MCG INTERPRETATION

Overview

The Multifunction Cardiogram® (MCG) is unlike any other diagnostic cardiology test. It a physiologic test, not an imaging or anatomic one. The wide array of its assessments is embodied in the term *multifunction* in its name. The MCG test acquires its data by setting up two standard electrocardiographic leads to have a "conversation" over several cycles. The "conversation" is a series of signals sent out for exploration by the first lead, followed by a response and signal returned by the second lead, and then followed by a continuous back and forth signal and response exchange between the 2 leads. This exploration is made possible by the application of several steps, namely the conversion of the time domain of the voltage display to a frequency domain; the adaptation of Legrangian mathematics to identify the physiological and physical nature of various components of the dynamic cardiovascular system; and the use of artificial intelligence (AI) in the comparison of these findings with a laboriously built data base.

The MCG Test conducts a physiologic analysis and evaluation of multiple cardiac functions, evaluating strengths and weaknesses at the cellular and subcellular levels. The MCG test is able to detect physiological damage or dysfunction at a far earlier than other modalities. Once it is understood that the MCG test is a physiologic analysis, one should not be surprised that the findings may not yet be confirmed by an echocardiogram, a stress test or CT angiogram because the MCG data uncovers the footprints of pathology long before it is clinically significant enough to be detected with standard diagnostic tools.

What Does the MCG Test Measure?

The MCG test is an analysis of the intrinsic frequencies of various components of the cardiac system. The MCG test is measuring the frequency signals of the heart to map the spatial separation of normal responses versus diseased responses from all the relevant components of the cardiac system. These components go beyond anatomical changes at the tissue level but may reflect analyses at the **cellular or subcellular levels**.

One example of this kind analysis is the identification of ionic flow gradient patterns seen in certain channelopathies that are predictive substrates of arrhythmias. Another is the analysis of ATPase activity which has relevance to wall motion, mitochondrial function, oxidative stress and overall ventricular function. The clinical relevance of this information is underscored in two elegant papers. The first paper by Gerd Hasenfuss et al, and referenced below, described the relationship between myocardial function and the expression of sarcoplasmic reticulum Ca2+ - ATPase in the failing and nonfailing heart[1]. The second paper, also referenced below, by Paul Territo et al describes the role F0/F1-ATPase in the calcium activation of myocardial oxidative phosphorylation[2].

[1] Hasenfuss, G et al 1994, "Relation Between Myocardial Function and Expression of Sarcoplasmic Reticulum Ca^{2+} - ATPase in Failing and Nonfailing Human Myocardium", Circulation Research, Vol 75, No. 3, pp. 434-442.

- [2] Territo, P et al 2000, "Ca^{2+} activation of heart mitochondrial oxidative phosphorylation: role of the F_0/F_1-ATPase", Am. J. Physiol. Cell Physiol., Vol. 278, pp. C423-435.

How is the Data Presented by the MCG Test?

The MCG Report is presented as a set of numbers and descriptors or markers for each cycle of 82 seconds of inquiry. Each set begins with an indication of the presence or absence of ischemia and number to describe the burden of ischemia or myocardial dysfunction. This followed by two charts with plus (+) and negative (-) indicators that describe a range of physiologic and pathophysiologic markers. The plus and minus signs do not represent a binary status of yes or no status for a given marker, but rather they reflect whether the amplitude of the frequency of a specific physiologic/pathologic marker has reached the threshold level of pathological relevance. As stated earlier one should not be surprised that the findings may not yet be confirmed by an echocardiogram, a stress test or CT angiogram because the MCG data provides early indications of the pathology long before it is detected with these current tools.

The Role of Electrical Interference in the Accuracy of MCG Test

The importance of maintaining electrical interference-free zone while conducting an MCG Test cannot be overstated. A good study must have a stable baseline and the absence of any electrical “noise.” The number of “cycles” or runs is also important. The nature of the MCG test is a dynamic process. In the same way that no two consecutive blood pressures are generally identical, there is generally some minor variation from one cycle to the next. In order to ascertain consistency of data, one should obtain a minimum of four (4) good cycles, but preferably five (5).

Approach to the interpretation

1. Comment on the quality of the tracings.
2. Describe the findings including the presence or absence of ischemia, as well as notation of borderline ischemia. Severity scores run from 0 to 22. While it is easy to think of these scores as ischemia levels, it is more accurate to view the score as the extent of dysfunctional burden. A score of 0 is the best available score. There is a gradual decrease in function as the scores move higher. Comment on the scores in your report. The variation between the scores should not be more than 2 points. If a score is more than 2 points from the range established by the other scores, check the quality of the tracing to determine whether or not that cycle should be accepted.
3. Local ischemia generally indicates disease in a single or confined area of one epicardial vessel.
4. Global ischemia generally indicates disease in more than one epicardial vessel, or non-obstructive disease in multiple epicardial vessels, or disease in the microcirculation. Metabolic disease (e.g. diabetes) tends to have a more diffuse pattern.
5. The absence of both local and global ischemia in the setting of relatively high severity scores should raise other concerns. It may be an indication of an infiltrative or non-ischemic cardiomyopathy. For example cardiac amyloidosis and chemotherapy induced cardiomyopathy can present in this manner. Consider the clinical correlation.
6. Comment on the expression of the pathological and physio-pathological markers. There are 24 makers, but do not be overwhelmed by the multitude of these markers Take some time to look at the meaning of each marker. An understanding of the significance of each marker is central to arriving at a working diagnosis. There are several patterns that the interpreter may use, but I find it useful to follow one that that is consistent with vascular insult and vascular response to injury. The following approach utilizes the vascular injury-vascular response evolution.
    - Look for Myocardial Damage. It usually means endothelial dysfunction, which is the primary vascular insult. It is usually positive in the presence of ongoing injury, but may resolve ahead of downstream markers.

- Cardiomyopathy (wall motion) and Remodeling give immediate insight on the timing and extent of injury. Cardiomyopathy (wall motion abnormalities) gives an earlier chronological connection to the timing and extent of the initial injury. Remodeling gives information on a more sustained (but not always) timing of the injury.
- Decreased Compliance indicates a longer term presence of the injury, and is more indicative of a compensatory state.
- Increased Compliance is a hemodynamic response. The hemodynamic response is often metabolic in nature, such as co-existent thyroid disease or anima or fever etc. When Increased Compliance is seen with Myocardial Damage, Remodeling and Diminished Compliance simultaneously, one should always suspect an acute or near term acute ischemic episode.
- Check for Hypertrophy. Correlate Hypertrophy with the presence or absence of systemic hypertension. One should also correlate this with remodeling and compliance markers.
- The Asynchrony markers are closely tied to these vascular responses. The presence of asynchrony (also referred to as a phase shift) always indicates a significant pathology. When there is Global Asynchrony, the tip of the spear points to the ventricle or region that has the greater burden. The tip of the spear in Local Asynchrony points to a blockage in the coronary artery distribution of that specified region. See below for more details.
- Decreased Ejection marker should be noted and assessed in association with other vascular markers. Report the presence of this marker as an indicator of impaired ventricular function since this will occur before reduced ejection fraction is clinically documented.
- Check for inflammation. Inflammation is described as Myocarditis. It is a marker of significant inflammation and/or oxidative stress. It may be a part of the initiating insult or may be a response to the injury. The precise role of inflammation/oxidative stress should be explored either clinically or by biomarkers. In any event it must be addressed in management of the patient.
- Rheumatic and Pulmonary markers are explained below. Most of the time they represent localization of dysfunction. Rheumatic marker usually represent left heart involvement, while pulmonary marker usually represents lung or right heart involvement. Further details are given below.
- Then we look at arrhythmias. The details below in the next section may be confusing, but try to categorize arrhythmias as atrial fibrillation, or ventricular arrhythmias or ill-defined incipient arrhythmias, or a combination. There are a host of environmental or ischemic factors to be considered. One should always try to see whether the arrhythmia is a cause of the problems revealed by the MCG study, or whether the arrhythmia is the end-result of the primary problems. For example one should be aware of the strong correlation between atrial fibrillation and obstructive sleep apnea. The pulmonary heart disease marker is a reminder to address obstructive sleep apnea in the patient's evaluation.
- Congenital heart disease marker is sometimes persistent. This may not be problematic in most situations. A patent foramen ovale (PFO) is the most common cause for this marker in a patient who has no clinical manifestations of **congenital** heart disease.
- Acute Power Failure is the neuro-hormonal marker. If present, comment on mental stress or an anxiety disorder as a contributing factor to the patient's cardiovascular impairment.

7. Identify the lettered classification (A through H, also known as the 8 Categories) for the study based on the severity scores. Use the pathological and physio-pathological markers to help identify the category if necessary. These secondary markers are crucial for identifying Category D, the situation where the patient may have natural collateralization or has had a revascularization procedure.

8. Create a narrative summary or interpretation. Use the sequence of findings, then significance of findings and finally, clinical correlation if possible.
9. A reminder: The MCG Report is a tool used for the physician to arrive at a diagnosis. It does not make a diagnosis. Only the attending physician can make the diagnosis after assessing the clinical history, the examination, the pertinent labs, and the MCG Report.
10. *Sample Report*

SAMPLE NARRATIVE INTERPRETATION

Findings:
Five (5) cycles were completed. Quality of tracings are acceptable for interpretation. Severity scores were 4.5, 4.0, 3.5, 3.0, and 4.0, with predominantly global ischemia. Pathological and Physio-pathological markers most remarkable for significant endothelial dysfunction (Myocardial damage) with associated myocardial remodeling and early indications of diminished compliance. Prominent markers for substrates of atrial fibrillation. Inflammatory markers also notable. Localization in right and left heart, but cannot rule out pulmonary contribution to right heart

Impression:
Intermediate Myocardial Dysfunction (Category E)
Pattern is consistent with microvascular disease but cannot rule out non-obstructive disease.

Suggestions:
Aggressive work up and control of diabetes, lipids, hypertension and environmental factors
Evaluate for sleep apnea, if clinically appropriate.

[redacted]

ID: N/A

MCGID: [redacted] Name: [redacted] Gender: M Age: 64

MCG Results

Ischemia Results

ID	Testing Date	ECG Tracing Quality	Local	Global	Severity
55192729	2018-12-29 18:55	good	Absent	Present	4.5
55192728	2018-12-29 18:53	good	Absent	Present	4.0
55192727	2018-12-29 18:52	good	Absent	Present	3.5
55192726	2018-12-29 18:51	good	Absent	Present	3.0
55192725	2018-12-29 18:49	good	Present	Absent	4.0

Signature: _____

[redacted]

Suggestions

Pathological Conditions:

	Cardiomyopathy	Potential Fibrillation	Atrial Fibrillation	Incipient Ventricular Fibrillation	Atrial-Ventricular Fibrillation	Incipient Arrhythmia	Ventricular arrhythmia	Myocardial Damage	Myocarditis	Pulmonary Heart Disease	Rheumatic Heart Disease	Congenital Heart Disease	Ventricular Hypertrophy
55192729	-	-	+	-	-	-	-	+	+	-	-	-	+
55192728	-	-	+	-	-	+	-	+	+	+	+	-	+
55192727	-	-	+	-	-	+	-	+	+	+	+	-	-
55192726	-	-	+	-	-	+	-	+	-	+	+	-	-
55192725	-	-	-	-	-	-	-	+	-	+	-	-	-

Physiopathological Conditions:

	Myocardial Remodeling	Decreased myocardial compliance	Increased myocardial compliance	Decreased ejection fraction	Bradycardia	Tachycardia	Acute Power Failure	Global asynchrony (II < V5)	Global asynchrony (V5 < II)	Localized Asynchrony	Local asynchrony (II < V5)	Local asynchrony (V5 < II)
55192729	-	+	-	-	-	-	-	-	-	-	-	-
55192728	+	-	-	-	-	-	-	-	-	-	-	-
55192727	+	-	-	-	-	-	-	-	-	-	-	-
55192726	+	-	-	-	-	-	-	-	-	-	-	-
55192725	+	+	-	-	-	-	-	-	-	-	-	-

Disclaimer:
This section contains comments and suggested diagnoses or conditions which require rigorous clinical validation. These suggestions and comments should be considered expert opinions and not a definitive diagnosis.

PREMIER HEART

NAME

ID: N/A

MCGID: 1990360 Name: NAME Gender: M Age: 64

MCG™ Results

Each ID represents an individual test run of 82 seconds

Ischemia Results

ID	Testing Date	ECG Tracing Quality	Oxygen Supply and Imbalance: Local (Relative regional ischemia. Consistent with large epicardial vessel involvement)	Global (Entire myocardial involved. Consistent with either multi-vessel disease or metabolic derangement)	Severity (Log scale of relative ischemia. Range from 0 to 22.)
55141234	2016-11-09 12:48	good	Absent	Present	4.5
55141233	2016-11-09 12:47	good	Absent	Present	4.0
55141232	2016-11-09 12:46	good	Absent	Present	3.5
55141231	2016-11-09 12:44	good	Absent	Present	3.0
55141230	2016-11-09 12:43	good	Present	Absent	4.0
		Good & Marginal reflect adequate baseline stability ensuring accurate results. Poor is unacceptable	A	B	C

A VASCULAR SUGGESTS SINGLE EPICARDICAL VESSEL

B METABOLIC SUGGESTS SEVERAL EPICARDIAL OR DIFFUSE SMALL VESSELS. NOT VISIBLE WITH IMAGING DEVICES

C SCORE SUGGESTS SEVERITY RANGE: 0 - 22. ZERO IS EXCELLENT

Signature: _____

Suggestions

Pathological Conditions:

Red Numbers denote location on glossary pages

1 Cardiomyopathy	2 Potential Fibrillation	3 Atrial Fibrillation	4 Incipient Ventricular Fibrillation	5 Atrial-Ventricular Fibrillation	6 Incipient Arrhythmia	7 Ventricular arrhythmia	8 Myocardial Damage ★	9 Myocarditis ★	10 Pulmonary Heart Disease R	11 Rheumatic Heart Disease L	12 Congenital Heart Disease	13 Ventricular Hypertrophy
-	-	+	-	-	-	-	+	+	-	-	-	+
-	-	+	-	-	+	-	+	+	+	+	-	+
-	-	+	-	-	+	-	+	+	+	+	-	-
-	-	+	-	-	+	-	+	-	+	+	-	-
-	-	-	-	-	-	-	+	-	+	-	-	-
Wall Motion Abnormalities	Rhythm Disturbances - clinical and/or preclinical								Potential Localization			Wall Thickness

★ **Myocarditis**
Indicates inflammation at the cellular level, a driver of almost every MI event.

★ **Myocardial Damage**
Subcellular injury

Physiopathological Conditions:

	14 Myocardial Remodeling	15 Decreased myocardial compliance	16 Increased myocardial compliance	17 Decreased ejection fraction LV	18 Bradycardia Slow	19 Tachycardia Fast	20 Acute Power Failure (autonomic function)	21 Global asynchrony (II < V5) R	Global asynchrony (V5 < II) L	Localized Asynchrony	Local asynchrony (II < V5) R	Local asynchrony (V5 < II) L
55141234	-	+	-	-	-	-	-	-	-	-	-	-
55141233	+	-	-	-	-	-	-	-	-	-	-	-
55141232	+	-	-	-	-	-	-	-	-	-	-	-
55141231	+	-	-	-	-	-	-	-	-	-	-	-
55141230	+	+	-	-	-	-	-	-	-	-	-	-
		Response to Injury			Rate Variation			1a	1b	2	3a	3b

Electro-Mechanical Synchronization

1a. Predominately right heart pathology

1b. Predominately left heart pathology

2. Localized lesion

3a. Probably lesion in the RCA distribution

3b. Probably lesion in the LAD/LCx distribution

This is not just TEMPORAL synchrony, but the degrees of deviations from normal synchronization between the signal input and the output from leads II and V5 or vice versa (local problems almost certainly shows up here). II and V5 are not corresponding at the same rate, in the same time frame, per tracings.

Disclaimer:
This section contains comments and suggested diagnoses or conditions which require rigorous clinical validation. These suggestions and comments should be considered expert opinions and not a definitive diagnosis.

FOR PHYSICIAN USE ONLY

Significance of the pathological and physio-pathological markers

1. **Cardiomyopathy**
   Disturbance: This is often a sign of structural changes in the myocardium secondary to ischemia or other disturbances. This is detected by the MCG test at the cellular level.

   Clinical significance: This may indicate ***Preclinical or cellular*** wall motion/functional disturbances (may or may not yet be seen on echocardiogram).

NOTE: General comment on arrhythmia items 2 through 7.

These markers (where noted) refer to the ***potential for the specified arrhythmia***, not the actual presence of the arrhythmia. This is derived by analyses of frequencies pertaining to ion channelopathies and repolarization gradients. Japanese researchers have identified high clinical correlation with the atrial fibrillation marker.

2. **Potential fibrillation.** Refer to atrial and ventricular fibrillation above
3. **Atrial fibrillation (Substrate of potential of atrial fibrillation).** See above.
4. **Incipient ventricular fibrillation (Substrate of potential ventricular fibrillation).** See above
5. **Atrial-Ventricular fibrillation.** A misnomer but retained for regulatory application purposes. Refer to atrial and ventricular fibrillation above.
6. **Incipient arrhythmia (Substrate of ill-defined arrhythmias).** See above
7. **Ventricular arrhythmia (Substrate of potential ventricular arrhythmia).** See above

8. **Myocardial damage**
   This is the description of an injury or a physical disruption of a barrier. This is an exquisitely sensitive marker.

   Clinical significance: The injury that initiates the evolution of plaque buildup in the artery is a dysfunction of the endothelium (a thin, one-cell thick membrane that lines the inner surface of the of the vessel). This injury can progress to involve segments of the wall of the vessel and eventually to cells of the myocardium that may lead to disruption of those cells and leakage of enzymes. The marker usually refers to endothelial dysfunction with or without secondary myocardial involvement. Endothelial dysfunction with or without myocardial injury can be caused by external sources such as blunt trauma, electric shock, hypoxemia, viral infection, heavy metal, environmentally induced oxidative stress, etc.

9. **Myocarditis**
   This term is a misnomer (but retained for regulatory application purposes). The term generally describes inflammation (and oxidative stress to a lesser extent) that may be at play as part of the mechanistic response to an injury or insult.

   Clinical significance: This is seen in metabolic heart disease; as well as inflammatory disorders such autoimmune disease and environmental toxins. It is important to address the cause of inflammation in these patients.

10. Pulmonary heart disease

This is a regional localization marker, not a marker of structural heart disease.

Clinical significance: This is an indication that a disease burden is present on the right side. This may indicate RV myocardium as localization of ischemia and/or myopathy. This may also suggest, but not diagnostic of tricuspid valve disorder or pulmonic valve disorder. Finally, this may also suggest the presence of pulmonary disease with pulmonary hypertension.

11. Rheumatic heart disease

This is a regional localization marker. This is not a marker of structural heart disease.

Clinical significance: This is an indication that disease burden is on the left side. It may indicate LV myocardium as localization of ischemia and/or myopathy. It may also suggest, but not diagnostic of mitral valve and/or aortic valve disorder. It may also suggest the presence significant systemic hypertension.

12. Congenital Heart disease

This suggests a congenital anomaly.

Clinical significance: This may indicate the presence of overt or silent disturbances such as PFO or congenital aortic stenosis.

Suggestion: The persistence of marker over multiple settings should raise the suspicion of a presence of an anomaly here.

13. Ventricular Hypertrophy

This disturbance is captured at the ***cellular level***. This may reflect disturbance in systemic pressures but may also be a derangement in structural pathology with signaling at the cellular level.

Clinical significance: This may indicate a potential thickening of wall dimensions. This may be preclinical (may or may not be seen on echocardiogram).

14. Myocardial remodeling

This is a vascular response to an injury or insult.

Clinical significance: This is the adaptive response to disturbances in flow or pressure within the heart. Generally, this indicates the presence of enough disease in either the epicardial or microvascular circulation to cause the disturbance. Untreated systemic hypertension should also be considered as a potential issue here.

15. Decreased myocardial compliance

This is a compensatory (late) response to prior or on-going injury.

Clinical significance: This is often seen in coronary artery disease (CAD) even in the absence of epicardial disease. This is also often the marker of nonobstructive CAD or microvascular disease.

16. Increased myocardial compliance

This is increased elasticity of the myocardium. May be global or regional (when seen with diminished compliance).

> Clinical significance: Early or near-term adaptive response to injury or systemic metabolic factors. Global hyperdynamic response often reflects metabolic factors like anemia, hyperthyroid disorder, etc. Regional increased compliance with coexisting diminished compliance and myocardial injury should raise suspicion of active or recent ischemic event.

17. Decreased ejection fraction

This a ***subcellular*** recognition of diminished energy production at the mitochondrial level.

> Clinical significance: This may indicate ***Pre-clinical (subcellular)*** markers of decreased energy production. This might be a sign of possible mitochondrial or thyroid dysfunction. ***This precedes echocardiographic findings***, so this may not correlate with measured ejection fraction.

18. Bradycardia

Disturbance: This is a heart rate disturbance. This indicates a slower heart rate.

> Clinical significance: This may indicate a vagal response or related to medications.

19. Tachycardia

Disturbance: This is a heart rate disturbance. This indicates a faster heart rate.

> Clinical response: This may indicate a sympathetic response or related to medications.

20. Acute power failure

Heart rate variability analyzed in the frequency domain (power spectrum) reveals information on frequency peaks and amplitudes that correlate with autonomic innervation and energy production. The term acute power failure should not be confused with structural failure of adequate cardiac output.

> Clinical significance: This can be a sign of a hypothalamus-pituitary-adrenal axis dysfunction. This can indicate a neurohormonal connection in cardiac function: the effect of mental stress on thyroid and mitochondrial function in energy production. Stress reduction would be a critical part of health management here.

21. Right heart- Left heart asynchrony

Asynchrony is a measure of the coordinated response to the mutual signals from both leads in the MCG analysis. Lead II sends out a signal. Lead V5 receives it and responds. V5 sends out a signal. Lead II receives it and responds.

The entire study is based on this continuous communication between these two leads. The signal and receipt are synchronized and is independent of any timing conduction abnormalities. If there is a lag of one side vis-a-vis the other, the tip of the spear (<) points to the more diseased side.

Global asynchrony refers to general disease burden. Local asynchrony indicates a blockage in a specific distribution of an epicardial vessel. These markers are at the very top of the hierarchy in the expression of disease severity. For emphasis, these markers do NOT represent an electrocardiographic timing issue.

Clinical significance: Localization of ischemic burden-left vs right

E.g. Global asynchrony (II<V5) Greater disease burden on right side
Global asynchrony (V5< II) Greater disease burden on left side
Local asynchrony (II< V5) Probably a lesion in the right coronary artery (RCA) distribution
Local asynchrony (V5< II) Probably a lesion in the left anterior descending/Left circumflex (LAD/LCx) distribution

MCG Categorization Guide		
H	*Extremely High Myocardial Dysfunction*	**All MCG Severity Scores ≥ 15**
G	*Very High Myocardial Dysfunction*	**All MCG Severity Scores ≥10.5 All MCG Severity Scores < 15**
F	*High Myocardial Dysfunction*	**All MCG Severity Scores ≥ 7 All MCG Severity Scores < 10.5**
E	*Intermediate Myocardial Dysfunction*	MCG Severity Scores Fluctuating All scores above 3.5 and below 7 appearing in the same session.
D	*Collateral Circulation Group*	MCG Severity Scores Fluctuating Scores above and below 3.5 appearing in the same session. (Distinguish from Category **C** by angiography evidence of collaterals)
C	*Low Myocardial Dysfunction*	**All MCG Severity Scores > 2.0** **All MCG Severity Scores ≤ 3.5**
B	*Clinically Normal*	**All MCG Severity Scores ≤ 2.0** *or* All Scores 0 with Pathological/Physiopathological Findings
A	*True Normal*	All MCG Severity Scores 0 No Pathological/Physiopathological Findings

Note: The incidental findings of structural abnormalities such as transient or persistent signs of Rheumatic Heart Disease, Pulmonary Heart, and/or Congenital maybe transient structural changes. These may be the result of his myocardial ischemia mimicking the structural anomalies. They tend to resolve once the ischemia is resolved. If these incidental MCG findings persist in the repeat test sessions, an echocardiography examination maybe warranted to define their existence and potential impact on cardiac functions. All these findings collectively contribute to the overall cardiac dysfunctions represented by the severity scores.

H	High Cardiogenic shock potential with systems instability. Apply tertiary and secondary prevention measures, stabilizing any hemodynamic instability, and optimizing medications. Referral to coronary intervention to maintain or improve status may be appropriate if the presence of local or global ischemia is detected, with post-intervention MCG Monitoring. Simultaneously manage and treat co-morbidities such as metabolic, endocrinological, hematologic, hemodynamic, infectious, neurohormonal, structural, lifestyle, environmental, or inflammatory conditions.
G	Intermediate risks for cardiogenic shock and systems instability. Apply tertiary and secondary prevention measures, stabilizing any hemodynamic instability, and optimizing medications. Referral to coronary intervention to maintain or improve status may be appropriate if the presence of local or global ischemia is detected, with post-intervention MCG monitoring. Simultaneously manage and treat co-morbidities such as metabolic, endocrinological, hematologic, hemodynamic, infectious, neurohormonal, structural, lifestyle, environmental, or inflammatory conditions.
F	Diagnose, manage, and treat the primary cause and its co-morbidities such as metabolic, endocrinological, hematologic, hemodynamic, infectious, neurohormonal, structural, lifestyle, environmental, or inflammatory conditions via primary prevention, medications or referral to inpatient care. Coronary intervention may be required to maintain or improve status if the presence of local or global myocardial ischemia is detected, with post-intervention MCG monitoring.
E	Diagnose, manage, and treat the primary cause and its co-morbidities such as metabolic, endocrinological, hematologic, hemodynamic, infectious, neurohormonal, structural, lifestyle. environmental, or inflammatory conditions. There are simultaneous fluctuating myocardial ischemia patterns related to all causes of supply and demand imbalance which can be managed via primary and secondary prevention or are treatable by medications. If the patient presents in an Emergency or Urgent Care setting he or she can be sent back home to follow up with a primary care physician within 24 hours, plus 30 to 60 day MCG event monitoring following chest pain due to higher propensity to sudden cardiac deaths in this group, especially those suffering from preoperative myocardial infarctions.
D	Patients in this group have recovered from high levels of chronic myocardial ischemia. Angiographic evidence of collateral formation is required to place the patient in this group for long term monitoring. Knowing that further coronary intervention may exacerbate the problems (such as phenomena like "coronary steal") check for the presence of ventricular arrhythmia, asynchronization, low ejection fraction, acute power loss, ventricular remodeling, and hypertrophy. Other abnormal expressions can be treated per generally applicable, individualized physician suggested options.
C	Diagnose, manage, and treat the primary cause and its co-morbidities such as metabolic, endocrinological, hematologic, hemodynamic, infectious, neurohormonal, structural, lifestyle, environmental, or inflammatory conditions. If higher disease burden represented by either local or global myocardial ischemia is found referral to coronary intervention may be appropriate. If admitted to an ER or Urgent Care facility the patient can be to a primary care physician for follow up. Follow up frequency will be determined by the physician on a case by case basis, considering patient history and other risk factors.
B	Clinically normal with minimal early signs of CVD or other mixed automatically detected signs of pathologies. If admitted to an ER or Urgent Care facility the patient can be safely discharged home with physician supervised observation and annual follow up unless symptomatic.
A	No MCG-detectable abnormalities. If admitted to an ER or Urgent Care facility the patient can be safely discharged home for physician follow up and monitoring using MCG.