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Original Research Article

Role of Pulmonary Artery Acceleration Time in Detecting Pulmonary Hypertension and It's Severity

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Abstract

Transthoracic echocardiography with Doppler study is recommended as the initial noninvasive test in the screening, evaluation & monitoring of pulmonary hypertension by estimating pulmonary artery systolic pressure by measuring PA acceleration time which can estimate pressure severity. The aim of this study is to estimate the validity of pulmonary artery acceleration time as an parameter for evaluation and measuring pulmonary artery systolic pressure . This study: A cross sectional study has been carried out from December 2015 to May 2016 in El-Imamein Al-Kadhimain teaching hospital at echocardiography 61 patients (both normal &abnormal pulmonary pressure). Patients at first were assessed for presence of TR to include in the study& both estimated systolic & mean pulmonary pressure were assessed by both TR derived Doppler assessment & then measuring PAAT & PAAT derived pulmonary pressure with comparison between them .It was shownIn this studythat61patients were involved regardless of pulmonary pressure,with mean age of 55.1±17.6 (range 18-85 yr), 18 were male (29.5%) & 43 were females (70.5%). The mean of estimated peak systolic pulmonary artery pressure(EPSPAP) & mean pulmonary arterial pressure (MPAP) derived from tricuspid regurgitation done was 44.9±20.2 mmHg& 32.1±14.2 respectively. The mean value of pulmonary artery acceleration time (PAAT)was 106.89± 27.5ms& mean of PAAT derived MPAP was 30.9 ± 12.35 mmHg .The correlation of pulmonary artery acceleration time (PAAT) to pulmonary systolic pressure shows a significant association (r= - 0.693)(p=0.001) & the correlation of PAAT derived MPAP to TR derived MPAP showed significant correlation P<0.001.The sensitivity and specificity of pulmonary acceleration time in detection of pulmonary hypertension (using the value of 100ms as a cut off value) found sensitivity of 100% and specificity of 63.1%. PAAT was more useful in detecting moderate & severe pulmonary hypertension P<0.001.conclusion**:**Pulmonary artery acceleration time is a noninvasive obtainable echocardiographic parameter well correlated with measures of pulmonary artery systolic &mean pressure derived by tricuspid regurgitation .

Key Words : pulmonary artery acceleration time, estimated pulmonary artery systolic pressure, pulmonary hypertension.

دور فحص وقت تسارع الشريان الرئوي في تحديد وجود شدة مرض ارتفاع ضغط الشرياني الرئوي

الخلاصة

يعد فحص صدى القلب عبر الصدر مع دراسة دوبلر من الفحوصات الأولية في فحص وتقييم ورصد ارتفاع ضغط الدم الرئوي عن طريق تقدير الشريان الرئوي الضغط الانقباضي عن طريق قياس التسارع الوقتي للشريان الرئوي والذي يمكن تقدير شدة الضغط من خلاله. الهدف من هذه الدراسة هو تقدير صلاحية وقت تسارع الشريان الرئوي كمؤشر لتقييم وقياس الضغط الرئوي الانقباضي الشريان. هذه الدراسة: تم إجراء دراسة مقطعية من ديسمبر 2015 إلى مايو 2016 في المستشفى التعليمي العام في مدينه الإمامين الكاظمين الطبية وجرى تقييم المرضى في البداية لوجود TR ثم

تقييمها من قبل كل من تقييم دوبلر TR المشتقة وثم قياس PAAT و PAATالمستمدة الضغط الرئوي مع المقارنة بينهما. شملت هذه الدراسة 61 مريضا يعانون من ارتفاع الضغط الرئوي، بمتوسط عمر 55.1 ± 17.6 (المدى 18–85 سنة)، 18 كانوا من الذكور (29.5٪) والإناث 43 (70.5٪). وبلغت القيمة الوسطية للشريان الرئوي تسارع الوقت 27.5 ± 106.89 (PAAT) مللي وتعني من PAAT تستمد كان MPAP 30.9 ± 12.35 ملم زئبقي. والترابط بين الشريان الرئوي تسارع الوقت (PAAT) للضغط الانقباضي الرئوي يدل على ارتباط كبير (ص = 0.693 –) (ع = 0.001) وارتباط PAAT تستمد MPAP إلى TR أظهر MPAP المستمدة ارتباط كبير .0.001> P وحساسية وخصوصية تسارع الوقت الرئوي في الكشف عن ارتفاع ضغط الدم الرئوي (باستخدام قيمة 100 MS كقيمة قطع) حساسية وجدت من 100٪ وخصوصية 63.1٪. كان PAATأكثر فائدة في الكشف المعتدل والحاد وارتفاع ضغط الدم الرئوي .0.001> P الاستنتاج: هذا المقياس يمكن استخدامه في دراسة و تقيم امراض الشريان الرئوي من خلال فحص صدى القلب عن طريق الصدر.

الكلمات المفتاحية :وقت تسارع الشريان الرئوي، الضغط الرئوي الانقباضي الشرايين، ارتفاع ضغط الدم الرئوي، فحص صدى القلب.

Abbreviations

PAAT	pulmonary artery acceleration time
TR	tricuspid regurgitation
EPSPAP	estimated peak systolic pulmonary artery pressure
MPAP	mean pulmonary artery pressure
TR vmax	TR maximal velocity
RVSP	right ventricle systolic pressure

Introduction

Pulmonary hypertension (PH) is a hemodynamic and pathophysiologic state defined as a raised in mean pulmonary artery pressure(MPAP) of 25 mm Hg at rest as measured by right-heart catheterization (RHC)It can be found in many clinical conditions with distinct pathogenetic and clinical features, such as pulmonary arterial hypertension (PAH) and left-heart, lung, and thromboembolic diseases [1,2]

Five categories of disorders that cause PH were seen: pulmonary arterial hypertension (Group 1); pulmonary hypertension due to left heart disease (Group 2); pulmonary hypertension due to chronic disease of the lungs and/or hypoxia (Group 3); chronic thromboembolic pulmonary hypertension (Group 4); and pulmonary hypertension due to unclear multifactorial causes (Group 5) as state in updated clinical classification of pulmonary hypertension in 2013[3].

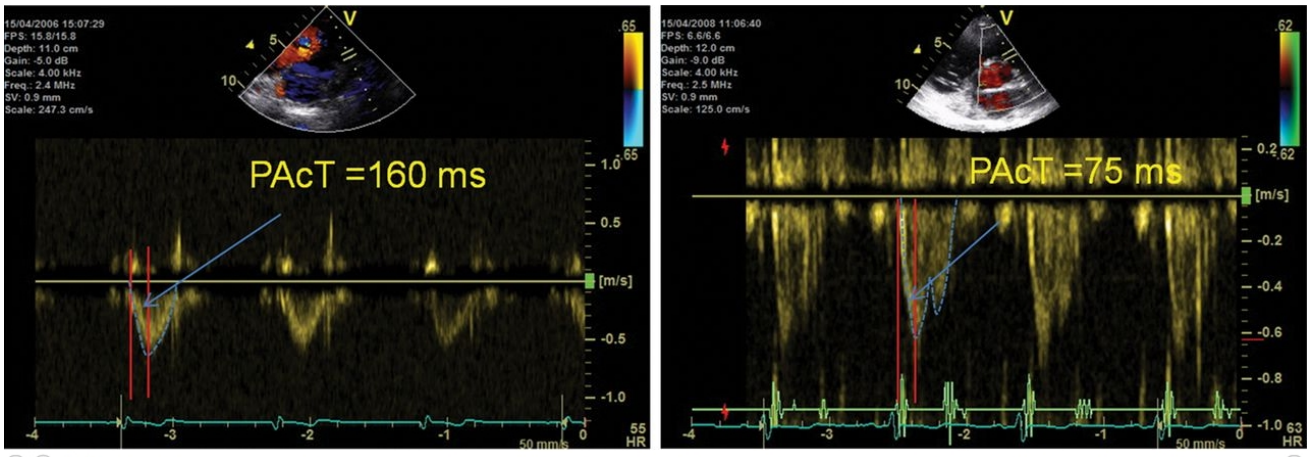
Given the subtle symptoms and physical signs, particularly in the early stages, a high index of suspicion is mandatory to diagnose the disease before irreversible pathophysiologic changes happened.

In this regard, cardiac echocardiography, by providing directand/or indirect evidences of elevated pulmonary artery pressure (PAP), is a very excellent noninvasive screening test for those patients with symptoms orrisk factors for PH, such as connective tissue disease, anorexigen drugs user, pulmonary embolism, heart failure, and heart diseases. It may alsovital key information on both the etiology and the prognosis of PH[4,6].

measurement of pulmonary artery pressure by using transthoracicechocardiography (TTE) are most commonly done by usingcontinuous-wave Doppler to record the maximum velocity of tricuspid regurgitation (TRVmax). This technique of pulmonary arterypressure quantification has been widely accepted for clinical and researchaims because it provides a non-invasive, direct measureof right ventricular (RV) systolic pressure (RVSP) that correlates

closely with invasive hemodynamic estimation[7].In the absence of significant RVout flow tract obstruction, this way providesA good means of obtaining estimatedEPSPAP.

However, this method of pulmonary arterypressure assessment is not possible when TR is absent or non-significant.Alternative transthoracic echocardiographic methods for pulmonary artery pressure estimation also have been used. These includethe measurement of blood flow through an anatomic defect (ventricularseptal defect,[8,9] patent ductusarteriosus,[10] or aorto-pulmonaryshun [11], estimation of the peak systolic and end-diastolic pulmonicvalve regurgitant velocity[12] and measurement of the pulmonary arteryacceleration time (PAAT)by Pulsed-wave Doppler interrogation of the RVoutflow tract often which reveals an acceleration time of <100 msec, that reflects abnormal MPAP[13-16].



The mean pulmonary arterial pressure can be measured through the PAATby an equation described byDabestan[16] as the following, MPAP =73 -(0.42 - PAAT).

The aim of this study is to assess the accuracy of pulmonary artery acceleration time in evaluation and measuring pulmonary artery systolic & mean pressure (using a determined cut off value) andcompare it with the standard method of TR –derived PSPAP measurement by transthoracic cardiac echocardiography and to see its abilityto detect the grade of pulmonary hypertension.

Materials and Methods

A61 patients were enrolled in this cross-sectional study, all patientswere involved had a sinus rhythm with in 50 and 100 beats/min regardless of absolute level of pulmonary artery pressure but with measurable tricuspid regurgitation flow to estimate pulmonary meanand peaked systolic pressure.Study conducted in the period between October 2015 –March 2016 in Al-Imamin Al-Kadhumain teaching hospital at the echocardiography specialized unit.The patients were evaluated by single operator using echocardiography machine GE ,Vivid E9 for measurement of tricuspid regurgitation maximum velocity from which peak systolic pulmonary pressures were obtained using the modified Bernoulli equation: **4×TRVmax2 + (5 – 15 mm Hg)** (to account for right atrial pressure) and mean pulmonary pressures were also obtained by automatic measurement of the mean doppler gradient of the TR signal as in figure -1-, then measuring pulmonary artery acceleration time (from which mean pulmonary artery was obtained by this formula: **MPAP =73 -(0.42 - PAAT)**[16], in addition to general assessment of left and right sides of the heart structures and functions. Measurement done for three cardiac cycles.

Demographic data obtained including age, sex, cause of pulmonary hypertension ,heart rate , presence of atrial fibrillation Pulmonary hypertension was defined as PASP >35 mm Hg. Patients were

categorized according to the following categories: normal PSPAP (PSPAP ≤ 35 mm Hg), mild (PSPAP 36–45 mm Hg), moderate (PSPAP: 46–60 mm Hg) and severe PH (PSPAP>60 mm Hg)[17].

Exclusion criteria

were Patients with pulmonary valve stenosis (defined by a continuous-wave peak jet velocity >2 m/sec across the pulmonic valve)& other congenital heart diseases, patients with no adequate imaging , patients with no measurable TR & patients with atrial fibrillation were excluded from assessment.

Statistical Analysis

The analysis done by the Statistical package for social sciences (SPSS)version 21. Numerical data described as mean and standard deviation. Categorical data described as count and percentage. Chi-square test used to evaluate the association between tested parameters. Pearson correlation test used to assess the significance of correlation between parameters. Sensitivity, specificity, positive and negative predictive values with confidence interval were calculated.

Ethical consideration

informed consent was obtained from all patients enrolled in this study.

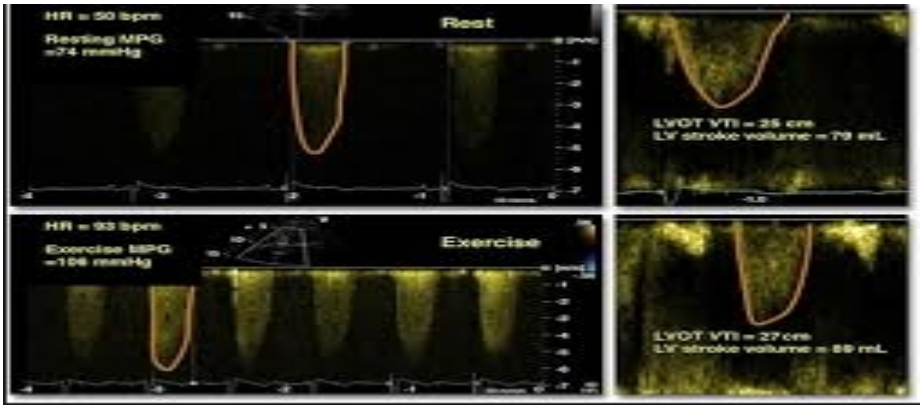


Figure 1: Measurement of mean Doppler gradient of TR signal by transthoracic echocardiography

Results

In this study, 61 patients were involved regardless of magnitude of pulmonary pressure,with mean age of 55.1±17.6 (range 18-85 yr), 18 were male (29.5%) & 43 were females (70.5%) in general as shown in figure -2-.

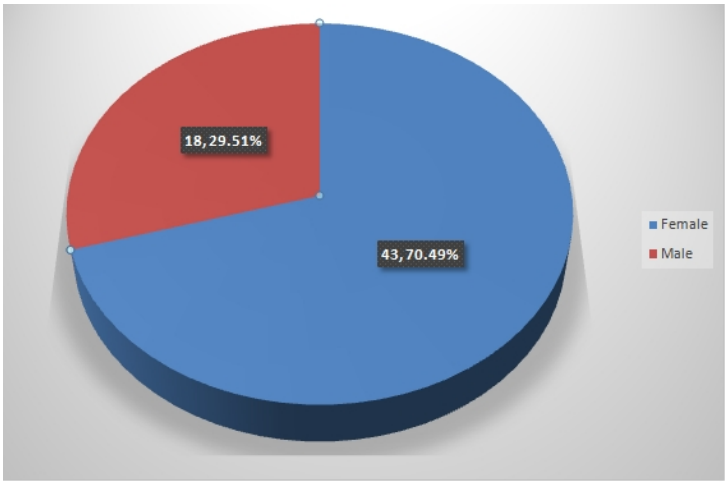


Figure 2: Sex distribution of patients

Causes of pulmonary hypertension in those patients detected to have systolic pulmonary pressure more than 36 mmHg(41 patients, 67.2%) are referred in figure -3- according to Dana point updated clinical classification of pulmonary hypertension in 2013[3]. From which 28 were females (68.3%) & 13 were male (31.3%)

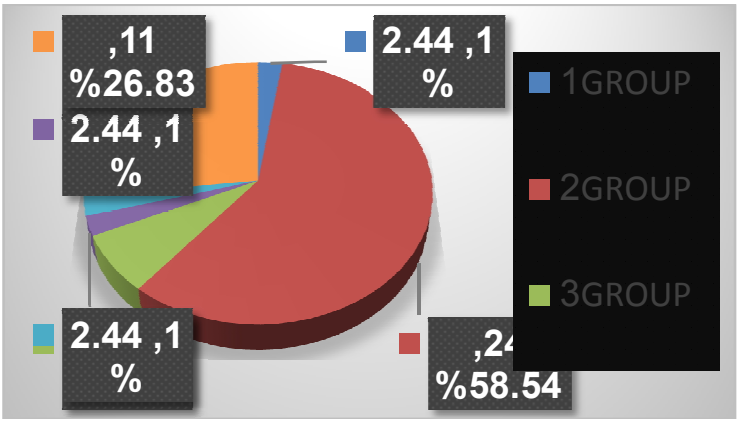


Figure 3: Causes of pulmonary hypertension

TR velocity ranged from 1.5– 5.6 m/sec ; mean 3.01 ± 0.7 m/s, mean of pulmonary systolic arterial pressure (PSAP) & mean pulmonary arterial pressure (MPAP) derived from tricuspid regurgitation done was 44.9±20.2 mmHg & 32.1±14.2 respectively,The mean value of pulmonary artery acceleration time (PAAT)was 106.89± 27.5 ms& mean of PAAT derived MPAP was 30.9 ± 12.35 mmHg illustrated in Table 1- showing the mean values and ranges for all TR-derived and pulmonary artery–derived flow variables including demographic parameters.

Table 1: Mean values and ranges for all TR-derived and pulmonary artery–derived flow parameters including demographic variables

	Mean	**Minimum**	**Maximum**	**Standard Error of Mean**	**Standard Deviation**
Age (years)	55.1	18	85	2.25	17.57
Heart rate(beat /min)	88.49	60	130	2.12	16.55
TR maxvelocity (m/s)	3.01	1.5	5.6	0.09	0.7
TR - derived (PSPAP mm Hg)	44.91	14	138	2.59	20.21
TR-derived-MPAP mmHg	32.11	11	100	1.82	14.2
PAAT (ms)	106.89	42	160	3.51	27.45
PAAT derived –MPAP mmHg	30.9	7	60.1	1.58	12.35

The correlation of pulmonary artery acceleration time (PAAT) to pulmonary systolic pressure shows a significant inverse correlation **(r=-0.693)** (p=0.001) as shown in figure-4.

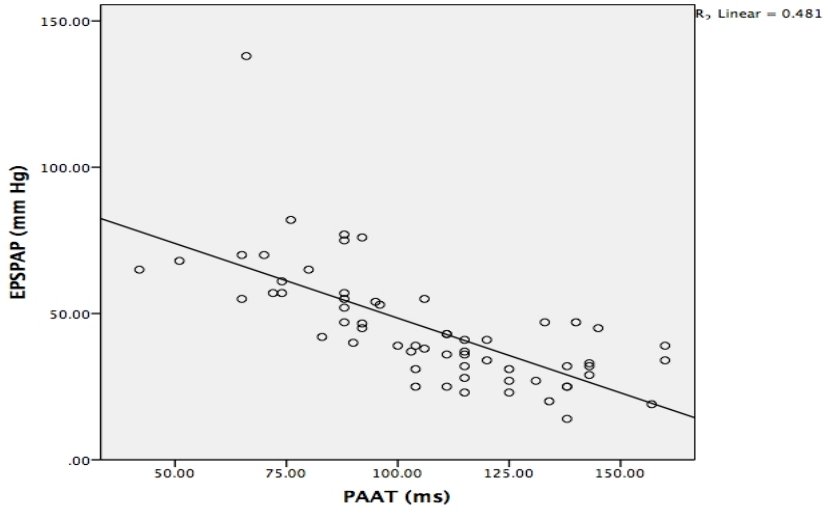


Figure 4: Correlation between TR derivedpeak systolic pulmonary artery pressure (SPAP) & pulmonary artery acceleration time (PAAT)

The sensitivity and specificity of pulmonary acceleration time in detection of pulmonary hypertension (using the value of 100 ms as a cut off value) found sensitivity of 100% and specificity of 63.1%, with a negative predictive value of 100% &positive predictive value of 62.16 % as shown in **table -2.**

Table 2: Correlation between Pulmonary artery acceleration time (PAAT) & systolic pulmonary artery pressure including sensitivity & specificity of PAAT 100 as a cut off value in detecting pulmonary hypertension

		SPAP		Total
		Normal	Abnormal	
PAAT	Normal	23	14	37
	%	62.16%	37.84%	100.00%
	Abnormal	0	24	24
	%	0.00%	100.00%	100.00%
Total		23	18	61
P value		<0.001		
Effect size		Value (95% CI)		
Sensitivity		100.00 (85.69 to 100)		
Specificity		63.16 (47.28 to 76.62)		
Positive Predictive Value		62.16 (46.1 to 75.94)		
Negative Predictive Value		100 (86.2 to 100)		

The correlation of PAAT derived MPAP to TR derived MPAP showed significant correlation as shown in figure-5-
(r = 0.714)P<0.001

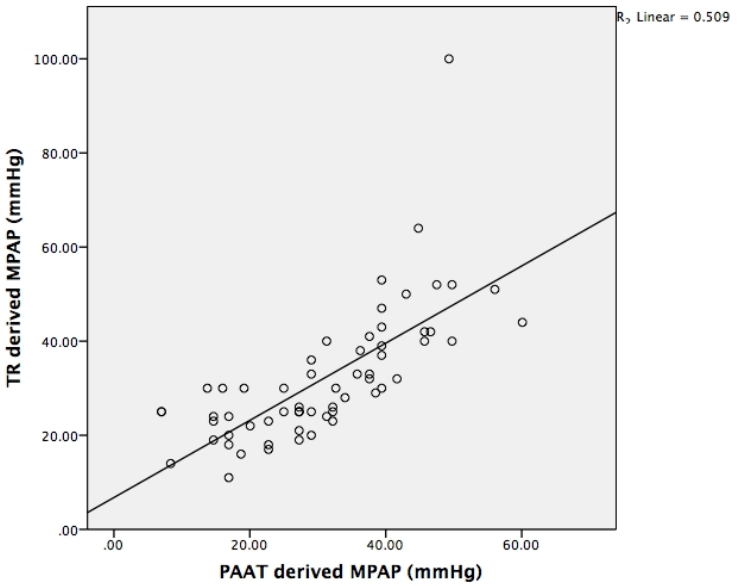


Figure 5: Correlation between TR derived mean pulmonary artery pressure (MPAP) &PAAT derived MPAP

PAAT <100 ms was more useful in detecting(14/15) moderate &(5/5) severe pulmonary hypertension P<0.001i.e pulmonary systolic pressure more than 45 mmHg as in table -3-

Table 3: Diasgnostic PAAT value in detecting different levels of PHT

		PSPAP				Total
		Normal < 36mmHg	Mild PHT 36-45 mmHg	Moderate PHT 46-60 mmHg	Severe PHT >60 mmHg	
PAAT	Abnormal (<100ms)	0	5	14	5	24
	%	0.00%	27.78%	93.33%	100.00%	39.34%
	Normal (≥100 ms)	23	13	1	0	37
	%	100.00%	72.22%	6.67%	0.00%	60.66%
Total		23	18	15	5	61
p value		<0.001				

Discussion

The most commonly accepted transthoracic echocardiography method for the estimation of pulmonary artery pressures relies on the measurement of TRVmax. However, TR is frequently not enough to perform this measurement, as demonstrated by the fact that 25% of patients in one randomly selected clinical cohort had insufficient TR for pulmonary artery systolic pressure determination[18], so short acceleration time may be the only evidence of pulmonary hypertension and may lead to further evaluation of pulmonary artery pressure.

Pulmonary hypertension was found more in females in our study (more than two thirds of those with pulmonary hypertension). Previous literature is divided

on this point, as some studies have found differences and others have not [19-23].

Regarding the causes of pulmonary hypertension, group 2 due to left heart diseases according to Dana point classification [3]including (systolic & diastolic left ventricle dysfunctions, hypertension—etc)was more common in our study than other causes and this and this coincides with other literatures[24,25].

In our study, we found that PAAT had a statistically significant association with PSPAP measured by TRVmax, r=- 0.69 and p=0.001, that is consistent with other studies, as inYared et al[26], which observed a strong, inverse correlation between PAAT and PSPAP among patients with a wide range of PSPAP values (r= -0.95). Quantification of this relationship by linear regression led to the derivation of an equation by which PAAT can be used to provide PSPAP values comparable with those obtained using TRVmax, That present a fact, TR-independent approach to the quantitative assessment of SPAP that appears to perform well across a wide range of pulmonary artery pressures.

The inverse relation between PAAT and PSPAP measured by TTE is apparent with the fact that patients with PHT have PAAT less than 100ms showed significant association between this value and PSPAP (TR-derived) p< 0.001, which can be used as cut off value for noninvasive assessment of PHT in patient with difficulty to measure TRV max with a sensitivity of 100% and specificity of 63.1%, with a negative predictive value of 100%and positive predictive value of 62.16 %

A 100 msec was chosen as a cut off value in our study as It has been shown that measurement of the pulmonaryartery acceleration time (PAAT) through Pulsed-wave Doppler interrogation of the RV outflow tract usually which reveals an acceleration time of <100 msec, that reflects abnormal MPAP[13-16].

In Granstam et al[27],which used the cut-off for PAAT of 100 ms to detect PSPAP of 38 mmHg (for suspecting PH) resulted in a sensitivity of 89% and specificity of 84%. In our study we studied the sensitivity and specificity PAAT of 100 msin relation to PSPAP(TR-derived) and found that all patients in the with PAAT less than 100 ms had PSPAP more than 36mmHg, with sensitivity of 100%, but only around 62 % of those with PAAT more than 100 ms had SPAP less than 36 mmHg which means that a group of patients with PAAT more than 100 ms had PSPAP more than 36 mmHg and in our study we found that those with mild PHT i.e. PASP = 36-45 mmHg72% of them had PAAT more than 100 ms& 28% with PAAT less than 100 ms as in TABLE -2- while those with moderate& severe PHT,14/15&5/5 had PAAT less than 100ms respectively as shown in TABLE -2-, P<0.001. This finding means that PAAT less than 100 s is more appropriate for detectionof moderate & severe PHT than mild PHT.other explanation of the low specificity is the small volume sample.

Regarding The relation of PAAT derived MPAP to TR derived MPAP in our study,itshowed significant correlation as shown in figure-5-P<0.001 (r = 0.714).

Kitabatake et al[28] which state that PAAT correlates well with MPAP and Its logarithm (r=0.82 and0.88, respectively)as determined by cardiac catheterization and alternatively PAAT derived MPAP showed significant correlation with TR derived MPAP in our study &this agrees with Dabestani et al[29], which described that same relationship after converting PAAT into MPAP by the following equation: MPAP =73 - (0.42- PAAT).

Bossone et al[30] and Lopez-Candales et al[31] who reported that recording PAAT has good correlation withDoppler estimated pressures as a short acceleration time usually reflects PHT.

Conclusion

PAAT is easily obtainable and is strongly correlated with TRVmax, PSAPand MPAP. PAAT more useful for detection of moderate and severe PHT.

Disclosure

Author have no conflict of interests and the work was not supported or funded by any company.

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