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Role of selvester scores in patients with acute coronary syndrome

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ABSTRACT

Acute Coronary Syndrome (ACS) is a major cardiovascular problem because it increases mortality and morbidity. ACS is the acute manifestation of coronary arteries atheromatous plaque torn or ruptured. ECG had been developed to enhance the clinical benefits. Selvester score is one of the assessment method in the ECG that aims to detect and determine the location of damage due to myocardial infarction, judging from the complex of QRS. In some studies it has been reported that Selvester score correlated with the extent of damage after myocardial infarction. By using a specific scoring criteria, with the total number of points earned, Selvester scores are considered a non-invasive tool to assess myocardial damage that is easy to use and provides precise results by using ECG especially in areas with limited diagnostic tools.

Key words: Acute, Coronary, Selvester
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INTRODUCTION

Acute Coronary Syndrome (ACS) is a major cardiovascular problem because it increases mortality and morbidity. Every year in the United States it is estimated that there are approximately 735,000 people with acute coronary syndrome; about 525,000 people first received ACS and about 210,000 have had ACS before.[1,2]

In Indonesia, the World Health Organization (WHO) on Non Communicable Diseases (NCD) Country Profiles, 2014, found that cardiovascular disease is a leading cause of death, amounting to 37% of total mortality. In research conducted at the department of Prof. Dr. R. D. Kandou Manado in North Sulawesi, it was found that there were 55 cases of acute coronary syndrome in 2006; 104 cases in 2007; 166 cases in 2008; 251 cases in 2009; and 354 cases in 2010.[3,5]

Acute Coronary Syndrome (ACS) is a term used to describe the symptoms caused by acute myocardial ischemia. ACS which causes necrosis of the myocardium is called a myocardial infarction. ACS is divided into three categories: the first of myocardial infarction with ST-segment elevation or ST segment elevation myocardial infarction (STEMI), which is both myocardial infarction without ST-segment elevation or non-ST segment elevation myocardial infarction (NSTEMI) and a third unstable angina pectoris or unstable angina pectoris (UAP).[1,6]

One way to determine the extent and location of damage to the myocardium caused by infarction is recording an electrocardiogram (ECG) with 12 leads performed within 10 minutes of the patient's arrival in the emergency room as recommended by the guidelines of the European Society of Cardiology (ESC) NSTEMI 2015. ECG is an examination that does not require a huge costs and is easy to use and works fast. Some of the modalities of non-invasive investigations, such as echocardiography, magnetic resonance imaging (MRI), angiography radionucleid, Single Photon Emission Computed Tomography (SPECT), or Positron Emission Tomography (PET), are not cheap and need a long vetting process. One option that is an affordable non-invasive examination is to assess the extent and location of infarction, that is, Selvester Score, assessing the QRS wave of the ECG, so as to make it easier to assess the location and myocardial damage in patients with ACS. Any points earned on the Selvester score indicate damage amounting to 3% in miokard.[1,7-11]

In this literature review will discuss the role of the Selvester score in determining the extent and location of damage caused by myocardial infarction in patients with acute coronary syndrome.

ACUTE CORONARY SYNDROME

ACS is the acute manifestation of coronary arteries atheromatous plaque torn or ruptured. It is associated with changes in plaque composition and thinning of the fibrous cap covering the plaque. This event will be followed by a process of platelet aggregation and activation of the coagulation pathway, thus forming a platelet-rich thrombus (white thrombus). This thrombus will clog coronary arteries, either totally or partially or become micro emboli that can clog the distal coronary arteries. Besides,

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the release of vasoactive substances that cause vasoconstriction aggravates interruption of coronary blood flow. Reduction in coronary blood flow causes myocardial ischemia. Oxygen supply stopped for over 20 minutes causes myocardial necrosis (myocardial infarction). Myocardial infarction is not always caused by total occlusion of the coronary arteries. Subtotal obstruction accompanied by dynamic vasoconstriction can lead to ischemia and necrosis of heart muscle tissue (myocardium). As a result of ischemia, it can also cause myocardial contractility due process and stunning hibernating (after ischemia missing), dysrhythmia, and ventricular remodeling (changes in shape, size, and ventricular function. Most patients do not experience ACS plaque rupture as described above. They experience ACS as a dynamic obstruction due to local spasm of an epicardium coronary artery (Prinzmetal angina). Narrowing of the coronary arteries, without any spasm or thrombus, may also be caused by the progression of plaque or restenosis after Percutaneous Coronary Intervention (IKP). Some extrinsic factors, such as fever, anemia, thyrotoxicosis, hypotension, and tachycardia, can trigger the occurrence of ACS in patients who already have atherosclerosis plaque.[1,12-16]

Table 1 ***Simplified*** **Selvester Scores**

Leads	Q Duration (msec)		R Duration (msec)	Ratio	Point	Max
I	≥ 30				1	2
				R/Q ≤ 1	1	
II	≥ 40				2	2
	≥ 30				1	
aVL	≥ 30			R/Q ≤ 1	1	2
					1	
aVF	≥ 50				3	
	≥ 40				2	
	≥ 30				1	5
				R/Q ≤ 1	2	
				R/Q ≤ 2	1	
V_1	Q				1	
			≥ 50		2	4
			≥ 40		1	
				R/S ≥ 1	1	
V_2	Q	Or	≤ 20		1	
			≥ 60		2	4
			≥ 50		1	
				R/S ≥ 1.5	1	
V_3	Q	Or	≤ 30		1	1
V_4	≥ 20				1	
				R/Q or R/S ≤ 0.5	2	3
				R/Q or R/S ≤ 0.1	1	
V_5	≥ 30				1	
				R/Q or R/S ≤ 1	2	3
				R/Q or R/S ≤ 3	1	
V_6	≥ 30				1	
				R/Q or R/S ≤ 1	2	3
				R/Q or R/S ≤ 3	1	

References: Wagner GS, Freye CJ, Palmeri ST et al. Evaluation of a QRS scoring system for estimating myocardial infarct size. I. Specificity and observer agreement. Circulation. 1982;65:342–7.

Based on history, physical examination, ECG, and cardiac markers examination ACS is divided into three categories: STEMI, NSTEMI, and UAP. STEMI is an indicator of the incidence of total occlusion of coronary arteries. This situation requires revascularization to restore blood flow and myocardial reperfusion as soon as possible, medically using fibrinolytic agent or mechanically by primary percutaneous coronary intervention. STEMI diagnosis is made when there is a complaint of acute angina pectoris with persistent ST segment elevation in two adjacent leads. Initiation revascularization procedure requires waiting for results from an increase in cardiac markers. Diagnosis NSTEMI and UAP can be enforced if there are complaints of angina pectoris without ST segment elevation acutely persistent in two adjacent leads. ECG recordings can be either ST segment depression, T wave inversion, flat T waves, pseudo-normalization of T wave, or even without any changes. While UAP and NSTEMI is distinguished by the incidence of myocardial infarction, they were also marked by an increase in cardiac markers. Cardiac markers that are commonly used are Troponin I/T or creatinine kinase-MB (CK-MB). When the results of the cardiac markers increased significantly, the diagnosis becomes NSTEMI. At UAP, cardiac markers do not increase significantly. At ACS, the threshold value for the increase in CK-MB is abnormal as some units exceeded the upper-normal value (upper limits of normal, ULN). As stated in the ESC guidelines NSTEMI 2015-B class I recommend recording 12 leads of ECG within 10 minutes after the patient's arrival in the emergency room. If the initial ECG showed no abnormalities (normal) or non-diagnostic while angina is still ongoing, the examination is repeated 10 to 20 minutes later. If a repeat ECG still showed a non-diagnostic picture while complaints of angina it is very suggestive of ACS, and the patient should be monitored for 12 to 24 hours. ECG was repeated every 6 hours and at every recurrent instance of angina. All patients with complaints of chest pain or other complaints that may lead to ischemia should undergo an ECG with 12 leads as soon as possible when they arrive at the emergency room. In addition, V3R and V4R, and V7–V9 leads should be recorded in all patients with ECG changes that lead to ischemia of

Table 2 Selvester scores with 54 criteria and 32 points

Lead		Criteria	Point	Max
I		Q ≥ 30 ms	1	
		R/Q ≤ 1	1	2
		R ≤ 0.2 mV	1	
II		Q ≥ 40 ms	2	2
		Q ≥ 30 ms	1	
aVL		Q ≥ 30 ms	1	2
		R/Q ≤ 1	1	
aVF		Q ≥ 50 ms	3	
		Q ≥ 40 ms	2	
		Q ≥ 30 ms	1	5
		R/Q ≤ 1	2	
		R/Q ≤ 2	1	
V_1	Anterior	Q	1	2
		Q or S ≥ 1.8 mV	1	
	Posterior	R/S ≥ 1	1	
		R ≥ 50 ms	2	
		R ≥ 1 mV	2	4
		R ≥ 40 ms	1	
		R ≥ 0.6 mV	1	
		Q and S ≤ 0.3 mV	1	
V_2	Anterior	Q	1	
		R ≤ 10 ms	1	1
		R ≤ 0.1 mV	1	
		R≤ R V1 mV	1	
	Posterior	R/S ≥ 1.5	1	
		R ≥ 60 ms	2	
		R ≥ 2 mV	2	4
		R ≥ 50 ms	1	
		R ≥ 1.5 mV	1	
		Q and S ≤ 0.4 mV	1	
V_3		Q	1	
		R ≤ 20 ms	1	1
		R ≤ 0.2 mV	1	
V_4		Q ≥ 20 ms	1	
		R/S ≤ 0.5	2	
		R/Q ≤ 0.5	2	
		R/S ≤ 1	1	3
		R/Q ≤ 1	1	
		R ≤ 0.7 mV	1	
		Notch R	1	
V_5		Q ≥ 30 ms	1	
		R/S ≤ 1	2	
		R/Q ≤ 1	2	
		R/S ≤ 2	1	3
		R/Q ≤ 2	1	
		R ≤ 0.7 mV	1	
		Notch R	1	
V_6		Q ≥ 30 ms	1	
		R/S ≤ 1	2	
		R/Q ≤ 1	2	
		R/S ≤ 2	1	3
		R/Q ≤ 2	1	
		R ≤ 0.7 mV	1	
		Notch R	1	

References: Selvester RH, Wagner S, Hindman. The Selvester QRS scoring system for estimating myocardial infarct size. Arch Intern Med. 1985;145:1877–81.

the inferior and posterior. Wherever possible, the ECG recording should be made within 10 minutes after patient's arrival in the emergency room. ECG should be repeated for every incidence of angina complaint thereafter.[1,15-18]

Role of Electrocardiogram in ACS

In 1912, Einthoven described an equilateral triangle formed by I, II, and III leads, which was then known as the Einthoven triangle. Electrocardiogram in cases of acute myocardial infarction was first published by Harold Pardee of New York in 1920 and was described as a high T starting from point decline in R wave, hereinafter known as ST-segment elevation. Clinical use of precordial leads was initiated by Charles Wolferth and Francis Wood in 1932 when diagnosing coronary occlusion. Furthermore, Emanuel Goldberger in 1942 added unipolar leads aVR, aVL, and aVF and formed the 12-lead ECG. In 1954, the American Heart Association (AHA) set the standard ECG recording tool with 12 leads as used now. ECG had been developed to enhance the clinical benefits. In 2005, Clemmensen et al. reported the success of reducing the time between the onset of chest pain and primary angioplasty with the use of wireless electrocardiogram of the patient from the ambulance to a handheld Personal Digital Assistant (PDA) for heart specialists at the hospital. Thus, patients in critical situation can be handled as soon as possible.[10,19-23,42-43]

ECG is recorded on paper at a speed of 25 mm/sec. Each small block of ECG paper has an area of 1 mm^2. The horizontal axis measures time, distance 1, small box, which the ECG paper translates into 0.04 seconds (40 ms) and one large box consists of 5 small boxes of 0.2 seconds; a vertical axis measures the voltage where the distance between the small squares is 0.1 mV and between large boxes is 0.5 mV.[24-25]

Distribution changes of ECG provide some information about myocardial area involved, that ECG changes on V2–V6 leads show a decrease in anterior ischemia. Extensive infarction in this area is associated with a high risk of heart failure, arrhythmias, mechanical complications, and premature death. Changes in I, aVL, V5, and V6 leads shows lateral ischemia; infarction in this area has a good prognosis compared with large anterior infarctions. Changes in II, III, and aVF leads showed inferior ischemia. High R wave in V1–V3 is associated with depression of the ST segment that can also indicate ischemia of posterior.[26]

Selvester Scores in Acute Coronary Syndrome

Selvester score is one of the assessment methods used with the ECG that aims to detect the

Table 3 Selvester scores criteria in ECG with conduction disturbance

	RBBB		LAFB		LAFB + RBBB		LVH		
Lead	**Criteria**	**Point**	**Criteria**	**Point**	**Criteria**	**Point**	**Criteria**	**Point**	**Max**
I	Q ≥ 30 ms	1	Q ≥ 30 ms	1	Q ≥ 30 ms	1	Q ≥ 30 ms	1	2
	R/Q ≤ 1 R ≤ 0.2 mV	1	R/Q ≤ 1 R ≤ 0.2 mV	1	R/Q ≤ 1 R ≤ 0.2 mV	1	R/Q ≤ 1 R ≤ 0.2 mV	1	
II	Q ≥ 40 ms	2	Q ≥ 40 ms	2	Q ≥ 40 ms	2	Q ≥ 40 ms	2	2
	Q ≥ 30 ms	1	Q ≥ 30 ms	1	Q ≥ 30 ms	1	Q ≥ 30 ms	1	
aVL	Q ≥ 30 ms	1	Q ≥ 40 ms	1	Q ≥ 40 ms	1	Q ≥ 40 ms	1	2
	R/Q ≤ 1	1	R/Q ≤ 1	1	R/Q ≤ 1	1	R/Q ≤ 1	1	
aVF	Q ≥ 50 ms	3	Q ≥ 50 ms	3	Q ≥ 50 ms	3	Q ≥ 60 ms	3	5
	Q ≥ 40 ms	2	Q ≥ 40 ms	2	Q ≥ 40 ms	2	Q ≥ 50 ms	2	
	Q ≥ 30 ms	1	Q ≥ 30 ms	1	Q ≥ 30 ms	1	Q ≥ 40 ms	1	
	R/Q ≤ 1	2	R/Q ≤ 1	2	R/Q ≤ 1	2	R/Q ≤ 1	2	
	R/Q ≤ 2	1	R/Q ≤ 2	1	R/Q ≤ 2	1	R/Q ≤ 2	1	
V_1 anterior	Q ≥ 50 ms	2	QR	1	Q ≥ 50 ms	2	QR	1	(2)
	Q	1			Q	1	.04 R notch		1
	R ≤ 20 ms								
			R/S ≥ 1	1			R/S ≥ 1	1	
V_1 posterior (kecuali **)	Init R ≥ 60 ms Init R ≥ 1.5 mV	2	Init R ≥ 50 ms Init R ≥ 1 mV	2	Init R ≥ 60 ms Init R ≥ 1.5 mV	2	Init R ≥ 50 ms Init R ≥ 1 mV	2	4
	Init R ≥ 50 ms Init R ≥ 1.0 mV	1	Init R ≥ 40 ms Init R ≥ 0.7 mV	1	Init R ≥ 50 ms Init R ≥ 1.0 mV	1	Init R ≥ 40 ms Init R ≥ 0.7 mV	1	
			Q&S ≤ 0.2 mV	1			Q&S ≤ 0.2 mV	1	
V_2 anterior	Q≥50 ms	2			Q≥50 ms	2	QR		
	Q	1	QR	1	Q	1	(or QS*)	1	(2)
	R≤ 10 ms		R≤ 10 ms		R≤ 10 ms		.04 S notch		1
	R≤ 0.1 mV		R≤ 0.1 mV		R≤ 0.1 mV				
			R/S ≥ 1.5	1			R/S ≥ 1.5	1	
V_2 posterior (kecuali**)	Init R ≥ 70 ms Init R ≥ 2.5 mV	2	R ≥ 60 ms R ≥ 2 mV	2	Init R ≥ 70 ms Init R ≥ 2.5 mV	2	R ≥ 60 ms R ≥ 2 mV	2	4
	Init R ≥ 50 ms Init R ≥ 2.0 mV	1	R ≥ 50 ms R ≥ 1.5 mV	1	Init R ≥ 50 ms Init R ≥ 2.0 mV	1	R ≥ 50 ms R ≥ 1.5 mV	1	
			Q&S ≤ 0.3 mV	1			Q&S ≤ 0.3 mV	1	
V_3	Q ≥ 30 ms R≤ 10 ms	2	Q ≥ 30 ms R≤ 10 ms	2	Q ≥ 30 ms R≤ 10 ms	2	QR+Q ≥ 30 ms	2	2
	Q ≥ 20 ms R≤ 20 ms	1	Q ≥ 20 ms R≤ 20 ms	1	Q ≥ 20 ms R≤ 20 ms	1	.04 S notch QR (or QS*)	1	
	Q ≥ 20 ms	1	Q ≥ 20 ms	1	Q ≥ 20 ms	1	Q ≥ 20 ms	1	
V_4	R/Q ≤ 0.5 R/S ≤ 0.5	2	R/Q ≤ 0.5 R/S ≤ 0.5	2	R/Q ≤ 0.5 R/S ≤ 0.5	2	R/Q ≤ 0.5 R/S ≤ 0.5	2	3
	R/Q ≤ 1 R/S ≤ 1 R ≤ 0.5 mV .04 R notch	1	R/Q ≤ 1 R/S ≤ 1 R ≤ 0.5 mV .04 R notch	1	R/Q ≤ 1 R/S ≤ 1 R ≤ 0.5 mV .04 R notch	1	R/Q ≤ 1 R/S ≤ 1 R ≤ 0.5 mV .04 R notch	1	
	Q ≥ 30 ms	1	Q ≥ 30 ms	1	Q ≥ 30 ms	1	Q ≥ 30 ms	1	
V_5	R/Q ≤ 1 R/S ≤ 1	2	R/Q ≤ 1 R/S ≤ 1	2	R/Q ≤ 1 R/S ≤ 1	2	R/Q ≤ 1 R/S ≤ 1	2	3
	R/Q ≤ 2 R/S ≤ 2 R ≤ 0.6 mV .04 R notch	1	R/Q ≤ 2 R/S ≤ 1.5 R ≤ 0.6 mV .04 R notch	1	R/Q ≤ 2 R/S ≤ 1.5 R ≤ 0.6 mV .04 R notch	1	R/Q ≤ 2 R/S ≤ 2 R ≤ 0.6 mV .04 R notch	1	

Table 3 Continuous....

Lead	RBBB		LAFB		LAFB + RBBB		LVH		Max
	Criteria	Point	Criteria	Point	Criteria	Point	Criteria	Point	
V_6	Q ≥ 30 ms	1	Q ≥ 30 ms	1	Q ≥ 30 ms	1	Q ≥ 30 ms	1	3
	R/Q ≤ 1 R/S ≤ 1	2	R/Q ≤ 1 R/S ≤ 1	2	R/Q ≤ 1 R/S ≤ 1	2	R/Q ≤ 1 R/S ≤ 1	2	
	R/Q ≤ 3 R/S ≤ 3	1	R/Q ≤ 3 R/S ≤ 2	1	R/Q ≤ 3 R/S ≤ 2	1	R/Q ≤ 3 R/S ≤ 3	1	
	R ≤ 0.6 mV .04 R notch		R ≤ 0.6 mV .04 R notch		R ≤ 0.6 mV .04 R notch		R ≤ 0.6 mV .04 R notch		

*LVH criteria if there is ≥ 4 point anterior or apical (beside QS), better count QS in V1-V3; **Posterolateral criteria is not used if there is sign of right atrium overload (refer to RVH) if P wave amplitude was positive in V1 or V2 ≥ 0.1 mV or aVF P ≥ 0.175 mV.

References: Strauss DG, Selvester R. The QRS complex—a biomarker that "images" the heart: QRS scores to quantify myocardial scar in the presence of normal and abnormal ventricular conduction. Journal of Electrocardiology. 2009;42:85–96.

Table 4 Criteria of Selvester score in ECG without conduction disturbance

Lead	Others conduction disturbance		Max
	Criteria	Point	
I	Q ≥ 30 ms	1	2
	R/Q ≤ 1 R ≤ 0.2 mV	1	
II	Q ≥ 40 ms	2	2
	Q ≥ 30 ms	1	
aVL	Q ≥ 30 ms	1	2
	R/Q ≤ 1	1	
aVF	Q ≥ 50 ms	3	5
	Q ≥ 40 ms	2	
	Q ≥ 30 ms	1	
V_1 anterior	R/Q ≤ 1	2	1
	R/Q ≤ 2	1	
	Q	1	
	R/S ≥ 1	1	
V_1 posterior	Init R ≥ 50 ms Init R ≥ 1 mV	2	4
	Init R ≥ 40 ms Init R ≥ 0.7 mV	1	
V_2 anterior	Q&S ≤ 0.2 mV	1	1
	Q R≤ 10 ms R≤ 0.1 mV	1	
V_2 posterior	R/S ≥ 1.5	1	4
	R ≥ 60 ms R ≥ 2 mV	2	
	R ≥ 50 ms R ≥ 1.5 mV	1	

Table 4 Continuous....

Lead	Others conduction disturbance		Max
	Criteria	Point	
V_3	Q&S ≤ 0.3 mV	1	2
	Q ≥ 30 ms R≤ 10 ms	2	
	Q ≥ 20 ms R≤ 20 ms	1	
	Q ≥ 20 ms	1	
V_4	R/Q ≤ 0.5 R/S ≤ 0.5	2	3
	R/Q ≤ 1 R/S ≤ 1	1	
	R ≤ 0.5 mV .04 R notch		
V_5	Q ≥ 30 ms	1	3
	R/Q ≤ 1 R/S ≤ 1	2	
	R/Q ≤ 2 R/S ≤ 2	1	
	R ≤ 0.6 mV .04 R notch		
V_6	Q ≥ 30 ms	1	3
	R/Q ≤ 1 R/S ≤ 1	2	
	R/Q ≤ 3 R/S ≤ 3	1	
	R ≤ 0.6 mV .04 R notch		

References: Strauss D.G, Selvester R, The QRS complex—a biomarker that "images" the heart: QRS scores to quantify myocardial scar in the presence of normal and abnormal ventricular conduction. Journal of Electrocardiology. 2009;42:85–96.

Table 5 Criteria of Selvester score in ECG with LBBB conduction disturbance

Lead	LBBB		Max
	Criteria	Point	
	Anterosuperior side		
I	R/Q ≤ 1.5 R/S ≤ 1.5	1	1
aVL	Q ≥ 50 ms Q ≥ 40 ms	2 1	4*
	R/S ≤ 0.5 R/Q ≤ 0.5	2	
	R/S ≤ 1 R/Q ≤ 1	1	
	Inferior side		
II	Q ≥ 40 ms Q ≥ 30 ms R/S ≤ 0.5 R/Q ≤ 0.5	2 1 1	3
aVF	Q ≥ 50 ms Q ≥ 40 ms R/S ≤ 0.5 R/Q ≤ 0.5	2 1 1	3
	Anteroseptal side**		
V_1	NCHINIT 40	1	3**
	R ≥ 30 ms R ≥ 0.3 mV	2	
	R ≥ 20 ms R ≥ 0.2 mV	1	
V_2	NCHINIT 40	1	3**
	R ≥ 30 ms R ≥ 0.4 mV	2	
	R ≥ 20 ms R ≥ 0.3 mV	1	

Table 5 Continuous....

Lead	LBBB		Max
	Criteria	Point	
	Posterolateral side		
V_1	S/S' ≥ 2 S/S' ≥ 1.5 S/S' ≥ 1.25	3 2 1	3
V_2	S/S' ≥ 2.5 S/S' ≥ 2 S/S' ≥ 1.5	3 2 1	3
	4 apical segment		
I	Q R ≤ 0.2 mV	1	2
	R/Q ≤ 1 R/S ≤ 1	1	
V_5	Q R/R' ≥ 2 R/R' ≥ 1 R/S ≤ 2	1 2 1	4
	R ≤ 0.5 mV	1	
	Q ≥ 20 ms	1	
V_6	R/R' ≥ 2 R/R' ≥ 1 R/S ≤ 2	2 1	4
	R ≤ 0.6 mV	1	
Total Point			33

*If there is a figure of right atrium overload (RVH) (positive P wave amplitude in V_1 or V_2 ≥ 0.1 mV or aVF ≥ 0.175 mV) release Criteria Point from anteroseptal; **if there is axis deviation to the right (mean axis QRS ≥ 90O) release Criteria Point from anterosuperior.

References: Strauss DG, Selvester R, The QRS complex—a biomarker that "images" the heart: QRS scores to quantify myocardial scar in the presence of normal and abnormal ventricular conduction. Journal of Electrocardiology. 2009;42:85–96.

location of damage to myocardial judging from the complex of QRS.[8] Selvester scores were introduced by Selvester et al. in 1972. The Selvester scores method has been used to assess the location of the area of myocardial damage of the heart, where the ECG criteria have accumulated into a point scoring system. From 10 leads of ECG (I, II, aVL, aVF, V1–V6 leads) with 57 criteria with the maximum points of 32, each point earned a portrait of 3% of the left ventricle muscle. In 1982 Wagner et al. simplified Selvester score to 37 criteria that assess the duration and QRS complexes where the maximum value of the points is 29, and this can be seen in Table 1. In 1985 Selvester et al. evaluated the criteria of Selvester scores to assess posterior infarction; the results of the criteria are presented in Table 2.[8,27-36,42]

In 2008, Andresen and Selvester reevaluates the Selvester scores by adding the criteria for age and sex. In 2009 Strauss and Selvester renewed the score of Selvester on ECG with conduction disturbances such as Left/Right Bundle Branch Block (LBBB/RBBB), Left Anterior Fascicular Block (LAFB), Left ventricle hypertrophy (LVH), and a combination of LAFB with RBBB can be seen in Tables 3–5.[8]

If more criteria for Selvester scores were found in the patient's ECG, it means more damage to the myocardium. Selvester also predicts areas of myocardial damage through specific scoring criteria. Selvester scores can also be used as a non-invasive tool to quickly and easily assess the location and extent of myocardial damage.[37]

Role of Selvester Scores in Acute Coronary Syndrome

In some studies it was reported that Selvester scores correlated with the extent of damage after myocardial infarction. Some recent studies discuss the

usefulness of the Selvester scores in determining left ventricular ejection fraction after occurrence of ACS and its relationship with ejection fraction results obtained from the ECG examination.[41]

In Palmeri et al.'s (1982) study it was mentioned that a Selvester score of more than 3 points was associated with infarction that causes a decrease in left-ventricular ejection fraction. Wagner et al. also mentioned the clinical utility of Selvester score on the ECG to assess left-ventricular function in patients with myocardial infarction. Ashkenazi et al. evaluated left-ventricular ejection fraction by the number of R waves of the QRS complex in 6 precordial leads obtained on echocardiography. Samuel et al. found a significant correlation between left-ventricular ejection fraction obtained by QRS score and a left-ventricular ejection fraction obtained in the ECG examination.[8,40-41]

Bergovec et al. (1993) studied 71 patients with infarction-rated ejection fraction of the left-ventricle using radionuclide ventriculography and comparable assessment of the QRS score; therefore, it was concluded that Selvester scores are an easy method to assess the ejection fraction of the left ventricle. According to Uyarel Hussein et al. (2006), QRS score is an independent predictor factor return of ST segment in patients with STEMI who underwent primary Percutaneous Coronary Intra (IKPP) and 30-day mortality. Walther's (2007) study of patients with acute myocardial infarction who had thrombolytic and non-thrombolytic showed the same results significantly by using ratings of Magnetic Resonance Imaging (MRI) of the heart which was in contrast to the evaluations made using Selvester scores. Shaimaa et al. (2015) used Selvester scores where judgment was easily done at affordable prices and the results were compared with SPECT to assess the extensive damage caused by infarction in 30 patients with STEMI who qualified for IKPP. Watanabe et al. (2015) reported that Selvester scores can be used to assess microvascular obstruction and concluded that Selvester scores are a good parameter to assess microvascular obstruction.[46-48]

CONCLUSION

Acute coronary syndrome is an acute manifestation from atheromatous plaque rupture, divided into STEMI, NSTEMI, and UAP, which is a major problem because it increases cardiovascular mortality and morbidity. AHA had set the 12-lead ECG as the standard, which is currently used in the field. ESC recommends that 12-lead ECG recording be carried out within 10 minutes after first medical contact. By using a specific scoring criteria, with the total number of points earned, Selvester scores are considered a non-invasive tool that assess myocardial damage and is easy to use and yields precise results especially when using ECG in areas with limited diagnostic tools.

REFERENCES

1. Irmalita, DKK, editor, 2015. Pedoman Tatalaksana Sindrom Koroner Akut. Ed ke-3. Indonesia: PERKI.
2. Mozaffarian D, Benjamin EJ, Go AS, et al. Heart disease and stroke statistics—2015 update: a report from the American Heart Association. Circulation. 2015; 131(4):e29-322.
3. WHO, 2014. Noncommunicable diseases country profiles 2014. WHO. 92.
4. Gaziano TA, Bitton A, Anand S, Abrahams-Gessel S, Murphy A. Growing epidemic of coronary heart disease in low- and middle-income countries. Curr Probl Cardiol. 2010;35:72-115.
5. Torry Srv., Panda AL, Ongkowijaya J. Gambaran Faktor Risiko Penderita Sindrom Koroner Akut. E-clinic 2014;2:1-8.
6. Antman EM, Braundwald E. Acute myocardial infarction. In: Braundwald E (ed). Heart disease: a text book of cardiovascular medicine, 5th ed. WB Saunders Company, Philadelphia; 1997, pp. 1184–288.
7. Selvester R, Wagner G, Hindman N, et al. Electrocardiographic for clinician the Selvester QRS score system for estimating the size of myocardial infarction: the development and application of system. Arch Intern Med. 1995;145:1877–81.
8. Strauss DG, Selvester R. The QRS complex—a biomarker that images" the heart: QRS scores to quantify myocardial scar in the presence of normal and abnormal ventricular conduction. Journal of Electrocardiology. 2009;42:85–96.
9. Mostafa SA, Alian MA, Mansour HA, et al. Electrocardiographic predictor of final infarct size by Selvester QRS score in correlation to rest Tc99M Sestamibi SPECT after Primary PCI. Med J Cairo Univ. 2015;83(1):499–506.
10. Jahuddin NHR, Suaib AH. 2015. Fokus Utama Pembaharuan Pedoman American Heart Association 2015 untuk CPR and ECC. American Heart Association.
11. Foley TA, Mankad SV, Anavekar NS, et al. Measuring left ventricular ejection fraction techniquesand potential pitfalls. European Cardiology Journals. 2012;8(2):108–14.
12. Libby P. Mechanisms of acute coronary syndromes and their implications for therapy. N Engl J Med 2013;368:2004–13.
13. Badimon L, Padro T, Vilahur G. Atherosclerosis, platelets and thrombosis in acute ischaemic heart disease. Eur Heart J Acute Cardiovasc Care 2012;1:60–74.
14. Davi G, Patrono C. Platelet activation and atherothrombosis. N Engl J Med. 2007;357:2482–94.
15. Roffi M, Patrono C, Collet JP, et al. 2015 ESC guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation—web addenda. European Heart Journal. 2016:2–14.
16. Roffi M, Patrono C, Collet JP, et al. 2015 ESC guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. European Heart Journal. 2016;37:267–315.
17. Thygesen K, Alpert JS, White HD. Universal definition of myocardial infarction. European Heart Journal. 2007; 28:2525–38.
18. Steg G, James SK, Atar D, ESC guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. European Heart Journal. 2012;33:2569–619.
19. Al Ghatrif M, Lindsay J. A brief review: history to understand fundamentals of electrocardiography. Journal of Community Hospital Internal Medicine Perspectives. 2012;2:143–83.

20. Titomir LI. The remote past and near future of electrocardiology: view-point of a biomedical engineer. Bratisl Lek Listy. 2000;101(5):272–79.
21. Pardee HEB. An electrocardiographic sign of coronary artery obstruction. Arch Int Med. 1920;26:244–57.
22. Wolferth CC, Wood FC. The electrocardiographic diagnosis of coronary occlusion by the use of chest leads. Am J Med Sci.1932;183:30–35.
23. Clemmensen P, Sejersten M, Sillesen M, dkk. Diversion of ST-elevation myocardial infarction patients for primary angioplasty based on wireless prehospital 12-lead electrocardiographic transmission directly to the cardiologist's handheld computer: a progress report. J Electrocardiol. 2005;38(4):194–8.
24. Cooper J. Electrocardiography 100 years ago. Origins, pioneers, and contributors. N Engl J Med. 1986;315(7):461–4.
25. Zainul Abedin and Roberth Corner, 2008. ECG interpretation the self-assesment approach, 2nd edition. Blackwell.
26. Rampengan SH. 2016. Hubungan Helicobacter Pylori dengan Fraksi Ejeksi Ventrikel Kiri pada Pasien Infark Miokard Akut. Unsrat.
27. Durrer D, van Dam RT, Freud GE, Janse MJ, Meijler FL, Arzbaecher RC. Total excitation of the isolated human heart. Circulation. 1970;41:899–912.
28. Moss AJ, Hall WJ, Cannom DS, Klein H, Brown MW, et al. Cardiac-resynchronization therapy for the prevention of heart failure events. N Engl J Med. 2009;361:1329–38.
29. Anderson WD, Wagner NB, Lee KL, et al. Evaluation of a QRS scoring system for estimating myocardial infarct size.6. Identification of screening criteria for non-acute myocardial infarcts. American Journal of Cardiology. 1988;61:729–33.
30. Freye CJ, Wagner NB, Howe CM, et al. Evaluation of a qrs scoring system for estimating myocardial infarct size.8. Specificity in a control-group with left-ventricular hypertrophy and proposal of a new scoring system for use with this confounding factor. Journal of Electrocardiology. 1992;25:19–23.
31. Hindman NB, Schocken DD, Widmann M, et al. Evaluation of a QRS scoring system for estimating myocardial infarct size.5. Specificity and method of application of the complete system. American Journal of Cardiology. 1985;55:1485–90.
32. Howe CM, Freye CJ, Wagner NB, et al. Evaluation of a QRS scoring system for estimating myocardial infarct size.7. Specificity in a control-group with right ventricular hypertrophy due to mitral-stenosis. American Journal of Cardiology. 1988;62:322–324.
33. Ideker RE, Wagner GS, Ruth WK, et al. Evaluation of a QRS scoring system for estimating myocardial infarct size.2. Correlation with quantitative anatomic findings for anterior infarcts. American Journal of Cardiology. 1982;49:1604–14.
34. Roark SF, Ideker RE, Wagner GS, et al. Evaluation of a QRS scoring system for estimating myocardial infarct size.3. Correlation with quantitative anatomic findings for inferior infarcts. American Journal of Cardiology. 1983;51:382–89.
35. Wagner GS, Freye CJ, Palmeri ST, et al. Evaluation of a QRS scoring system for estimating myocardial infarct size.1. Specificity and observer agreement. Circulation. 1982;65:342–47.
36. Ward RM, White RD, Ideker RE, et al. Evaluation of a QRS scoring system for estimating myocardial infarct size.4. Correlation with quantitative anatomic findings for posterolateral infarcts. American Journal of Cardiology. 1984;53:706–14.
37. Engblom H, Wagner GS, Setser RM, et al. Quantitative clinical assessment of chronic anterior myocardial infarction with delayed enhancement magnetic resonance imaging and QRS scoring. Am Heart J. 2003;146:359–66.
38. Xiong Y, Wang L, Liu W, Hankey GJ, Xu B, Wang S. The prognostic significance of right bundle branch block: a meta-analysis of prospective cohort studies. Clin Cardiol. 2015;38:604–13.
39. Wagner GS, et al. Evaluation of a QRS scoring system for estimating myocardial infarct size. Circulation. 1982;65:2;342–7.
40. Palmeri ST, et al. A QRS scoring system for assesing left ventricular function after myocardial infarct. The New England Journal. 1982;306:1;4–9.
41. Prashant G, Sharma R. Accuracy of ECG in assessment of post-myocardial infarction left ventricular function and its comparison with echocardiography. IOSR-JDMS. 2014;13:10;80–4.
42. Selvester RH, Wagner S, Hindman. The Selvester QRS scoring system for estimating myocardial infarct size. Arch Intern Med, 1985;145:1877–81.
43. Wilson FN, Kossmann CE, Burch GE, et al. Recommendations for standardization of electrocardiographic and vectorcardiographic leads. Circulation. 1954;10(4):564–73.
44. Fye B, MD. A history of origin, evolution, and impact of electrocardiography. American Journal of Cardiology. 1994;73(13):937–49.
45. Bergovec M, Prpic H, Mihatov S, Zigman M, et al. The value of the QRS scoring system in assessing regional and global left ventricular ejection fraction early after myocardial infarction. European Heart Journal. 1993;14:1102–9.
46. Uyarel U , Cam N, Okmen E, et al. Level of Selvester QRS score is predictive of ST-segment resolution and 30-day outcomes in patients with acute myocardial infarction undergoing primary coronary intervention. Am Heart J 2006;151:1239.e121239.e7.
47. Walther CMR, van Boreen MC, Robinson M, et al. Comparison of the correlation of the Selvester QRS scoring system with cardiac contrast-enhanced magnetic resonance imaging–measured acute myocardial infarct size in patients with and without thrombolytic therapy. Journal of Electrocardiology. 2009;42:139–44.
48. Shaimaa MA, Mohamed AA, Heba AM, et al. Electrocardiographic predictor of final infarct size by Selvester QRS score in correlation to rest Tc99M Sestamibi SPECT after Primary PCI. Med. J. Cairo Univ.2015;83(1):499–506.