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# Evaluation of Measuring Propagation Velocity of the Descending Aorta as Predictor of Coronary Artery Disease

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## ABSTRACT

**History:** Elastic features of the aorta affect the left ventricular function and blood supply from the epicardial arteries. For this reason several criteria are used to check particular characteristics, so that the likelihood of atherosclerosis-induced coronary artery disease, a known risk factor in coronary arthriology, can be calculated. These parameters include aortic pressure, aortic distensibility and the anterior aortic wall's early diastolic speed.

**Aims of the study:** To compare aortic velocity and the occurrence and extent of coronary artery disease with other conventional aortic rigidity parameters for predicting coronary artery disease among patients with atherosclerosis
Patients and methods: 140 people, 70 in the group of coronary artery diseases and 70 in the category of non-coronary artery diseases were included. In addition to the measures required to achieve aortic rigidity and distensibility, clinical data were checked and coronary Angiography reports, routine echocardiographic echocardiography measures were collected. The velocity of the anterior aortic wall, aortic spread velocity and intimate carotid thickness were all measured.

**The result:** Aortic propagation velocity associated with aortic distensibility was estimated, pressure, anterior early diastolic velocity and statistical thickness of the total carotid intimate medium (r = +0.481 p <0.001; r = + +0.548, p <0.001; r = +595, p < 0.001; r = <0.361, p <0.001). Aortic propagation velocity has been found to predict coronary artery disease (CAD) (p < 0.001) and Correlates to the number of affected coronary arteries (r = -0.711, p<0.001).

**Conclusion:** Aortic spreading speed is an early-applied echocardiographic artic stiffness parameter that can predict non-invasive coronary artery disorders caused by vascular atherosclerosis. This also refers to the number of coronary arteries affected.

**Keywords**: Aortic stiffness, Atherosclerosis, Propagation velocity, Coronary artery disease.

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## INTRODUCTION

The predisposing factor for CVD is known for atherosclerosis, which typically has a long latent, asymptomatic duration and provides an opportunity for early prevention. There are many risk factors such as diabetes dyslipidemia, hypertension, BMIs, smoking, DM, and genetic predisposition. It has many risk factors. The accelerating arterial stifling that affects the arterial system in various degrees and locations was a result of these classic risk factors. [1,2] The [3] The aorta, the largest vessel in the arterial system, provides insight into the mechanism of aortical pressure and diameter alters in the Elastic properties of the aorta, and the loss of the arterial properties of the left ventricular function and coronary blood flow. [4] Stiffing and reduced arterial compliance is one of the earliest detectable indicators that adverse structural and functional changes occur within the artery wall and a number of studies have reported the prediction of blood rigidity as an independent indicator of cardiovascular morbidity and causal mortality in various populations. [5] Aortic strain (AS), aortic distensibility (AD), diastolic anterior aortic movement velocity (Eao) are well known, while the aortic spread velocity (APV) is a new parameter for aortic stiffness defined and used in guns and associated with the association between atherosclerosis of the aorta and carotids[6]. An external carotid ultrasound, on the other hand, is likely to remain one of the most widely used imaging techniques for the measurement and monitoring of sublinical atherosclerosis in the carotid artery intima median thickness (CIMT). [7] The study attempted to compare APV to other traditional aortic rigidity parameters, and their correaction to the presence and gravity of CAD in the prediction of CAD among atherosclerosis patients.

## PATIENTS AND METHODS

Between July 2015 and June 2016, a total of 140 participants participated in the study. The population in the sample was divided into two categories; the 1st category consists of a group of (70) patients confirmed to have coronary angiography confirming CAD. The second group is the non-CAD group, composed of (70) average coronary angiographer participant.
Heart failure, cardiomyopathy, atrial fibrillation, Serious or severe valvular stenosis or regurgitation, brain rhythm, early stage of acute myocardian infarction (in six weeks), some congenital heart disease, generalized aorta (Marfan, Ehro-Dahnlos), and weak echocardiographical accuracy. Severe or severe valvular stenosis and regurgitation.
Medical and general age, sex, DM, HT, obesity (represented as MBI), CAD history (FH), diastolic & systolic blood

pressure, along with pulse pressure controls (PP). Positive FH of premature atherosclerotic CAD is not reported to occur unless the patient has a first degree of the female relative diagnosed with CAD before age 65.8] A stenosis of at least 70% of the diameter of o was determined by a single vessel coronary artery disease in all CAD group members. Two and three vessels of disease were diagnosed with at least 70 percent stenose for one or two additional main epicardial arteries. Two-vessel disease is diagnosed in patients who have 70 percent or more of the central left coronary artery stenosis.

**I. Echocardiographic Transthoracic test 1. 1. TTE routine**

Echocardiography of the two patients was performed with Philips or Vibrant E 9 (General Electric, Fairfield, CT, USA) 4–5 MHz transduting device TTE. In the latest guidelines published by the American Society of Echocardiography (ASE), the left lateral decubitus task examined systolic and diastolic cardiac function. Calculated using the amended 2D-Mode method, LVeF was defined by LVEF as LVEF < or= 52 percent for female, and by LVEF = 54 percent for male. LVEF was calculated using the updated 2D-Mode method. The diastolic function LV (grade I LVDD) was considered to have been affected when the early ante grade trans MV diastolic flow velocity(e) is lower than 1, i.e. (E/A) <1 with a deceleration time extension >250 ms; LVDD grade II (normal pseudo filling) with a normal E/A rate >1 and <2, with a normal LVDD grade of E'<8 and E/E '> 10; [9] 2.At 3 cm above the para-sternal long axis, M — mode was used to determine systolic and diastolic diameters of the ascent aorta. When fully opening the aortic valve, the aortic systolic diameter was measured while the diastolic diameter was measured on the basis of the QRS electro-cardiographic peak. The average measurements were made in five consecutive measurements. (Fig. 1). [9].

3. The echocardiographic device was then changed into TDI mode with the same view, and with the use of a pulsed wave Doppler with a sampling volume of 1 mm the previous aortic diastolic velocity of Eao was measured in the same point as that used in the measurements of the M-mode. (Fig. 2). [10].

4. APV measurement: Color M -mode Doppler recording was obtained using a long axis aortic view with the cursor parallel to the main track flow in the downing aorta from the supraastern window, the Nyquist limits were set at 30–50 cm / s, and a flame-like space-time velocity map was displaced by switching to space-mode M-. By simply tracing the velocity curve, the aortic velocity of propagation was calculated by dividing the spread curve by time. There was a mean of at least three consecutive measurements and the APV value was taken into account. (Fig. 3) 5. [11]. The aortic diameters AS and AD were measured using an echocardiographic sphygmomanometer and the blood pressure was clinically assessed. By subtracting the aortic diastolic pressure from the systolic aortic pressure, the aortic pulse pressure was measured. AS and AD were used as the principal parameters of aortic elasticity. In order to calculate the parameters mentioned above, AS (percent) = (AoS) * 100/AoD (cm2.dyn-1.10-3) = 2 * AS / Pulse Pressure Where AoS is a systolic aortex diameter (AoD) is a diastolic aoretic diameter. [12] 3. 2. The same sonographer has been using a Philips or Vivid E 9 (GE) system with a LATAL array (5-7.5) MHz, to check traditional carotid arteries to the left and right. Patients in their supine position were filmed from their side with their heads turning 45. 10millimeter of The Common Carotid Artery Segment for common carotid artery calculations is determined after bulbus. The anterior (closer) and posterior (far) walls of the carotid artery are presented as two bright white lines separated by a hypo-epocytic layer in a longitudinal, two-dimensional ultrasonic image of the carotid artery. There is a difference in intimate thickness between the front of the first bright line of the far walls and the forefront of the bright second line. (Fig. 4). CIMT measures were carried out at four different locations, which established a median value, in addition to a distance of one cm from each other. For the right and left carotid arteries, the mean and average CIMT values have been measured and the total average and mean values calculated.. [11] [11] [2]


**Figure 1: Aortic systolic and diastolic (AoS and AoD respectively) diameter measurement in M- mode[14]**

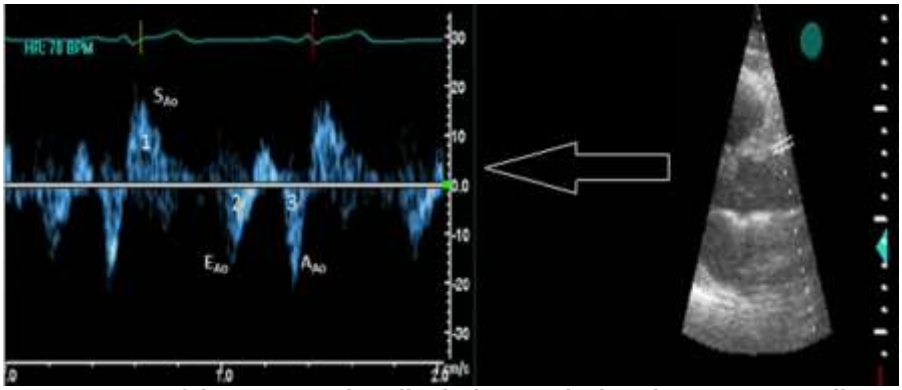


**Figure 2: Tissue assessment of the upper aortic wall velocity Doppler imaging. 1=Sao: systolic aortic upper wall velocity while .2=Eao: early diastolic aortic upper wall velocity; 3=Aao: late . . diastolic aortic upper wall velocity[15]**

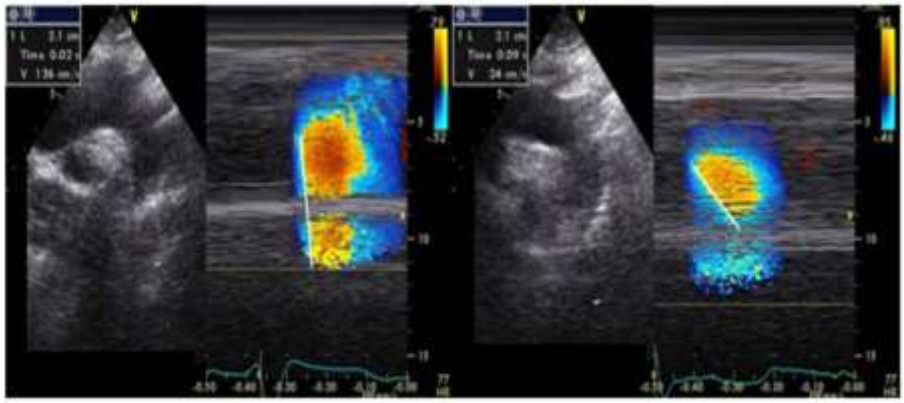

**Figure 3: To the left is the measurement of APV in an individual with normal coronary angiography and to the right is APV measured in a patient with coronary artery disease. [The APV is determined by dividing the interval between starting and ending points of the propagation path by the time between the time points in question].[11]**

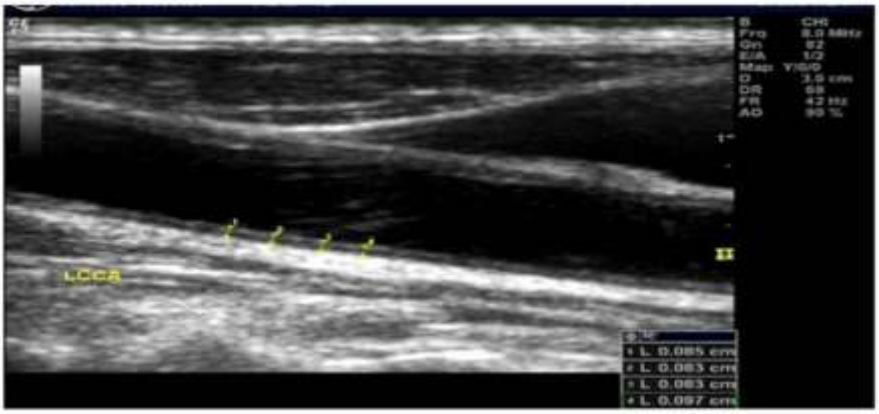


**Figure 4: CIMT measurement of carotid intima media thickness by means of M-mode trans thoracic echocardiography, LCCA is the left common carotid artery.[11]**

## Statistical analysis

A number for the serial identification was allocated to each case sheet. The data have been analyzed with the Social Sciences Statistics Kit (SPSS) version 23. Data are viewed as a medium variance and the norm. The ongoing variables among analytical groups were compared with independent t-test (two tailed). Table of frequencies and percentages display the categorical results. Pearson's Chi-square test was used to evaluate the statistical relationship between sample groups and the category results. The correlations between aortic spread speed and carotid intima medium-thickness, an aortic-strain, aortic disagreement and number affected by both groups of studies were assessed with the correlation of Pearson. The correlation was considered weak when the correlating coefficient(r)(0-0.3), mild if (r=0.3-0.7, and strong if (r>0.7)), when the correlation was proceeding by (+), the correlation was direct or positive, and then the correlation was reverse or negative if preceded by-) (sign. Receiving Operator (ROC) curves used to detect aortic propagation velocity sensitivity and specificity for CAD, HT and DM detection. The p-value was important less than 0.05.

Results 42 (61,7 and 38,3 percent) males and 28 females were in the CAD community. Its average age was (56.1±8.8). The non-CAD category is evenly split between the sexes, with a mean of 35 (52.5±9.3) men and 35 females (50%). There were no major differences in sex, DM, or FH between the demographics and the clinical characteristics of both classes, while their average mean BMI, HT, mean LVEF and LVDD were substantially differentiated. In patients in the CAD group of this study, the age was higher and the mean BMI was significant at the P level of 0.05, the mean LVEF was reduced, more patients had hypertensive and more of LVDD were significant at 0.01 level. (Table 1) Unlike AOD and AoS which are substantially different and higher in the CAD group, aortic systolic and diaastolic blood and PP did not vary between the study groups at statistically significant rates. In comparing the APV values, AS, AD and Eao varied from the statistically relevant point of two classes. In non-CAD group the mean APV was 38.2±15.3, and in the p value was < 0.001 (range 56 – 148 cm / s); in Non CAD community it was 87±24.2. (Paragraph 2)

**Table 1: The CAD and non-CAD goup' clinical and demographic features.**

| | CAD group N=60 | Non- CAD group N=60 | P - value |
|---|---|---|---|
| Age (years), Mean±SD | 56.1±8.8 | 52.5±9.3 | 0.033* |
| Gender, No (%) [c] | | | |
| Male | 42 (55.2%) | 35 (44.8%) | 0.198 ns |
| Female | 28 (43.4%) | 35 (56.6%) | |
| BMI kg/m$^2$, Mean±SD | 31.4±4.5 | 29.7±3.9 | 0.024* |
| Hypertension, No (%) [c] | 43 (71.7%) | 28 (46.7%) | 0.005** |
| Diabetes mellitus, No (%) [c] | 16 (26.7%) | 16 (26.7%) | 1 ns |
| Family history, No (%) [c] | 26 (43.3%) | 18 (30%) | 0.13 ns |
| LV EF%, Mean±SD | 56.6±7 | 63.4±4 | <0.001** |
| LVDD, No (%) [c] | | | |
| Normal | 7 (11.7%) | 22 (36.7%) | |
| Grade I | 41 (68.3%) | 36 (60%) | <0.001** |
| Grade II | 12 (20%) | 2 (3.3%) | |

**Significant at 0.01 Level, ns= not significant,*Significant at 0.05 level,
Independent t-test, [c] Chi-square test
BMI, body mass index; CAD, coronary artery disease; LV EF%, left ventricular ejection fraction; LVDD, left ventricular diastolic dysfunction.

**Table 2: CAD and non-CAD Group, aortic stiffness parameters.**

| | CAD group N=60 Mean±SD | Non- CAD group N=60 Mean±SD | p-value |
|---|---|---|---|
| Systolic diameter (cm) | 33.1±5.5 | 30.7±4.3 | 0.011* |
| Diastolic diameter (cm) | 30.8±5.3 | 27.5±4.4 | <0.001** |
| Systolic BP | 147.1±21.3 | 142.7±18.1 | 0.222 ns |
| Diastolic BP | 84.1±13.2 | 83.5±13.6 | 0.791 ns |
| Aortic strain (%) | 7.1±2.1 | 12.1±3 | <0.001** |
| Aortic distensibility ($cm^2.dyn^{-1}.10^{-3}$) | 2.5±0.7 | 4.3±1.4 | <0.001** |
| Eao (cm/s) | 7.2±1.3 | 9.1±0.3 | <0.001** |
| APV (cm/s) | 38.2±15.5 | 87±24.4 | <0.001** |

**Significant at 0.01 Level, ns= not significant *Significant at 0.05 level,
Independent t-test; CAD, coronary artery disease; BP, blood pressure; Eao, early diastolic anterior aortic wall velocity; APV, aortic propagation velocity.

With respect to the right and left CCA CIMT, the mean values of the CAD Group in the non-CAD Group with a value < 0.001 have been found to be higher at a statistically significant point. The same result was made when the average CIMT values between the two sample groups were compared with the same p value. (Table 3)

**Table 3: Carotid intima media thickness, CAD and non- CAD groups.**

| | CAD group N=60 Mean±SD | Non-CAD group N=60 Mean±SD | P - value |
|---|---|---|---|
| CIMT of the Rt. Carotid artery (mm) | 0.7±0.2 | 0.5±0.1 | <0.001** |
| CIMT of the Lt. Carotid artery (mm) | 0.9±0.7 | 0.6±0.1 | 0.001** |
| The Overall CIMT (mm) | 0.8±0.2 | 0.5±0.1 | <0.001** |

CAD, coronary artery disease; CIMT, carotid intima media thickness**Significant at 0.01 Level
Independent t-test; *Significant at 0.05 level,

The Pearson analysis was used to obtain the correlation coefficients (r-value) as seen in Fig. 5, 6, 7 while attempting to compare the APV with specific variable. In the study, the anterior velocity aortic wall aortics, strain and early diastolic aortic wall ($r = + 0.481$, $p < 0.001$; $r = + 0.548$, $p < 0.001$; $r = + 0.595$, $p < 0.001$ respectively) was found significantly related to the aortic stiffness parameters used in this research. In Eao and in AD, the association between the APV and the Eao and the AS was positive or similar and statistically moderate. Statistically important negative and reverse association of the APV was evident from an association coefficient greater than 0.7 ($r=-0.711$, $p < 0.001$) with the amount of affected coronary arteries. (Table 4)

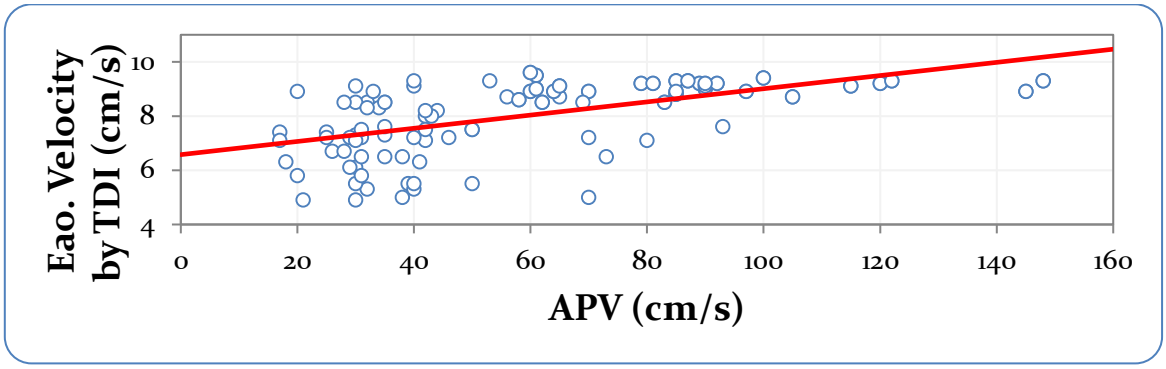


**Figure 5:** **The association between aortic propagation velocity (APV) and early diastolic anterior aortic wall velocity (Eao).**

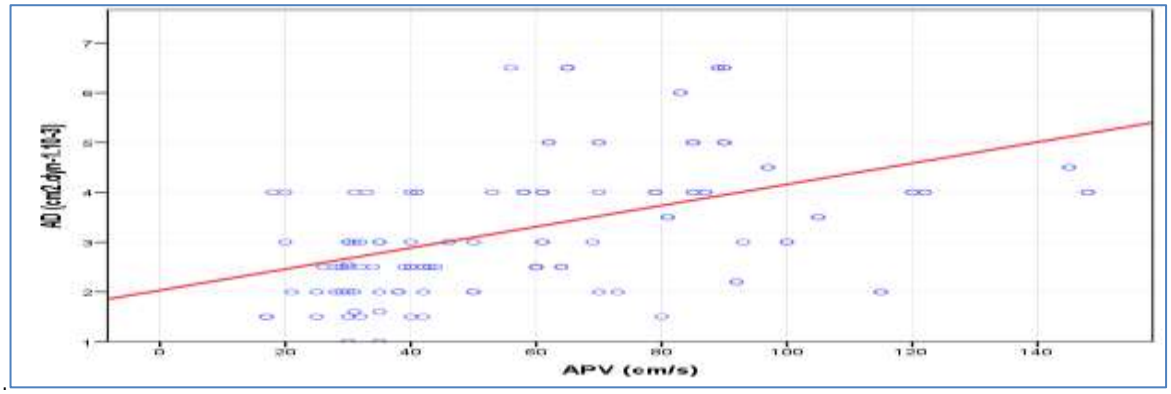


**Figure 6:** **The association between aortic propagation velocity (APV) and aortic distensibility (AD).**

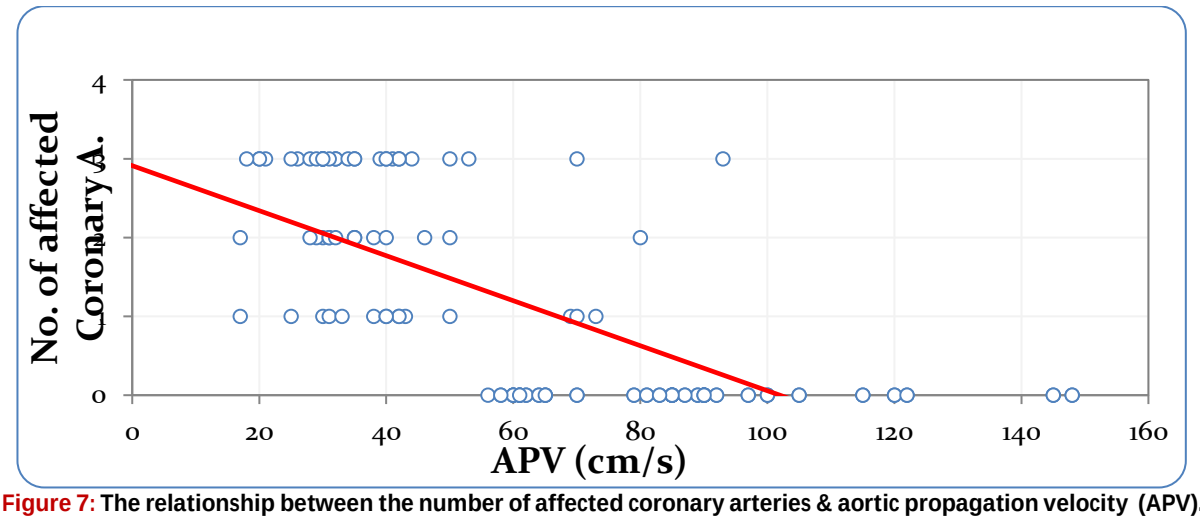


**Figure 7:** **The relationship between the number of affected coronary arteries & aortic propagation velocity (APV).**

**Table 4: Relationship between overall CIMT & APV, early diastolic anterior aortic wall velocity (Eao), aortic strain (AS), aortic distensibility (AD) and number of affected vessels.**

| Variables | APV (cm/s) | |
|---|---|---|
| | r-value | p-value |
| Overall CIMT (mm) | -0.361 | <0.001** |
| Aortic distensibility (cm2. dyn -1.10-3) | + 0.481 | < 0.001** |
| Aortic strain (%) | +0.548 | <0.001** |
| Eao. Velocity by TDI (cm/s) | +0.595 | <0.001** |
| No. of affected Coronary arteries | -0.711 | <0.001** |

**Significant at 0.01 level
CIMT, carotid intima media thickness; Eao, early diastolic anterior aortic wall velocity.

The mean APV value represented as mean ± SD was found to differ when The number of coronary arteries affected changes being highest in the non CAD group and lowest when three coronary arteries were affected (Table 5 )

**Table 5: The mean values ± SD of the aortic propagation velocity among the study groups**

| Number of affected vessels | Number of patients | Aortic Propagation Velocity (cm/s) | |
|---|---|---|---|
| | | Mean | Standard Deviation |
| .00 | 60 | 86.98 | 24.18 |
| 1.00 | 15 | 42.87 | 16.50 |
| 2.00 | 15 | 37.13 | 14.18 |
| 3.00 | 30 | 36.33 | 15.22 |

In differentiation of dependent and independent factors regarding CAD in this study, a multi variate regression analysis (MVRA) was built and it showed a significant relation between CAD on one hand and LVDD, AS, Eao, and APV on the other hand as independent CAD factors (P-value is 0.001, 0.049, <0.001, and <0.001 for the mentioned factors respectively). The highest significant P-values were those of Eao and APV. While dependent factors were age, BMI, sys.BP, LVEF%, CIMT and AD with not significant P values. (Table 6)

**Table 6: Multi variate regression analysis for CAD.**

| Variables | Beta | t | P-value |
|---|---|---|---|
| Age (years) | -0.021 | -0.373 | 0.71 ns |
| BMI (kg/m2) | -0.034 | -0.628 | 0.531 ns |
| Sys BP (mmHg) | -0.124 | -1.636 | 0.105 ns |
| LV EF% | -0.054 | -0.758 | 0.45 ns |
| LVDD | 0.217 | 3.259 | 0.001** |
| CIMT (mm) | 0.04 | 0.726 | 0.47 ns |
| AS% | -0.184 | -1.987 | 0.049* |
| AD (cm2.dyn$^{-1}$.10$^{-3}$) | -0.147 | -1.521 | 0.131 ns |
| Eao. Velocity by TDI (cm/s) | -0.253 | -3.589 | <0.001** |
| APV (cm/s) | -0.300 | -4.182 | <0.001** |

ns= not significant *significant at 0.05 level, **significant at 0.01 level

By using ROC for the validity criteria of APV (Fig.8), it was found that it is both sensitive and specific in the prediction of CAD with a sensitivity of 90.0% and a specificity reaching up to 100.0% when the value of APV is less than 53 cm/s (35 cm/s is the cutoff APV value obtained statistically from this study data).

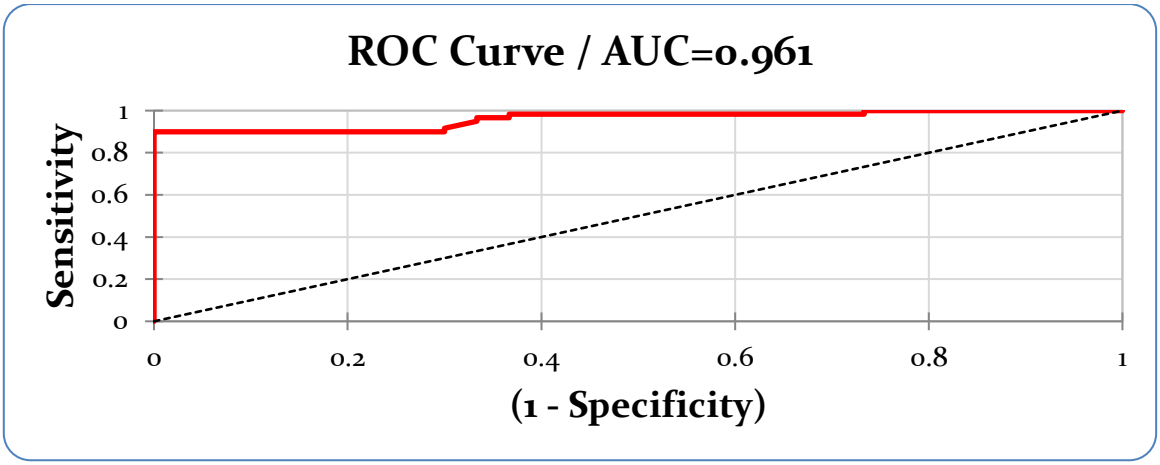


**Figure 8: Receiver operator curve (ROC), for validity criteria of the aortic propagation velocity in detection of Coronary artery diseases.**

## DISCUSSION

This work indicates that there were significantly different differences between the aortic stiffness, the aortic stiffness and the progressively atherosclerosis [16 and 17 and 18] of the CAD population and the non-CAD population in this group, as each variable is obviously related to the aortic stiffness of the artery [16, 17 and 18]. [19] Both AoS and AoD were found to be considerably higher in the CAD group and this possibly depends on the fact that more hypertensive pain was present in the CAD group than in the other group. Previous studies have proven that higher aortic measurements are correlated with HT. [20, 21] In 2014 the same finding was reported by Mirea O et al. [22] Statistically significant deficiency was found in the aortic rigidity parameter in the CAD community, as explained pathophysiologically; the starting point is that in this community the risk factor of atherosclerosis was significantly higher than in the other group (statistically confirmed) As the coronary arteries fill during diastolic disease, this reduction in diastolic BP can lead to a deterioration in myocardial infusion and thus cardiac ischemia and CAD. [23] The direct involvement of coronary arterial walls by an atherosclerotic transition, which leads to narrowing and leading to incompatibility in supply and demand, is another mechanism that contributes to the development of the CAD induced by atherosclerosis. In 2014, GungenB et al showed that CAD is related to significant decreases in the rate of eao [15], with the results obtained in the analysis on the relation between these four parameters, and the results of these two papers support the findings of SenT et al. in 2013, that the traditional aortic echocardiographic stiffness (AS andAD) and the CaD present significantly decreases in APV [16]. In comparison with patients from the non-CAD community, CIMT levels were found to be significantly higher. This is supposed to be done as the risk factors of atherosclerosis among the CAD patients were considerably higher. The average CAD population was (56,1±8,8) years with a mean total CIMT (0,8±0.4 mm) above the usual value for the age group predicted (< 0.71 mm)[24] due to the impact of the arteries of atherosclerosis, the thickness of the intimate medium increased by smooth cell proliferation and the migration of intimate muscle cells[25] early in disease, type I Stary classi Type I [25] [26] The APV has been found to have an AS, AD and Eao relationship. The latter three are parameters that represent the physical characteristics of the aortic wall when a resistant non-elastic vessel has less propagation velocity than flow through a healthy wall vessel [10]. This is why APV on the one hand is connected to aortic rigidity parameters, and Sen et al have concluded that in 2013[11] The APV was associated with the number of coronary arteries affected by coronary angiography and the mean APV was higher (86.9±24.19 cm / s) where the coronary artery (non-CAD) was not affected by Gune Y et al. in the 2008 analysis of 91 CAD patients[6];where the association between APV and the number of CAD damaged coronary arteries has not been identified in their study[11], as opposed to Sen T et al. This association has been shown by Gune Y et al, which involves 51 CAD patients[11], in the smaller number of their population.

The aorta's extent of atherosclerosis (reflected as decreased aPV) can generally not be associated with coronary atherosclerosis due to unequal distribution of atherosclerosis in various parts of the arterial system. There is growing evidence, however, that aortic atherosclerosis is a good predictor for generalized aorta. [27] In the years before 1993, Fazio et al registered a marker of substantial CAD angiographical sensitivity of 90 percent and 90 percent specimen for the presence of atherosclerotic plaque in a thoracic aorta. [28] Aortic plaque detected from TEE also had 93 percent sensitivity and 82 percent specificity for significant CAD detection, [29] it was reported that the use of TEE for routine risk assessment of CAD in daily practice is not suitable.

A multiple variant regression analysis found that LVDD and drei out of four parameters in Aortic Stiffness were independent contributors to CADs with statistically significant P values, 0.001, 0.049, < 0.001 and < 0.001, in order to distinguish the aortic stiffness from independent CAD bed-side forecasting factors or variables used in this research. The LVDD independence in this study was possibly due to the fact that LV DD and aortic rigidity are closely related because Lv DD itself is a result of blood flowing from L A in a four-degree pathology, creating

varying degrees of wall tension, to a more rigid than normal LV. [16] LV Wall Structural Changes, leading to an rise in systolic and diastolic myocardial rigidity, will gradually buffen blood pumping tension against a solid aorta. [16] [30] The fact that LV wall stresses are determined not only by the geometrical properties of the ventricle, but also by aortic rigidity is explained in the AS, Eao and APV independency of this report. [30] AD has probably not been independent because it is based on an equation which includes highly variable and multi-factorial pulse pressure readings.

To find a cutoff point of APV value in predicting CAD, the ROC analysis was used, it was found that any value of APV of =or<53cm/s, predicts CAD with a sensitivity of 90.0% and a specificity of up to 100%.In a published study in 2010, Kaya Y et al found that at an APV of ≤32 cm/ sec was 100% specific for the prediction of CAD. [31] Both Sen T et al in 2013 and Gunes Y et al in 2008 found that APV is both sensitive and specific in predicting CAD with sensitivity and specificity of 90.5% and 92.2% at cutoff point of =or<60.5cm/s by Sen T et al, and a sensitivity and specificity of 82.4%, 97.2% respectively at a lower cut off points of =or<41cm/s. [11] [6].

## LIMITATIONS

Not considering the effect of medical treatment of the participants on the parameters used in this study. This issue actually demands long follow up period. The usage of conventional TTE phased array probe for the CIMT measurement while typically it should be the linear probe that used for such purpose; this resulted in excluding some cases where a clear demarcation of the intima-media borders was difficult. Finally the study included limited number of participant who were all of the same race and from the same geographic area (Iraq).

## CONCLUSIONS AND RECOMMENDATIONS

APV is a modern, easy and inexpensive Aortic Stiffness Echocardiographic parameter feasible as a non-invasive bedside method for the prediction of CAD for early risk stratification in atherosclerosis patients. Furthermore, the number of affected coronary arteries shown to be associated with APV in coronary angiography was identified. Finally, we suggest conducting longitudinal studies to determine the medical care impact obtained by the population sample on APV standards, including the use of more sophisticated CAD rating systems such as the Gensini score system, with a greater number of participants of various groups. This also suggest that measurements obtained by magnetic resonance imaging or oscillometrie system techniques be compared to aortic rigidity parameters obtained through echocardiography (including APV).

## CONFLICT OF INTEREST

None

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