The Estimation of Pulmonary Artery Systolic Pressure in Normal Pregnancy

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ABSTRACT

Objective: To assess the effects of various stages of pregnancy as well as pregnant women age on the level of pulmonary artery systolic pressure.

Methods: One hundred and thirty five healthy pregnant women, aged 18 to 42 years, were included in this study. They underwent voluntary transthoracic echocardiogram measurements of pulmonary artery systolic pressure at rest. Three groups of pregnant women were designed according to gestational age; Group 1 included pregnancies less than 13 weeks of gestation and considered as control group. Group II included pregnancies equal to 13 weeks gestation and less than 28 weeks. Group III included those pregnancies with 28 weeks of gestation up to term. Each group included 45 women. The mean pulmonary artery systolic pressure was calculated for each group. Results for groups two and three were compared separately to the control group. The study population was then regrouped according to women's age; Group A included women aged less than 35 years (n=95), Group B included women aged 35 years and above (n=40). Difference in mean pulmonary artery systolic pressure between groups A and B were studied.

Results: Lower and upper limits of pulmonary artery systolic pressure during pregnancy were found to be 14 and 45 mmHg. Pulmonary artery systolic pressure during pregnancy showed an increase with maternal age after 35. Marginal change was also noted with gestational age; however that was of no statistical significance.

Conclusion: During the different stages of pregnancy, mean pulmonary artery systolic pressure does not alter; however, it may reach high values in healthy asymptomatic women. When interpreting pulmonary artery systolic pressure in pregnancy, age correction may be required since pulmonary artery systolic pressure is found to physiologically increase with increasing pregnant women's age.

Key words: Age, Gestational age, Pulmonary artery pressure, Pregnancy.

JRMS December 2015; 22(4) 78-82: /DOI: 10.12816/0018570

Manuscript received September 25, 2014. Accepted July 5, 2015

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Introduction

The assessment of pulmonary artery pressure is of great clinical importance not only for diagnostic but also for prognostic purposes. Pulmonary arterial pressure is generated by the right ventricle ejecting blood into the pulmonary circulation, which acts as a resistance to the output from the right ventricle. The smaller arteries and arterioles serve as the chief resistance vessels.

Significant respiratory and cardiovascular physiologic changes occur during pregnancy, in addition pulmonary vascular resistance reduces, (1) thus; the increased volume load to the pulmonary circulation does not cause elevation of pulmonary pressure. (2)

A decrease in systemic blood pressure is well recognized during pregnancy; therefore, pulmonary to systemic artery pressure ratio has to be rather higher in pregnancy than in general population.

Normal pulmonary artery systolic pressure in general population is established but there is limited data in the normal pregnant population. This maybe because the 'gold standard' investigation requires the invasive procedure of right heart catheterization. (3)

More recently the non-invasive technique of Transthoracic Echocardiogram (TE) has become an essential investigation in the evaluation of pulmonary arterial pressure and in the diagnosis of pulmonary artery hypertension (PAH). TE can accurately predict mean pulmonary artery pressure. Nevertheless, some studies questioned its accuracy. It may overestimate pulmonary artery systolic pressure (PASP) by more than 10 mm mercury in 50% of patients without PAH and in 30% of patients with PAH.

Echocardiographically determined pulmonary artery systolic pressures greater than 40 mm Hg are considered abnormal. (8) It varies according to age, BMI (8) as well as other factors. (9) A PASP of 35 mmHg is used to define mild pulmonary artery hypertension, (10) a limit taken in non pregnant status.

The determination of PASP can be performed by measuring the gradient from the right ventricle to the right atrium at peak right ventricle systolic pressure. A modified Bernoulli equation is used to calculate PASP. The formula is $4V^2$, where V is the maximum velocity of the tricuspid

regurgitant jet measured by continuous wave Doppler. This is added to an estimate of right atrial pressure. (11-13)

Published data on pregnancy with PH are limited to case reports or series, (14) some authors found no changes compared to the general population. (15)

Data were collected in this study with regard to PASP in different stages of pregnancy and in different age groups, to look for physiological changes that may arise.

Methods

One hundred and thirty five healthy pregnant women were included in the study, aged 18 to 40 years, parity zero to six, in all stages of pregnancy. They presented to the Obstetric Clinic in regular antenatal care visits. All subjects included had no past medical illness; they were having an uneventful singleton pregnancy, with specific complaint outside the benign pregnancy conditions. Women consented to a voluntary Trans-Thoracic Doppler Echocardiography. The cardiac assessment performed by a team comprising of one cardiology physician and two echocardiography operators, it was carried out at rest. Pulmonary artery systolic pressure was estimated using the modified Bernoulli formula (four times tricuspid valve regurgitation velocity squared adding an estimated right atrial pressure assumed to be 5 mm Hg). Study exclusion criteria included having a preconception body mass index of 25 or more, haemoglobin level of 105g/l or less at the echo study time, systolic blood pressure > 130 mmHg and diastolic blood pressure > 80 mmHg. Subjects with abnormal echocardiographic findings such as major valve regurgitation, valve or septal defects and left ventricular function impairment, were excluded from the study. Women with failed measurements of PASP due to improper technique or absence of mitral regurgitation were excluded as well.

The study was designed to measure PASP in 135 healthy pregnancies, 45 women in each trimester of pregnancy.

In the first part of the study, pregnant women were assigned according to their gestational age into three groups with 45 women recruited for each group; Group I: first trimester (up to 12⁺⁶ week), Group II: second trimester (13 to 27⁺⁶

week), and Group III: third trimester (28 and above). The mean PASP was extracted for each group independently. Group one was used as control for groups two and three based on the fact that changes in haemodynamics, although has already started by this time, are not markedly exhibited.

In the second part of the study, pregnant women were divided into two groups according to maternal age; Group A included pregnant women aged less than 35 years, (n= 95) and group B included women aged 35 years and above (n= 40). Difference of mean PASP for the two groups was calculated.

Statistical analysis for means and standard calculated deviations was using CalculationTM software. Mean difference significance was calculated using Answers researchTM statistical calculator. Student T tests for independent samples were used to calculate statistical significance, presuming a normal distribution, independent samples and a similar variance. The test was considered significant at P<0.05.

Results

Mean PASP for gestational age groups, one, two and three were found 26.8, 26.95, and 27.4 mmHg respectively, (Table I). The difference of the mean between control group one and each of the other study groups two and three was found insignificant. Change in PASP in both middle and late trimesters of pregnancy compared to the first trimester was minimal, with no statistical significance.

In the second part of the study, we found 95 women aged less than 35 years, (Group A), and the reminder 40 women were 35 years or above, (Group B). Mean PASP was found 24.38 and 33.42 mmHg for groups A and B respectively, (Table II). The deference of mean PASP between the two groups was calculated. Student T tests for independent samples showed a statistical increase of PASP in pregnant women 35 years or older when compared to their counterpart less than 35 years. Test was considered significant at P value <0.05.

Table I: Mean pulmonary artery systolic pressure / trimester of pregnancy

	Group1 (1st	Group2	Group 3
	trim.)	(2 nd trim)	(3d trim.)
	n=45	n=45	n=45
Mean	26.8	26.95	27.44
S.D	6.78	7.49	8.08
Variance SD	45.98	56.17	65.34
Population	6.70	7.41	7.99
SD			

S.D: Standard deviation

Table II: Mean pulmonary artery systolic pressure / age for groups A and B $\,$

	Group	Group	T-	Significance
	A	B (≥35	Value	C
	(<35	y.)		
	y)	n=40		
	n=95			
Mean	24.38	33.42	-7.67	P<0.05
S.D	6.13	6.28		
Variance	37.66	39.53		
S.D				
Population	6.10	6.20		
S.D				

SD: Standard deviation.

Discussion

Pregnancy is well recognized to be associated with several unique physiological changes; (16,17) cardiovascular system experiences most of these changes. During pregnancy, there is a 30-50% increase in blood volume, a 50% increase in cardiac output, and a significant reduction in systemic vascular resistance is observed. These physiological changes impose additional burden on the heart but these are generally well tolerated by healthy pregnant women. The increase in cardiac output and the decrease in vascular resistance affects both the systemic and pulmonary circulation compliance but in a different way. In the pulmonary arterial system compliance is distributed over larger arterial tree. This distribution depends on the number of peripheral vessels, which is approximately 8-10 times more in the pulmonary system than the systemic tree. (18)

The adaptation process in pregnancy is well recognized. The relative decrease in systemic blood pressure is well understood; nevertheless, pulmonary circulation is less studied. While some

studies showed no change in pulmonary artery pressure during pregnancy, normal ranges are still to be established.

We estimated PASP by Doppler Echocardiogram. It is usually used to screen patients suspected of PH⁽¹⁹⁾ as a useful substitute for invasive means to measure mean pulmonary artery pressure as shown in some studies. ⁽²⁰⁾

The results of this study show that PASP within population our pregnant ranges between 14mmHg and 45mmHg. Women with relatively high readings were asymptomatic and completed uneventful pregnancies. When the mean was calculated in different groups it was found, in concordance with other studies, that PASP did not differ in pregnancy compared to non pregnant status. Furthermore, the change in different stages of pregnancy was not revealed. Under normal conditions, pulmonary vascular resistance is 1/20 of systemic vascular resistance. (21) The systemic hypotension due to decreased vascular resistance is recognized during pregnancy; therefore, m-PASP/ mean systemic BP ratio is relatively increased. This may help explain the shortness of breath, commonly a major complaint parturient.

Some authors⁽²²⁾ stated an increase in PASP in the general population with increasing age. While similar results were established in this study, we found that this increase was detected in pregnancy starting in a younger age group. Larger studies however are needed to verify this finding.

In different occasions during this study, few women with elevated PASP demonstrated an effortless pregnancy course without any objective complaints. This may be explained either by faults in measuring PASP due to technical circumstances or the fact that higher upper range of PASP is physiologically accepted in pregnancy. This may leave the impression that hyper dynamic circulation of pregnancy may physiologically lead to higher pulmonary artery pressure in some cases. Nevertheless this needs further assessment.

The correlation between PASP and body mass index is recognized in general population. (8) It is not known if the increase in body weight during pregnancy has an effect on PASP. Larger studies are needed to establish this correlation.

Conclusion

In pregnancy, PASP does not demonstrate major changes when compared to systemic circulation. The resultant increase of m-PASP/ mean systemic BP ratio needs to be assessed in a clinical contest. Upper values for PASP may be found in healthy asymptomatic women.

Whilst the gestational stage of pregnancy apparently has no obvious effect on the variation in PASP, a statistically significant increase in PASP was found with increasing maternal age. This may suggest that a correction should be made for maternal age when interpreting the normal values. Larger studies are needed to establish the normal haemodynamics of pulmonary artery blood flow in pregnancy and the correlations between PASP and other variables.

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