according ST segment change on ECG as non-ST elevation and ST-elevation [33]. These groups are shown in figure 1.

**NSTEMI Acute Coronary Syndrome:** In NSTEMI thrombus, which did not lead to complete occlusion in the ruptured or eroded plaque is responsible for the pathophysiology. This is a white thrombus with low number of fibrins [34]. There is acute coronary syndrome presentation and ST elevation is not observed. Syndromes with this clinical picture are divided into two groups as USAP and NSTEMI. These groups are evaluated together as pathophysiology, clinic and treatment approach are similar between these groups.

USAP is a clinical table in form of severe exercise angina or changed characteristics, which lasts longer than 20 minutes in resting. It defines the clinic between stable angina pectoris and MI. It is distinguished from NSTEMI with serum markers [35]. Cardiac enzymes are elevated in NSTEMI. Although Q wave is not observed in most patients, some

**Figure 1.** Types of acute coronary syndromes

studies have observed Q wave between 2-15% [36, 37]. Among cardiac markers, troponin is a more precise indicator of myocardial necrosis. The condition in which cardiac troponin is elevated, but CK-MV levels are not, is evaluated as low myocardial damage. This is clinically significant, because it is of clinical importance in outcome and treatment [38].

**STEMI Acute Coronary Syndrome:** In the events where constant and occlusive thrombus is predominant, ST is elevated and this often indicate total obstruction of acute coronary artery. Approximately 30 minutes after complete occlusive lesion, necrosis begins at the subendocardium and begins to expand in the subepicardium [39]. Occlusion is resulted from thrombus clustering in thrombosis. Afterwards, fibrin plays a role in the consolidation of the plug in the early period [40].

### Diagnosis of Acute Coronary Syndrome

Early diagnosis is important in acute coronary syndrome. It is easy to establish the diagnosis in the presence of findings on ECG. However, a considerable number of cases have no ST elevation [41]. A good anamnesis is often diagnostic. Some cases require cardiac enzymes and ECG follow-up.

The most challenging cases for clinicians include presentations with atypical signs without significant ST elevation, and the cases showing bundle branch block and ventricular hypertrophy.

**Anamnesis:** Typical symptoms of acute coronary symptom sensation of retrosternal pressure or burn radiating to the left arm or neck. In addition to this sign, there may be other complaints such as vomiting, dyspnea, syncope and sweating.

On the contrary, there may be acute coronary syndrome presentations with atypical symptoms. These complaints may show symptoms similar to pleuritic and musculoskeletal pains that could lead us to other diagnoses. Atypical symptoms are more common in young, female gender, patients with renal failure, diabetes and dementia.

**Physical Examination:** Physical examination is usually normal in acute coronary syndrome. Some patients may have a fear of death, sympathetic discharge related restlessness, anxiety, tachycardia and hypertension, while some MI patients may have bradycardia and hypotension.

Other possible examination findings include splitting of S2, murmur of mitral regurgitation and changes in the characteristics of apical beat. In some cases pulmonary rales may be heard.

**Electrocardiogram:** ECG is the leading tool that can be used in the diagnosis. The first thing to do is ordering a 12-lead ECG in suspected cases. Making a comparison with previous ECG of the patient can increase its diagnostic value. It is important to repeat it during observation in terms of comparison. Sensitivity of ECG is not higher than 10% and specificity tan 90%.

Changes of ST in the repeated ECG may be seen especially in Prinzmetal's angina [38].

A ST segment elevation  $\geq 1$  mm in two or more adjacent derivations, and T negativity in derivations with dominant R wave strongly suggest acute coronary syndrome (38). A ST elevation  $\geq 0.5$  mm is considered significant by American guidelines [42].

Completely normal ECG does not rule out the possibility of acute coronary syndrome [43]. Studies have reported MI or USAP in approximately 10% of the patients who were discharged because of normal ECG [41].

**Biochemical Markers:** Some molecules are released from myocytes with impaired integrity due to onset of myocardial damage first into the interstitium and than into the circulation with vascular structures and lymphatics. Molecules such as CK, CK-MB, myoglobin, AST and LDH are found in the myocardial tissue.

According to a consensus report by European Society of Cardiology (ESC) and American College of Cardiology (ACC), the diagnosis of myocardial infarction can be made in the case of elevated CK-MB and troponin accompanied with symptoms compatible with myocardial ischemia [36].

- **Creatine Kinase:** Creatine kinase, which has three isoenzymes, is the most valuable marker following troponin. It elevates above normal level within 4-8 hours after acute damage, and peaks at the 24th hour. Although it varies from person to person, it regresses to normal levels in 3-4 days [44]. It can be used in detection of reinfarction at an early period and in hospital since it rapidly raised and is detected in blood.

Whereas MM isoform is concentrated in the skeletal muscle, isoenzyme BB is concentrated in the brain and kidneys. MB isoenzyme is the cardiac-sensitive isoform.

Creatine kinase may show false positivity in many disorders such as skeletal muscle trauma, intramuscular injections, heart failure, exercises and epileptic seizures.

- **Myoglobin:** Low molecular weight myoglobin is a sensitive, but non-specific protein. It raises about within 2 hours at the early period. Non-elevation of myoglobin rules out the diagnosis of myocardial infarction. Although its specificity is low, rapid elevation makes it of importance [45,46].
- **Troponin:** Troponin (Tn), which plays a role in contractility in the skeletal and heart muscles, is a molecule regulating the relationship between actin and myosin. Tn has three subtypes.  $Ca^{++}$  binding Tn C, Tn I which blocks actin-myosin interaction and Tn T which anchors the complex with thin filament structures.

Cardiac troponins can show necrosis, but do not provide information about the cause.

Tn T raises within 12 hours after the damage, and peaks at the 24th hour. It may remain high in the blood up to 14 days. Tn I begins to elevate approximately at the 6th to 12th hours, peaks at the 24th hour and may be found high in the blood for about 10 days [47,48].

Troponins are more sensitive than CK-MB. Levels of troponins can be guiding for clinicians in risk determination schemes. It is not appropriate to rule out the diagnosis based on troponin values. A follow-up over 6-12 hours is necessary to rule out acute coronary syndrome.

According to the risk classification by the American Heart Association (AHA), Tn T values between 0.01 – 0.1 ng/mL show moderate risk in NSTEMI patients. In the case of a Tn T value  $> 0.1$  ng/mL, the patient is at a high prognostic risk [42].

Similar to creatine kinase, Tn may be high in conditions other than acute coronary syndrome. Troponin may be high in many situations including myocarditis, cardiac trauma, resuscitation, pulmonary embolisms and tachyarrhythmias [9].

- **B type Natriuretic Peptide (BNP):** BNP is a neurohormone released following tension of the ventricles. It has several features such as natriuresis, diuresis and vasodilator. It is of importance in determination of disorders, because BNP is synthesized and released in a short time. It has a short half-life [49,50].

BNP can be helpful in distinguishing cardiac and non-cardiac causes of dyspnea as well as it can be used as a screening method high-risk groups for left ventricular systolic dysfunction and in some vascular disease.

It is particularly used in treatment evaluation and follow-up in heart failure. It is of paramount value in the differential diagnosis in emergency departments [51]. Whereas troponins are rather ischemia markers, BNP is associated more with heart failure and death [52].

### Risk classification

TIMI risk score was developed by the Thrombolysis in Myocardial Infarction work group (TIMI) in order to determine the possibility and treatment strategy and to predict the prognosis when diagnosing acute coronary syndrome. This is a risk classification based on low, moderate and high risk classes according to total score obtained by giving 0 or 1 point for each parameter. Patients with a 5 and higher points are classified as high-risk group, 3-4 points in moderate risk group and 0-2 points in low risk group [53]. TIMI data were obtained in ESSENCE and TIMI-11B, and the validity was confirmed in patients groups of studies including those conducted by Mayo Clinic [54, 55]. New and repeating myocardial infarction, emergency revascularization and all cause mortality have been reported by 26-40% in high-risk group, 13-20% in moderate-risk group and 4-8% in low risk group over 14 days [56]. TIMI risk score for NSTEMI patients is shown in Table 3. Each parameter is scored with 1 point.

FRISC, RITA 3 and TACTICS studies conducted in patients diagnosed with NSTEMI have reported that early application of invasive treatment decreased primary outcomes. However, early invasive intervention was not beneficial in all cases. Nevertheless, costs and likelihood of complications were in negative direction. A scoring system was developed in order to select eligible patients instead of all patients. Parameters of the FRISC risk score, which was developed with 1-year patient data, include age, gender, diabetes mellitus, ST depression, previous history of MI, elevated troponin, increased IL-6 or CRP. Early invasive method is recommended in patients who received points from 3 or more criteria [57-59]. FRISC risk score is shown in table 4. Each parameter is scored with 1 point.

In GUSTO study, the factors associated with mortality include age, low systolic blood pressure, tachycardia, high Killip class and anterior infarction. Killip classification is given in table 5 [60]. In GUSTO-1

**Table 3.** Risk score parameters for NSTEMI and USAP

1. > 65 Years old
2. Having more than 3 risk factors for coronary artery diseases
3. > 50% previously known stenosis
4. 2 or more angina within 24 hours
5. Elevated troponin level at follow-up
6. Presence of symptoms within the last 7 days despite using aspirin

**Table 4.** FRISC risk score

1. > 70 years old
2. Gender
3. Diabetes
4. Elevated troponin
5. ST depression on ECG
6. History of previous MI
7. Increased interleukin-6 or CRP

**Table 5.** Killip Classification

1. Stage 1: No rales and S3. No signs indicating heart failure
2. Stage 2: Rales + and S3.
3. Stage 3: S3 + More than 50% rals
4. Stage 4: Cardiogenic shock.

study, mortality rate was 1.1% in patients aged under 45 years, while this rate was 20.5% in patients aged over 75 years, supporting that age is a strong predictor in these patients [61].

GRACE risk score that can be used in all patients with acute coronary syndrome predicts mortality up to 6 months. When used with other risk scoring systems, GRACE, which includes data of a wide patient group, gives better results. The parameters used in GRACE scoring include age, systolic blood pressure, heart rate, presence of ST depression, Killip class, cardiac markers and cardiac arrest [62].

One of the risk parameters that should be considered in acute coronary syndromes is evaluation of ventricular functions. There is an inverse correlation between left ventricular ejection fraction and mortality. Left ventricular functions should be evaluated in patients diagnosed with MI [63].

## Cardiac physiology end evaluation

### Cardiac electrophysiology

Heart conduction system regulates cardiac cycle with specialized fibers. Eac beat consists of 3 consecutive phases. These phases of the cycle are:

1. Contraction (systole)
2. Relaxation (diastole)
3. Filling phase (diastole)

Contraction phase of the heart is known as systole. After the electrical signal create contraction with filling of the blood into the right atrium, it passes from the atrium to the ventricle through AV node in a delayed way to create ventricular contraction. The blood in the ventricle is pumped into the pulmonary trunk and aorta during the systolic phase. Relaxation follows the systolic phase. In this period, the blood is filled again with negative pressure. The aorta and pulmonary valve are closed at the beginning of the diastolic phase. Whereas atrioventricular valves are closed and pulmonary valves are opened during the systolic phase.

**Systolic Phase:** 55-70% of the blood is pumped to the periphery and lungs during the systolic phase. This phase consists of two stages.

1. Preejection stage is the time of isometric contraction, begins with the first heart sound or closing of AV valves, and involves the time to the opening of the semilunar valves. Volume does not change, but pressure is increased during this time.

2. Ejection stage begins with the opening of the aorta and pulmonary valves. Volume is rapidly decreased at the early period with sudden pressure increase and pumping of the blood into the aorta. This stage ends with the closing of the valves [64].

**Diastolic Phase:** The time from the closing of the aortic valves to the opening of the mitral valve is clinically defined as diastolic phase. Normal diastolic function is defined as the maintenance of end-diastolic volume at rest or during exercise without raising the diastolic pressure above 12 mmHg [65].

In relaxation phase, it is the phase to return pre-contraction, and this phase is energy dependent. The myofibrils return to their pre-contraction length with working of Na-K and calcium pumps.

Systolic relaxation begins in the middle of the systolic phase and lasts up to 1/3 of diastolic period. Isovolumetric relaxation time is measured with PW doppler. It is time until the colsing of the aortic valve



and opening of the mitral valve, while left ventricular volume remains constant [64]. It is influenced by the factors affecting the closing time of the aortic and mitral valves [66]. Isovolumetric relaxation time continues with rapid filling time.

Rapid filling phase begins with the opening of the mitral phase. A passive blood flow occurs with the opening due to the difference of a few mmHg, and continue until intraventricular pressure becomes equal to the left atrium pressure. Atrioventricular flow rate depends on flexibility and ventricular relaxation with pressure difference. Although the flow is passive, ventricular relaxation is a period requiring energy. Filling of the left ventricle with blood is a passive event, showing contractility of the myocardium. The more myocardium relaxes, the better the volume can it pump.

Pressure decrease in unit time is found with ' $-dP/dt$ '. It should be recorded with left ventricular pressure monitoring. It peaks with the closing of the aortic valve. As the rate will be decreased in the case of prolonged relaxation, the negative value will be decreased. Negative value is increased in sympathetic signals as the relaxation will be accelerated. Maximal value will not provide information on all events in the relaxation phase since it is an instant value. The most negative aspect of the left ventricle is to be influenced by the aortic pressure [67]. Because it increases independently from relaxation values when left ventricular pressure and aortic pressure increase [68].

There are several definitions for diastolic dysfunction: (1) an average pulmonary capillary pressure  $< 12$  mmHg, [2] right-shift of pressure-volume relationship with presence of ventricular filling resistance, (3) inability to provide ventricular filling capacity without a compensatory increase in the left atrium pressure [66].

Recent studies have shown that the main impairment occurs in diastolic functions at an early stage. According to Starling's law, low-pressure ventricle that could not be filled with a sufficient pressure increases atrium pressure over time in order to compensate [69]. Diastolic function is affected by compliance, relaxation, myocardial stress, heart rate and pericardial restriction [70].

Tau coefficient, which is known as the relaxation coefficient was formulated for the first time by Weiss et al. Being affected by afterload independently from preload shows the importance of Tau coefficient [71].

In addition to echocardiography, magnetic resonance imaging (MRI), ventriculography and invasive procedures are also used in the diagnosis of diastolic dysfunction [72]. Today the most commonly used method is Doppler echocardiography [73].

Measurement of cardiac flow rates when evaluating diastolic functions with Doppler, gives many clues in terms of dysfunction and prognosis.

In a normal physiological rhythm, flow from the left atrium to the left ventricle, which is measured via the mitral valve consists of two components.

1. E wave reflects early phase of diastolic filling
2. A wave reflects atrial contraction in the late diastolic phase

Because flow rate through the valve depends on transmitral pressure difference, E wave rate is influenced by both diastolic relaxation rate and left atrium pressure. Change pattern of E wave rate will give clues about left ventricular diastolic dysfunction and prognosis. These flow parameters are highly sensitive in showing diastolic functions.

Another diastolic evaluation is to assess the flow from the pulmonary veins to the left atrium with Doppler. Studies have shown that mitral flow is influenced by age.

Diastolic functions have been classified into four groups as normal, impaired relaxation, pseudonormal and restrictive patterns according to the standard diagnostic criteria and flow curves are shown in figure 2.

**1. Normal pattern:** E/A ratio is lower than 1 in young and healthy persons. Deceleration time is under 220 ms. IVRT is lower than 100 ms.

**2. Impaired (prolonged) relaxation pattern:** E/A ratio is lower than 1. This pattern begins to be seen with advanced age. Deceleration time and relaxation time increase. E flow rate decreases. A flow rate increases. Deceleration time is under 220 ms.

**3. Pseudonormal pattern:** This is the stage where filling pressure exceeds the upper limit as a result of decreased flexibility and prolonged relaxation. Left atrium pressure is further high. Compliance decreases in addition to the prolonged relaxation. The difference between atrium backflow and transmitral A flow is expected to be higher than 35 cm/sec in pulmonary flow analysis conducted for distinguish this pattern from the normal pattern. In normal pattern, AR is lower than 35 cm/sec. Whereas in pseudonormal pattern, E/A ratio is expected to be  $> 1$  and deceleration time between 150-200 ms. IVRT is  $< 100$  ms in this pattern.

**4. Restrictive pattern:** Myocardial wall stability is at the forefront. Flexibility and relaxation disappear [74-76]. There is a high left atrium pressure. Ventricular filling pressure increases [77]. E flow rate is 1 m/sec, deceleration time is shortened with progression of the disease and is lower than 150 ms. E/A ratio is higher than 1.5.

## Evaluation of Cardiac Functions

2003 guidelines of the American College of Cardiology stated "The use of echocardiography in acute coronary syndrome" as a procedure in Class I category [6].

The presence of wall motion anomaly in an important indicator of ischemia or infarction area. Regional left ventricular wall motions and general ventricular function can be analyzed and evaluated using many methods. These are classified as semi-quantitative and quantitative evaluations [6].

The first impaired echocardiographic parameter in acute ischemia is diastolic function, while systolic dysfunction occurs later. It manifests as segmental wall motion disorder on echocardiography.

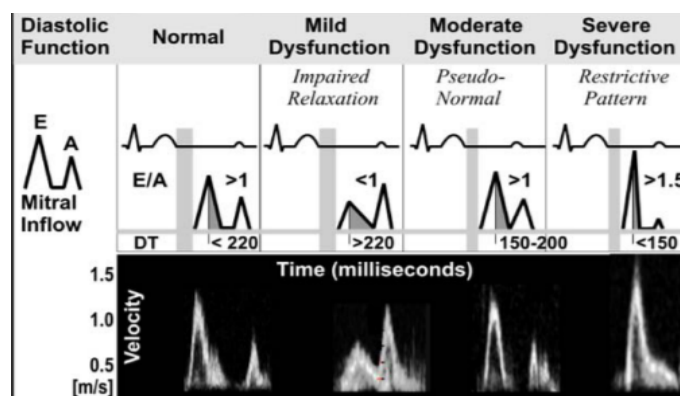


Figure 2. Flow curves in diastolic dysfunctions based on the stages [75]

**M Mode Echocardiography:** M Mode imaging method shows cardiac structure as one dimension in the left ventricle and section image is obtained only from the area on which the probe is located. In M Mode imaging, the section cut bey the ultrasound probe should be perpendicular to the posterior wall of the left ventricle. Whereas time is shown on the horizontal axis, depth of the structures is shown on the vertical axis. The longest distance between the septum and posterior wall of the left ventricle gives the diastolic diameter and the shortest distance shows the systolic diameter.

Ejection fraction is calculated by dividing the difference between the square of the diastolic diameter and square of the systolic diameter by square of diastolic diameter [78].

Since this is a one-dimensional imaging, it may give incorrect results in wall motion defects.

**Two Dimensional Echocardiography:** It is possible to view the heart on different planes with two-dimensional echocardiography which is superior over M Mode echocardiography. Images of the apical chambers are acquired, and ejection fraction, end-systolic and end-diastolic volumes can be calculated by drawing limits of the apical chamber diameters. This method is superior over M Mode echocardiography due to these features [79].

**Doppler Echocardiography:** Doppler echocardiography is a method in which the rate and direction of bloodstream can be measured. In today's clinical practice; three different methods are used as colour Doppler, pulsed wave Doppler and continuous wave Doppler. This method is often preferred in the measurement of diastolic functions since it is noninvasive [80].

It shows laminar flow, ventricular contraction, turbulence, severity and time of blood pulse as rate-time plot and pressure gradient.

## Conclusion

Acute coronary syndromes are the major thrombotic complications of atherosclerosis and include several clinical pictures occurring due to acute ischemia of cardiac myocardial cells. This clinical picture involves unstable angina pectoris, STEMI, and NSTEMI.

It is critical to establish the correct diagnosis as soon as possible in coronary heart diseases. Triage and appropriate referral of patients with acute myocardial infarction in emergency departments, a rapid diagnosis and treatment interventions will be helpful in reducing mortality and morbidity.

Echocardiography, which is a reliable, easy to use and noninvasive inexpensive method is of paramount importance in establishing the diagnosis rapidly.

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