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Measurements of cardiac function in a prospective cross-sectional study of pregnant women between 10 and 39 weeks of gestation using a computerized brachial blood pressure cuff

Abstract

Background: Pregnancy requires substantial cardiovascular adaptation to support maternal–fetal health but also increases the risk of hypertensive disorders, preeclampsia, and fetal growth restriction. Routine vital signs do not assess stroke volume, cardiac output, or total peripheral vascular resistance. Non-invasive pulse-wave analysis using a computerized brachial cuff can measure these parameters, yet pregnancy-specific reference intervals are lacking.

Objective: To establish gestational age–specific reference intervals and z-score equations for key hemodynamic parameters between 10 and 39 weeks' gestation and to evaluate the effect of maternal position.

Study design: In this prospective cross-sectional study, 300 pregnancies without hypertension, diabetes, fetal growth restriction, or incomplete data were analyzed. Hemodynamic measurements were obtained in the supine, left lateral, and sitting positions. Fractional polynomial regression using gestational age as the independent variable was used to derive means, standard deviations, percentiles, and z-scores. Positional differences were assessed using nonparametric tests.

Results: Heart rate increased from early pregnancy to the early third trimester and declined toward term. Stroke volume and stroke volume index decreased progressively across gestation. Cardiac output and cardiac index peaked at mid-gestation and declined near term. Total peripheral vascular resistance decreased through mid-pregnancy, reached a nadir in early third trimester, and increased at term. Compared with supine and sitting positions, the left lateral position was associated with lower brachial and central blood pressures, higher stroke volume, and lower vascular resistance, while cardiac output was similar across positions. Gestation-specific centiles and z-score equations were generated for all parameters.

Conclusion: This study establishes pregnancy-specific hemodynamic reference intervals using a computerized brachial cuff–based device. The observed gestational trends are consistent with prior studies and support left lateral positioning for hemodynamic assessment. These reference standards may facilitate improved cardiovascular risk assessment and individualized management in pregnancy.

Keywords: stroke volume, stroke volume index, cardiac output, cardiac index, total peripheral vascular resistance, pregnancy, fetal growth restriction, hypertension, vicorder

Volume 16 Issue 6 - 2025

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Received: December 05, 2025 | **Published:** December 19, 2025

Introduction

Assessment of maternal cardiac function is increasingly important in pregnancy, particularly for managing hypertension and identifying women at risk for preeclampsia, fetal growth restriction, and other adverse perinatal outcomes.[1–37] Pregnancy is associated with progressive and substantial changes in maternal hemodynamics, including alterations in cardiac output and systemic vascular resistance, that evolve across gestation.[38–40] Despite this, routine clinical assessment is largely limited to heart rate and brachial blood pressure, which do not capture key aspects of cardiovascular function needed to guide treatment decisions or stratify cardiovascular risk.[21,41]

Non-invasive assessment of maternal hemodynamics has therefore gained increasing attention. In 2019, the International Working Group on Maternal Hemodynamics reviewed available invasive, minimally invasive, and non-invasive techniques for measuring cardiac function in pregnancy.[42] The group emphasized that validation against reference standards such as thermodilution, direct Fick, or MRI is difficult in pregnant populations due to logistical and cost constraints. Although transthoracic echocardiography is commonly used and widely regarded as a reference method for assessing cardiac output and vascular resistance, it is not well suited for routine screening and is subject to operator variability.[42]

Importantly, the Working Group highlighted several limitations of current non-invasive approaches. Different technologies rely on distinct physiological assumptions and should not be used interchangeably. Most critically, pregnancy-specific reference intervals that account for gestational age and maternal characteristics are lacking for many devices, limiting their clinical applicability.

Among non-invasive techniques, the UltraSonic Cardiac Output Monitor (USCOM®) is the most extensively studied in both normal

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and high-risk pregnancies.[2–8,10–24,26,27,29,38–41,43–51] Studies using this device have reported clinically relevant hemodynamic patterns associated with adverse maternal and fetal outcomes (Table 1).[3,5,8,10,13–15,19,20,22,24,27,29,41,45,47–49,51,52]

Table 1 Studies of maternal cardiac function using the USCOM® device

Authors	Study	Number pregnant patients studied	Weeks studied	Findings
Kager,[51]	Compare USCOM® with transthoracic echocardiography	172	1st to 3rd Trimesters	Hemodynamic profiles were in line with data published in the literature.
Khalil[29]	Compared pregnant and non-pregnant patients	185	1st to 3rd Trimesters	In the first trimester, all the following vascular parameters were higher in pregnant women compared to non-pregnant controls: cardiac output, cardiac index, stroke volume, stroke volume index. Pregnant women had significantly lower total peripheral vascular resistance than non-pregnant controls.
Lo Presti,[52]	Assessment of total peripheral vascular resistance and total body water in normotensive women during the first trimester of pregnancy to predict hypertensive complications	120	1st Trimester	When patients were stratified by total peripheral vascular resistance < or > 1200 dynes/sec/cm5, there was no significant difference in total body water.
Lo Presti,[53]	1st Trimester	160	1st Trimester	Elevated total peripheral vascular resistance might indicate an abnormal vascular adaptation already in the first weeks of pregnancy. Moreover, in women who undergo an abortion, elevated total peripheral vascular resistance could be used to distinguish genetic or environmental causes of miscarriage.
McNamara,[49]	Compared USCOM® with 3D echocardiography	85	>25 weeks of gestation	USCOM® has acceptable agreement with 3D-TTE for the measurement of cardiac output in pregnancy.
Valensise,[27]	Evaluate patients with threatened preterm delivery	68	Pre-term Labor	Women with a diagnosis of preterm delivery showed total peripheral vascular resistance values of > 1000 dynes/sec/cm5.
Mulder,[45]	USCOM® vs transthoracic echocardiography (TTE)	77 to 106	12 to 30 weeks	Mean cardiac output in USCOM® was 0.6 L/min higher compared to TTE in all trimesters; percentage error ranged from 35% to 45%.
Perry,[22]	Hypertensive patients	231	3rd trimester	Compared with controls, women with preterm hypertension had significantly lower cardiac output and significantly higher systemic vascular resistance.
Perry,[19]	Evaluation of maternal hemodynamic function in growth restricted (FGR) and small for gestational age fetuses (SGA)	102 FGR, 64 SGA	3rd trimester	Pregnancies complicated by FGR presented with impaired maternal hemodynamic function, as evidenced by lower cardiac output, as well as higher mean arterial pressure, total peripheral vascular resistance and uterine artery resistance. Pregnancies delivering an SGA neonate, without evidence of FGR, had normal maternal hemodynamic function.
Kalafat,[20]	Prediction of fetal distress in patients undergoing induction of labor	27	3rd trimester	Increased total peripheral vascular resistance and decreased cardiac output occurred in patients who developed fetal distress in labor requiring operative delivery.
Farsetti,[15]	Systemic vascular resistance associated with fetal growth restriction (FGR)	24	29	Total peripheral vascular resistance > 1006 dynes/sec/cm5 identified all fetuses with FGR.
Mappa,[14]	Predict adverse outcome	133	37 to 39 weeks	Pre-labor modifications of maternal cardiovascular variables (stroke volume<50 ml, increased total peripheral vascular resistance) are associated with adverse perinatal outcome.
Ornaghi,[10]	Identify early and late fetal growth restriction (FGR)	102	32 weeks or at time of diagnosis	No difference in cardiac findings between early and late onset FGR. However, patients with hypertension and FGR vs. isolated FGR showed increased total peripheral vascular resistance lower cardiac output.
Mecacci,[13]	Gestational diabetics mellitus (GDM)	69	17 to 39 weeks	GDM group had significantly lower values of cardiac output and stroke volume than controls from the early third trimester (26-30 weeks) until term.

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Table 1 Continued...

Montaguti,[8]	1st trimester evaluation	187	1st trimester	Cardiac output lower and total peripheral vascular resistance higher in patients who developed fetal growth restriction in the 3rd trimester.
Farsetti,[5]	Dichorionic (DC) and monochorionic (MC) pregnancies5	40 MC 35 DC	1st to 3rd Trimesters	In DC pregnancy the maternal hemodynamics remain stable as manifest by higher cardiac output and lower total peripheral vascular resistance in the first-trimester when compared to singleton pregnancies. In contrast, in MC twin pregnancy, the rise in maternal cardiac output continues in the second trimester.
Lihme,[41]	Created reference ranges	405	28 to 40 weeks	Maximum cardiac output and stroke volume values were measured in gestational weeks 30-32 and decreased over the third trimester, whereas systemic vascular resistance increased during the same period.
Mangos,[3]	Predict preeclampsia (PE)	137	2nd and 3rd Trimesters	Unable to successfully predict the onset of PE or severe PE based on hemodynamic parameters.
Tiralongo,[48]	Predict preeclampsia	100	1st trimester	High total peripheral vascular resistance (>1200 Dynes/sec/cm5 during the first weeks of gestation may be an early marker of cardiovascular maladaptation.
Vinayagam,[47]	Comparing USCOM® with NICOM®	598	3rd trimester	USCOM® with NICOM® cannot be used interchangeably.
Vinayagam,[24]	Evaluation of morbidly obese pregnant women	23	2nd and 3rd Trimesters	Stroke volume index and cardiac index decreased, and total peripheral vascular resistance increased compared to controls.

*= dynes·sec·cm^{-5}

However, its use is limited by operator dependency, the need for specialized training, assumptions regarding aortic dimensions, and the requirement for a dedicated and relatively expensive stand-alone system.

The Vicorder® device offers an alternative non-invasive approach based on brachial cuff measurements combined with automated waveform analysis. To date, it has not been evaluated for assessment of cardiac function in pregnant patients, and pregnancy-specific reference intervals are not available. In the absence of a non-invasive gold standard for direct comparison, the primary aim of this study was to establish gestational age–specific reference intervals for cardiac function parameters measured with the Vicorder® device. Using fractional polynomial analysis, percentile curves were created and a z-score calculator to support clinical interpretation and potential use of this device in pregnancy.

Materials and methods

Study design and ethical approval

This was a prospective, cross-sectional study conducted from October 2023 to March 2024 at three maternal-fetal medicine outpatient clinics located in Los Angeles County, California, USA. The study protocol was approved by Pearl Institutional Review Board (Protocol 22-FEDC-102; approval date: January 7, 2023), and all participants provided oral informed consent prior to enrollment, in accordance with IRB guidelines and institutional policy.

Participants and sampling methodology and patient exclusion

A total of 330 pregnant patients were enrolled in the study. Thirty patients were excluded: 6 for elevated mean arterial pressure (>107 mm Hg), 9 for estimated fetal weight <10th percentile, 10 due to duplicate entries, and 5 for incomplete data. Thus, the final analytic cohort included 300 participants (Flow Diagram, Supplement 1). Eligible participants were pregnant individuals aged ≥18 years with singleton gestations between 10 and 39 weeks of gestation who consented for the study. The selected gestational ages were the result of the referral patterns to our facilities. Patients were excluded if they had any of the following at the time of or following the examination:

1) Mean arterial pressure (MAP) >107 mm Hg (equivalent to ≥140/90 mm Hg).
2) Estimated fetal weight <10th percentile or >90th percentile for gestational age determined using diagnostic ultrasound.
3) Preexisting chronic hypertension, diabetes mellitus, or other systemic maternal conditions known to affect cardiovascular physiology.

Study protocol

All patients were examined once only, consistent with the methodological framework for cross-sectional modeling described by Royston and further supported by Silverwood and Cole.[54,55] Exclusion criteria were applied based solely on contemporaneous clinical findings and not on future follow-up data.[56,57]

Cardiovascular measurements

Hemodynamic measurements were obtained using the Vicorder® device (80 Beats Medical. GmbH, Germany), a non-invasive, FDA-cleared instrument that employs plethysmograph pulse wave analysis of the brachial artery via an upper-arm blood pressure cuff. Measurements were performed in a temperature-controlled room (22–24°C) following a 15-minute rest period, with the participant in a relaxed state. Each participant underwent cardiovascular assessment in three body positions: supine, left lateral decubitus, and seated upright. A 3–5-minute interval was observed between positions to allow for hemodynamic stabilization (Figure 1) (Table 2).

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Table 2 Definitions of measurements obtained from the Vicorder® device used in the current study (Figure 1)

Measurements from the Vicorder®	Definition
Column 3 (Figure 1)	
BP	Brachial blood pressure (BP) in mmHg, reflecting the brachial systolic and diastolic pressures.
PP	Pulse pressure (PP) reflecting the brachial pulse pressure, the difference between systolic and diastolic pressure.
HR	Heart rate (HR) in beats per minute (BPM) reflecting the mean heart rate derived from the timing of the brachial waves.
MAP	Mean Pressure (MAP) in mmHg referring to mean arterial pressure, derived as the arithmetic means over the brachial pressure wave, where the mean pressure is supposed to be equal throughout the arterial tree.
CO	Cardiac output in liters/min relates to the non-invasive cardiac output that is calculated by Ohm's law: cardiac output = MAP / total peripheral vascular resistance
SV	Stroke volume in milliliters refers to the cardiac stroke volume that is derived from cardiac output and heart rate stroke volume = cardiac output / HR
Column 4 (Figure 1)	
AoBP	Aortic blood pressure (AoBP) in mmHg, reflecting the aortic central systolic and diastolic pressures, calculated from the global transfer function.
AoPP	Aortic pulse pressure (AoPP) in mmHg reflecting the central aortic pulse pressure, the difference between systolic and diastolic central aortic pressure.
TPR	Total peripheral vascular resistance in Peripheral Resistance Units (PRU) referring to the estimation of total peripheral vascular resistance derived from the post systolic and diastolic portion of the central pressure wave.*

*The units for total peripheral vascular resistance (PRU) are expressed as pressure (mmHg) divided by cardiac output (mL/min), or mmHg·min·mL^{-1}, which is sometimes abbreviated as peripheral resistance units. Alternatively, total peripheral vascular resistance may be expressed in centimeter-gram-second (cgs) units as dynes·sec·cm^5, where 1 mmHg = 1,330 dynes/cm^2 and flow (cardiac output) is expressed as cm^3/sec. The total peripheral vascular resistance value in peripheral resistance units can be converted to a corresponding value in cgs units by multiplying the peripheral resistance units value by 1333.[58] Although the cgs units are less intuitive, many clinical and experimental studies still express total peripheral vascular resistance in those units.

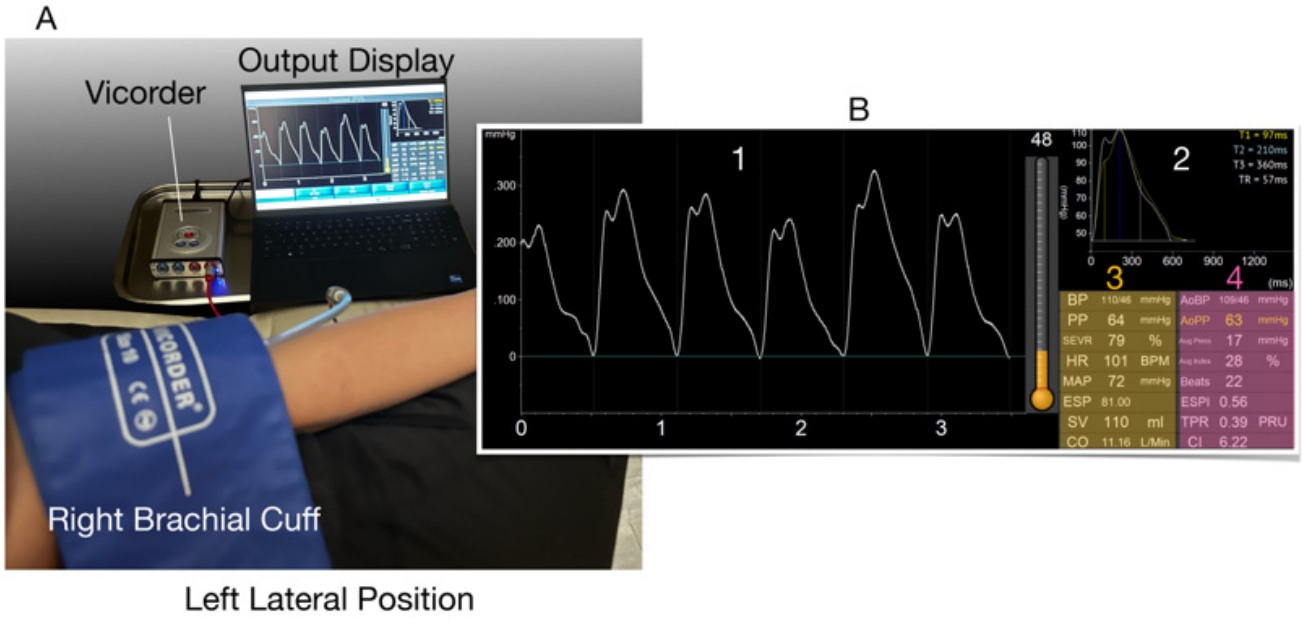


Figure 1 Analysis of the Brachial Waveforms Using the Vicorder® Device. (A) On the left side of the image the blood pressure cuff is placed on the right upper arm of a patient lying in the left lateral position. The Vicorder® recorder is attached to the blood pressure cuff and interfaces with the computer program that computes the measurements displayed on the computer screen. (B) is an enlarged image of the measurements obtained from the Vicorder® analysis. (1) On the left side of the screen the real time brachial pressure waves are displayed. (2) The right part of the panel shows the mean brachial wave (white color trace) averaged from previously recorded brachial pressure waves (1). While pressure waves on the left reflect the plethysmographic cuff reading, represented by a low pressure of only tenths of mmHg, the mean brachial wave in the right panel is scaled to systolic and diastolic pressure (mmHg). The yellow trace in B indicates the central aortic pressure wave derived from the brachial wave by applying the global transfer function. A yellow vertical cursor (dashed yellow line) is placed at the "shoulder notch", where the incident wave originating from the heart is superimposed by the reflected wave from the body. The blue line indicates peak systolic pressure of the central wave while the white cursor is set at the end-systolic point, separating systolic and diastolic wave sections. On the upper right side of this panel, the times T1 through T3 are given corresponding to the time related location of yellow, blue, and white cursors. TR gives the estimated Reflection Time. The right lower panel provides the computed measurements (3 and 4) (Table 2).

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All measurements were performed by two trained operators, certified in use of the Vicorder device and blinded to clinical data, to minimize operator variability.

The following cardiovascular parameters were recorded:

1) Heart rate
2) Brachial and central aortic systolic, diastolic, and mean arterial pressure
3) Pulse pressure
4) Stroke volume and stroke volume index
5) Cardiac output and cardiac index
6) Total peripheral vascular resistance (TPVR).

Calculation of total peripheral vascular resistance

TPVR was calculated using two methods to assess concordance:

1) Equation 1: TPVR (dynes·s·cm^{-5}) = (MAP × 79.96) / Cardiac Output (L/min)
2) Equation 2: TPVR = 1333 × Peripheral Resistance Units (PRU), where PRU is a direct output of the Vicorder device

The correlation coefficient between the two methods was r = 0.9997, indicating near-complete agreement and supporting their analytic interchangeability. Both values are reported for transparency and comparability with prior literature.

Statistical analysis

Data were analyzed using NCSS 24 (Kaysville, UT, USA) and XLStat (Addinsoft, Denver, CO, USA). For each cardiovascular parameter, fractional polynomial regression models were fitted using gestational age as the independent variable to estimate gestational age-specific means and standard deviations. From these, the 5th and 95th percentiles were computed as:

1) 5th percentile = mean − (1.65 × standard deviation)
2) 95th percentile = mean + (1.65 × standard deviation)

Comparisons between body positions were conducted using the Kruskal-Wallis test, followed by Dunn's post-hoc test for multiple comparisons. A p-value < 0.05 was considered statistically significant.

Results

Baseline characteristics

Table 3 describes the baseline characteristics of the study patients.

Table 3 Baseline characteristics of the study population (N = 300)

Characteristic	Value
Maternal age (years)	31.4 ± 6.6
Gestational age at exam (weeks)	22.3 ± 7.6
Maternal weight (lbs)	164 ± 34
Maternal height (inches)	63.0 ± 3.4
Race/Ethnicity, n (%)	
- Asian	10 (3.3%)
- Black or African American	16 (5.3%)
- Hispanic or Latina	120 (40.0%)
- White	154 (51.3%)

Reference intervals by maternal position

Gestational age-specific 5th, 50th, and 95th percentiles, along with the regression equations for the mean and standard deviation equations, and Z-score equations, are presented for each cardiovascular parameter by position in Supplement 2 (supine), Supplement 3 (left lateral), and Supplement 4 (sitting).

Heart rate

Heart rate increased across gestation, peaked in the early third trimester, then declined slightly (Figure 2).

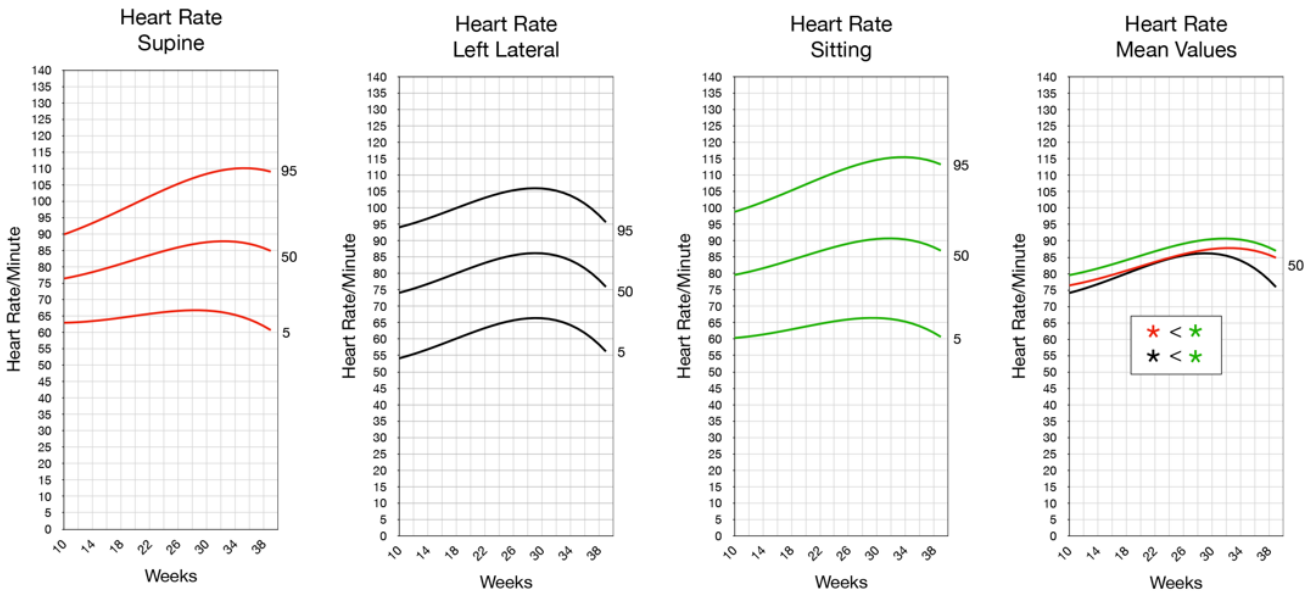


Figure 2 Reference intervals of the 5th, 50th, and 95th percentiles for maternal heart rate in the supine, left lateral, and sitting positions. When comparing the supine with the left lateral position the mean values were lower for the left lateral position after 30 weeks of pregnancy. The image that summarizes the supine, left lateral and supine graphs contains a colored * that represents the supine (red), left lateral (black) and sitting (green) mean results that are significantly different (P<0.05). The sitting position showed significantly higher heart rate compared to supine and left lateral positions (Table 4, p < 0.001).

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Figure 2 illustrates the heart rate from 10 to 39 weeks of gestation in the supine, left lateral, and sitting positions which suggests the heart rate increases, peaks, and then decreases during the 3rd trimester of pregnancy.

Brachial blood pressure

Systolic blood pressure (SBP) decreased until mid-gestation and rose again toward term (Figure 3A). SBP was highest in the sitting position, followed by supine and then left lateral ($p < 0.01$). Diastolic blood pressure (DBP) showed a similar U-shaped trend in the sitting and supine positions but remained stable in the left lateral position (Figure 3B) (Table 4).

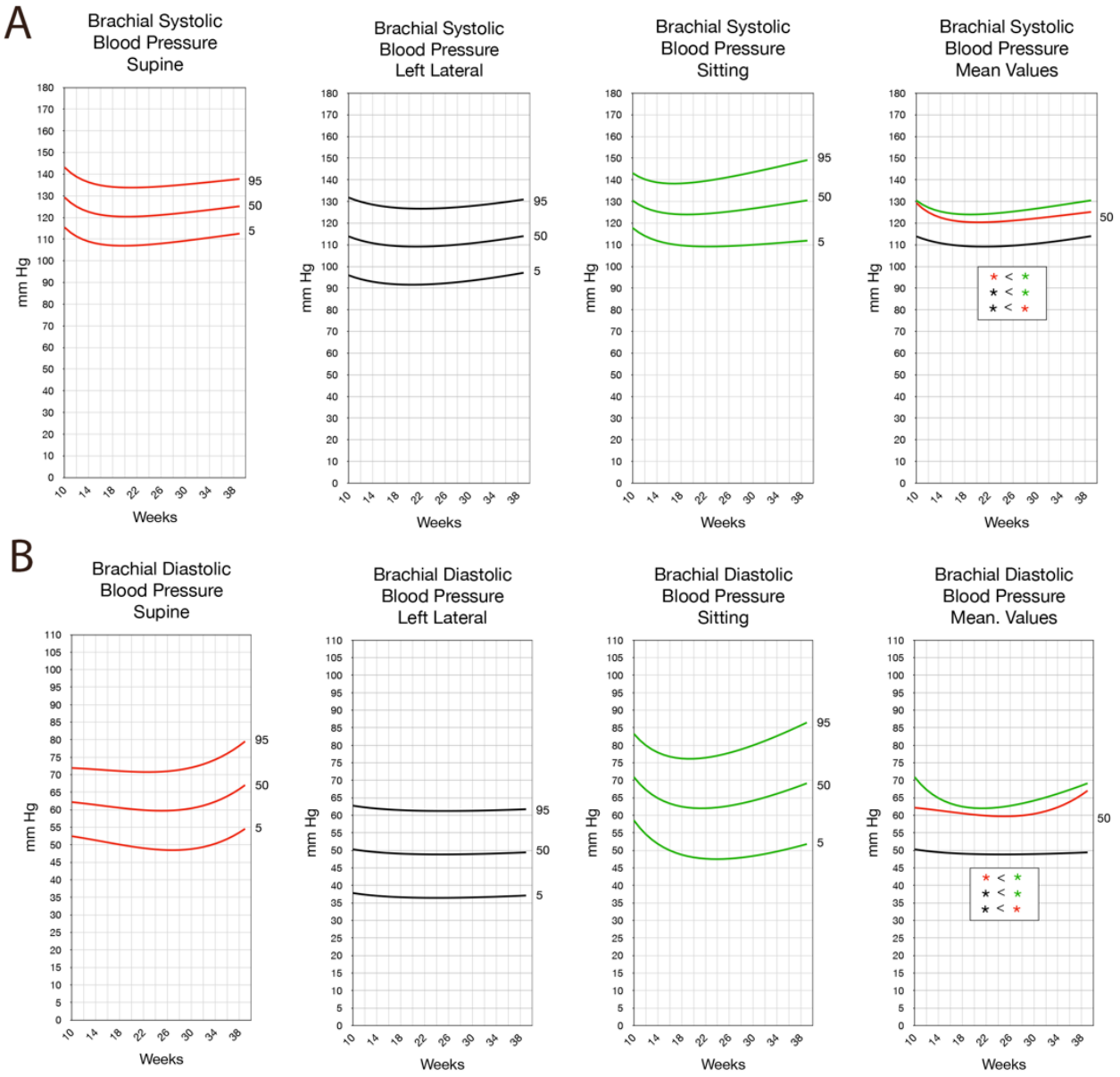


Figure 3 Reference intervals of the 5th, 50th, and 95th percentiles for the brachial systolic (A) and diastolic (B) blood pressures in the supine, left lateral, and sitting positions. When comparing the supine (yellow shaded area) and sitting positions with the left lateral position, the mean values were lower for the left lateral position for both the systolic (A) and diastolic (B) blood pressures throughout pregnancy. The image that summarizes the supine, left lateral and supine graphs contains a colored * that represents the supine (red), left lateral (black) and sitting (green) mean results that are significantly different ($P<0.05$).

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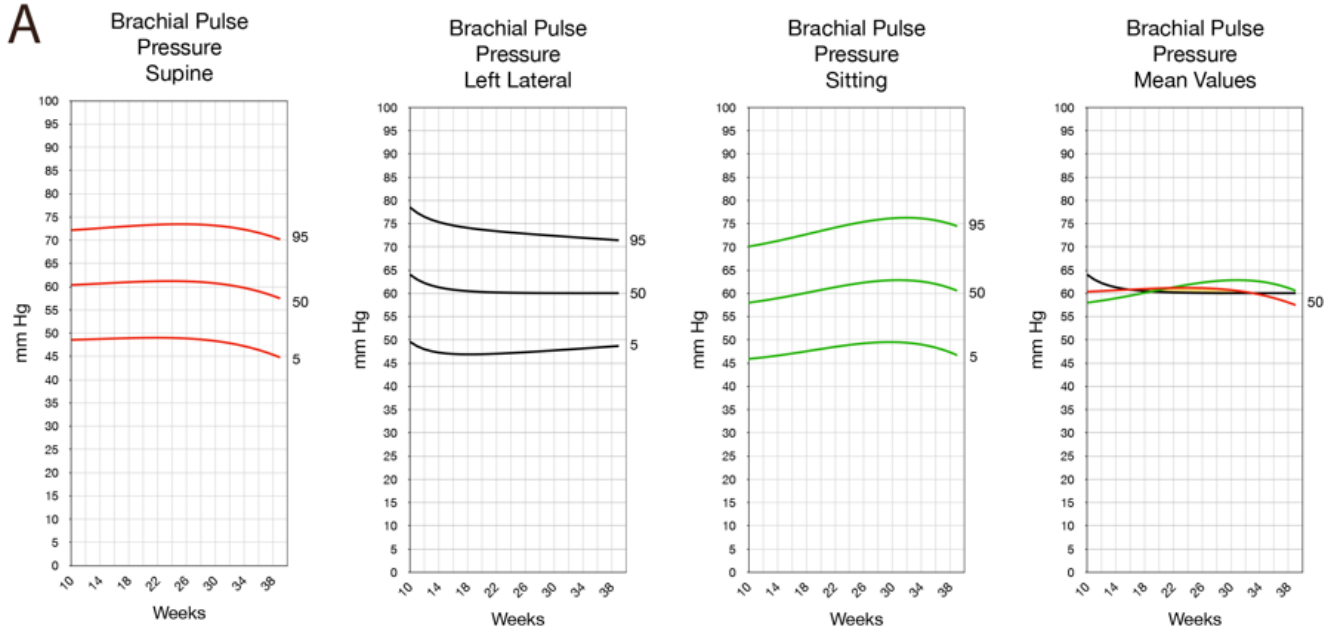
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Table 4 Kruskal-Wallis (Dunn Test) comparing the supine, left lateral and sitting positions cardiac measurements using the Vicorder® device

Measurement	Supine (Mean, SD)	Left Lateral (Mean, SD)	Sitting (Mean, SD)	P-Value Left Lateral Supine vs	P-Value Left Lateral vs Sitting	P-Value Supine vs Sitting
Heart Rate	84 (13)	82 (12)	86 (15)	0.08	<0.001	0.04
Brachial blood pressure						
Systolic (mm Hg)	122 (8.1)	110 (10.4)	126 (11.4)	<0.001	<0.001	<0.001
Diastolic (mm Hg)	61 (6.6)	49 (7.5)	63.8 (10.6)	<0.001	<0.001	0.01
Pulse Pressure (mm Hg)	60.6 (7.4)	60.5 (8)	62.3 (11.8)	0.68	0.25	0.46
Mean Arterial Pressure (mm Hg)	87.4 (6.9)	76.1 (8.4)	90.8 (10)	<0.001	<0.001	0.002
Central aortic blood pressure						
Systolic (mm Hg)	115 (8.7)	106 (10)	119 (11.5)	<0.001	<0.001	<0.001
Diastolic (mm Hg)	61 (6.7)	49 (7.5)	63 (10.6)	<0.001	<0.001	<0.001
Pulse Pressure (mm Hg)	53.8 (7.7)	56.7 (7.9)	55.8 (11.2)	<0.001	0.02	0.15
Mean Arterial Pressure (mm Hg)	70 (4)	67 (7.6)	79 (4)	<0.001	<0.001	<0.001
Ventricular function						
Stroke Volume (ml)	96.3 (15.6)	101.2 (18.3)	94.3 (22.3	0.005	<0.001	0.01
Stroke Volume Index (ml/BSA)	55.5 (12)	59.2 (14.7)	55.5 (12)	0.008	0.008	1
Cardiac Output (L/minute)	8 (1.5)	8..1 (1.8)	8.1 (2.2)	0.92	0.28	0.32
Cardiac Index (Cardiac Output/BSA)	4.6 (1.2)	4.7 (1.3)	5.1 (3.9)	0.87	0.3	0.38
Total peripheral vascular resistance						
Peripheral Resistance Units (mmHg·min·mL-1)	0.65 (0.13)	0.57 (0.14)	0.73 (0.36)	<0.001	<0.001	0.001
dynes/sec/cm5	871 (166)	764 (179)	919 (221)	<0.001	<0.001	0.003

mm, millimeters; Hg, mercury; min, minute; mL, milliliters; BSA, body surface area; L, liters

Pulse pressure declined in late pregnancy in the supine and sitting positions but not in left lateral (Figure 4A). No significant differences were observed between positions ($p > 0.05$). Mean arterial pressure (MAP) increased progressively in the supine and sitting positions but remained stable in the left lateral position (Figure 4B). MAP was significantly lower in the left lateral position compared to supine and sitting ($p < 0.001$) and sitting > supine (Table 4).



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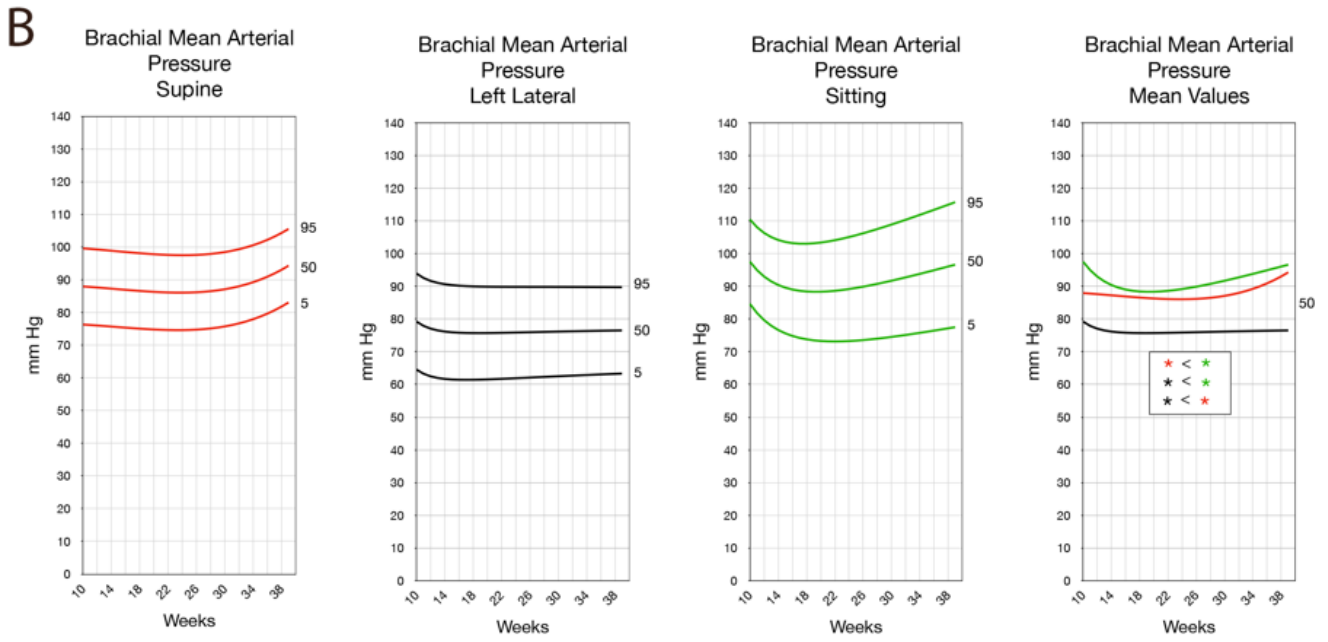
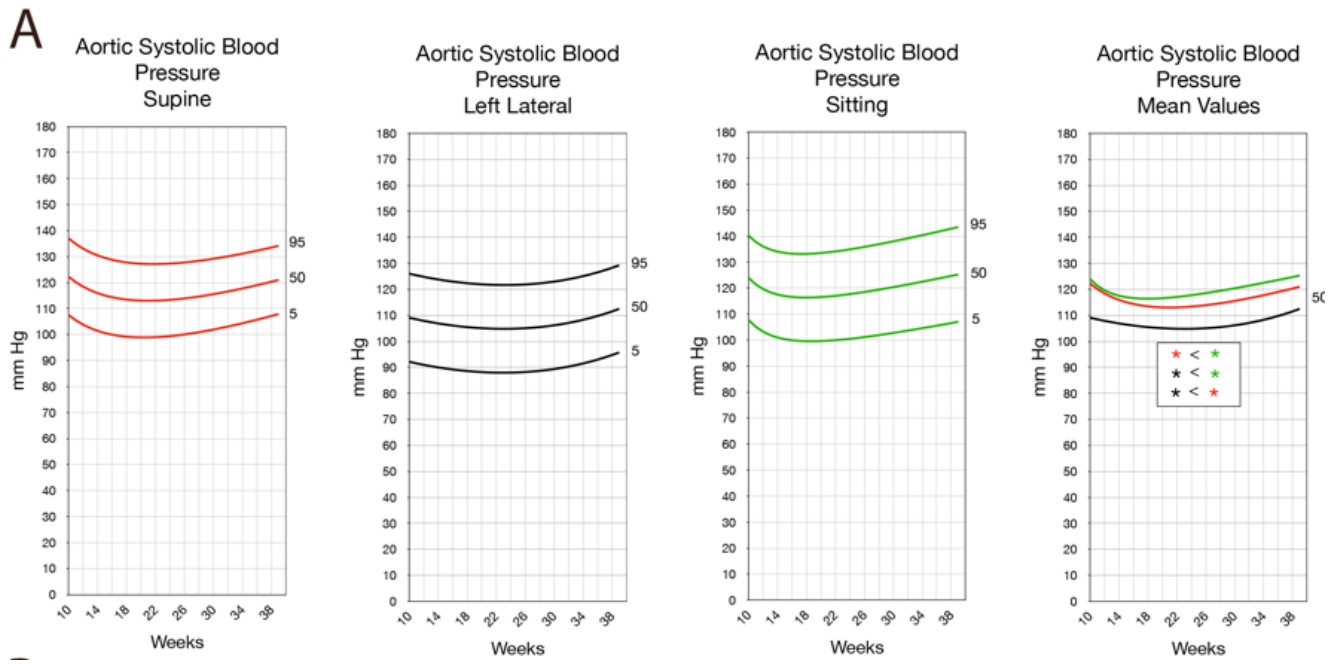


Figure 4 Reference intervals of the 5th, 50th, and 95th percentiles for the brachial pulse pressure (A) and brachial mean arterial pressure (B) in the supine, left lateral, and sitting positions. When comparing the supine (yellow shaded area) with the left lateral position the mean values were lower for the left lateral position for the mean arterial pressure (B) throughout pregnancy. The image that summarizes the supine, left lateral and supine graphs contains a colored * that represents the supine (red), left lateral (black) and sitting (green) mean results that are significantly different ($P<0.05$).

Central aortic blood pressure

Trends in central systolic and diastolic pressures paralleled those of brachial pressures (Figure 5A & 5B). The left lateral position consistently yielded lower values compared to supine and sitting positions ($p < 0.001$). Pulse pressure was lowest in the left lateral position across all trimesters (Figure 6A), and MAP was lowest in left lateral, followed by supine and sitting (Figure 6B) (Table 4).



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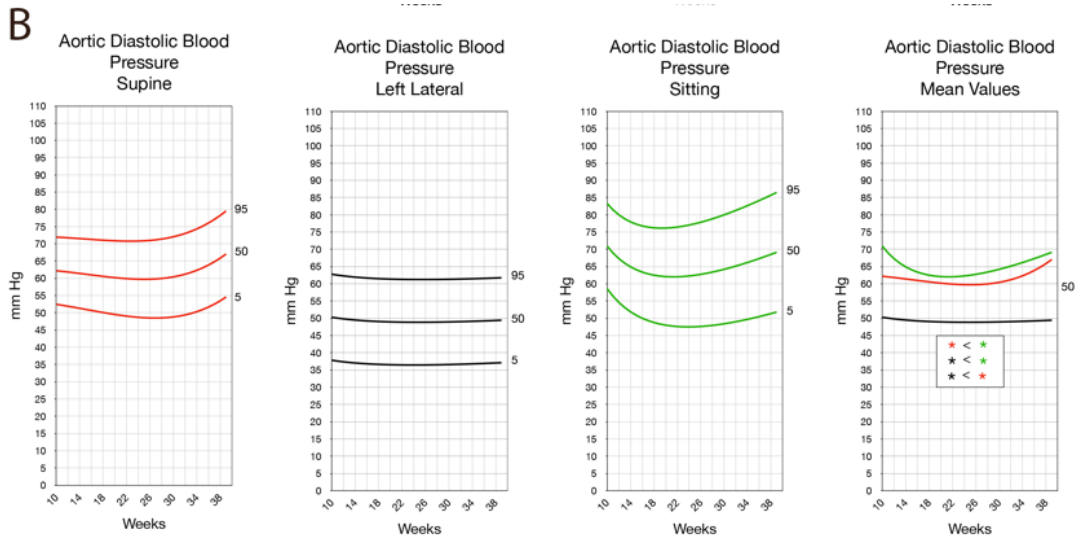


Figure 5 Reference intervals of the 5th, 50th, and 95th percentiles for the aortic systolic (A) and diastolic (B) blood pressures in the supine, left lateral, and sitting positions. When comparing the supine (yellow shaded area) and sitting positions with the left lateral position the mean values were lower for the left lateral position for both the systolic (A) and diastolic (B) blood pressures throughout pregnancy. The image that summarizes the supine, left lateral and supine graphs contains a colored * that represents the supine (red), left lateral (black) and sitting (green) mean results that are significantly different (P<0.05).

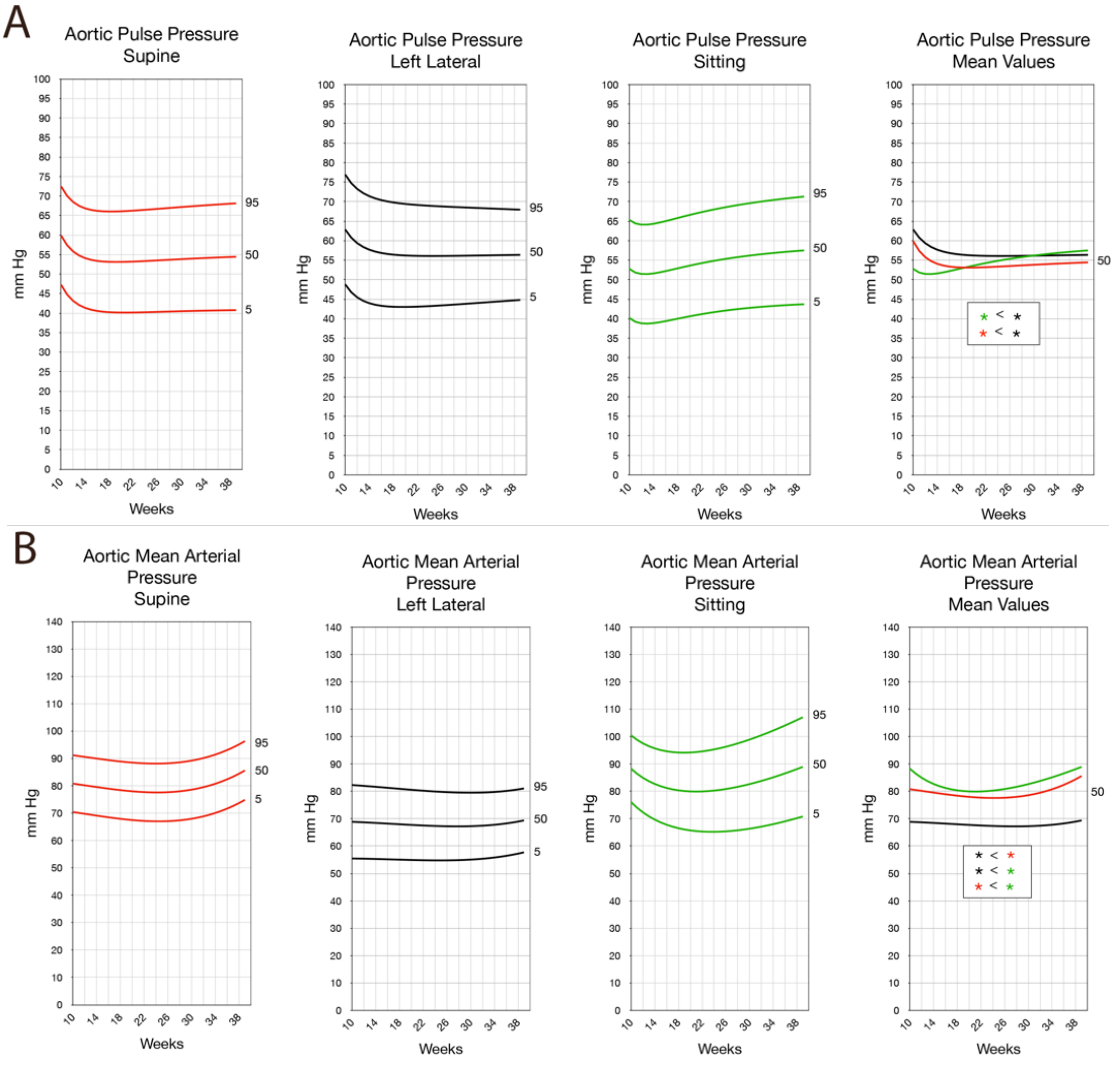


Figure 6 Reference intervals of the 5th, 50th, and 95th percentiles for the aortic pulse pressure (A) and aortic mean arterial pressure (B) in the supine, left lateral, and sitting positions. The pulse pressure was higher in the supine position (blue shaded area) (A) and the mean arterial pressure (B) was lower in the left lateral

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position (yellow shaded area). The image that summarizes the supine, left lateral and supine graphs contains a colored * that represents the supine (red), left lateral (black) and sitting (green) mean results that are significantly different (P<0.05).

Stroke volume and stroke volume index

Stroke volume (SV) declined steadily in the supine position and was relatively stable in the left lateral and sitting positions (Figure 7A). The stroke volume index (SVI) showed a similar pattern, with higher values in the left lateral position across gestation (Figure 7B, $p < 0.05$).

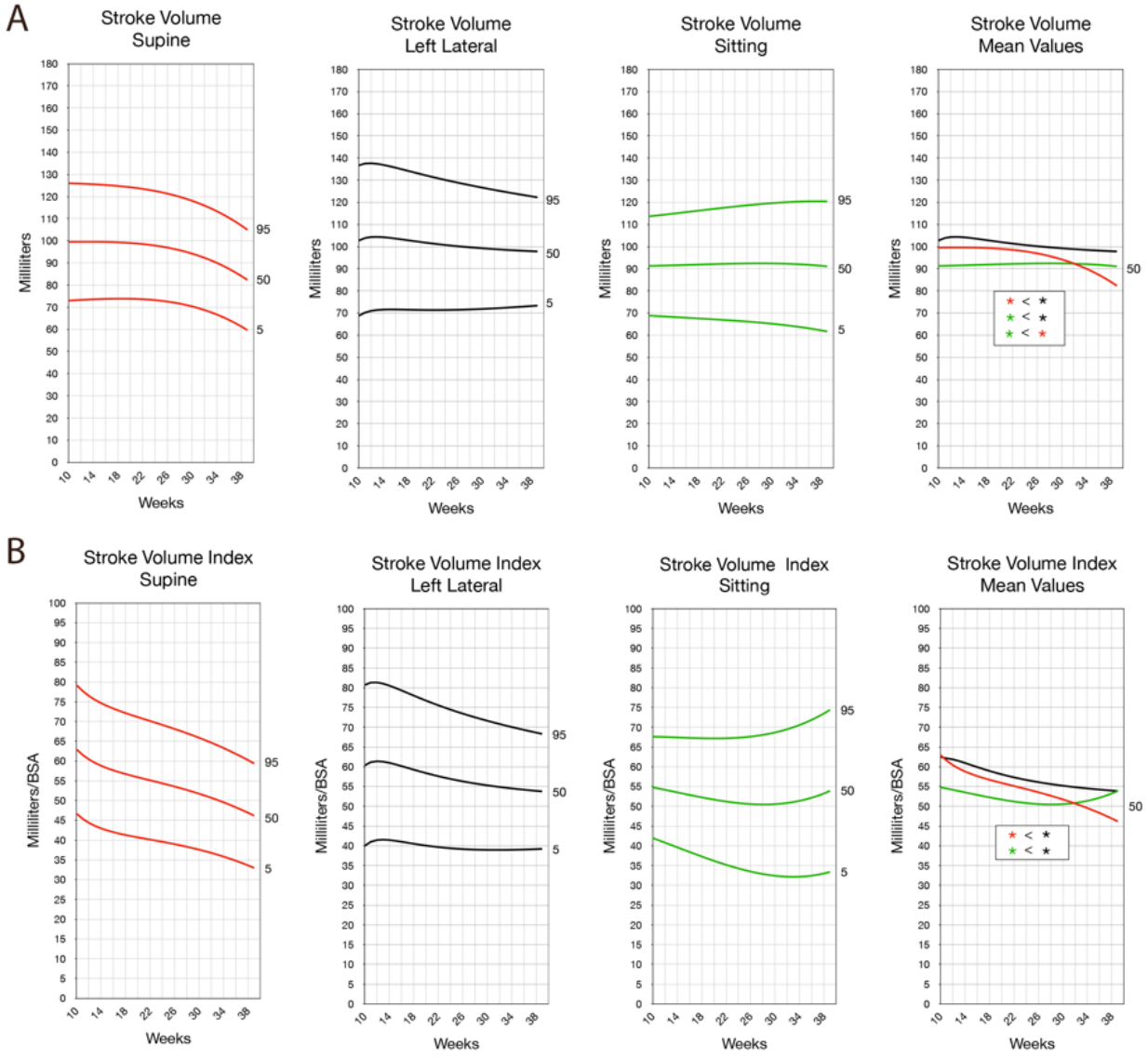


Figure 7 Reference intervals of the 5th, 50th, and 95th percentiles for the stroke volume (A) and stroke volume index (B) in the supine, left lateral, and sitting positions. When comparing the stroke volume (A) and stroke volume index (B) in the supine (blue shaded area) and sitting position with the left lateral position the mean values were higher in the left lateral position. The image that summarizes the supine, left lateral and supine graphs contains a colored * that represents the supine (red), left lateral (black) and sitting (green) mean results that are significantly different (P<0.05).

Cardiac output and cardiac index

Cardiac output (CO) rose from early gestation until the mid-second trimester and then declined in all positions (Figure 8A). No statistically significant differences were found between positions ($p > 0.05$). Cardiac index (CI) followed similar trends, peaking mid-gestation and then declining (Figure 8B) (Table 4).

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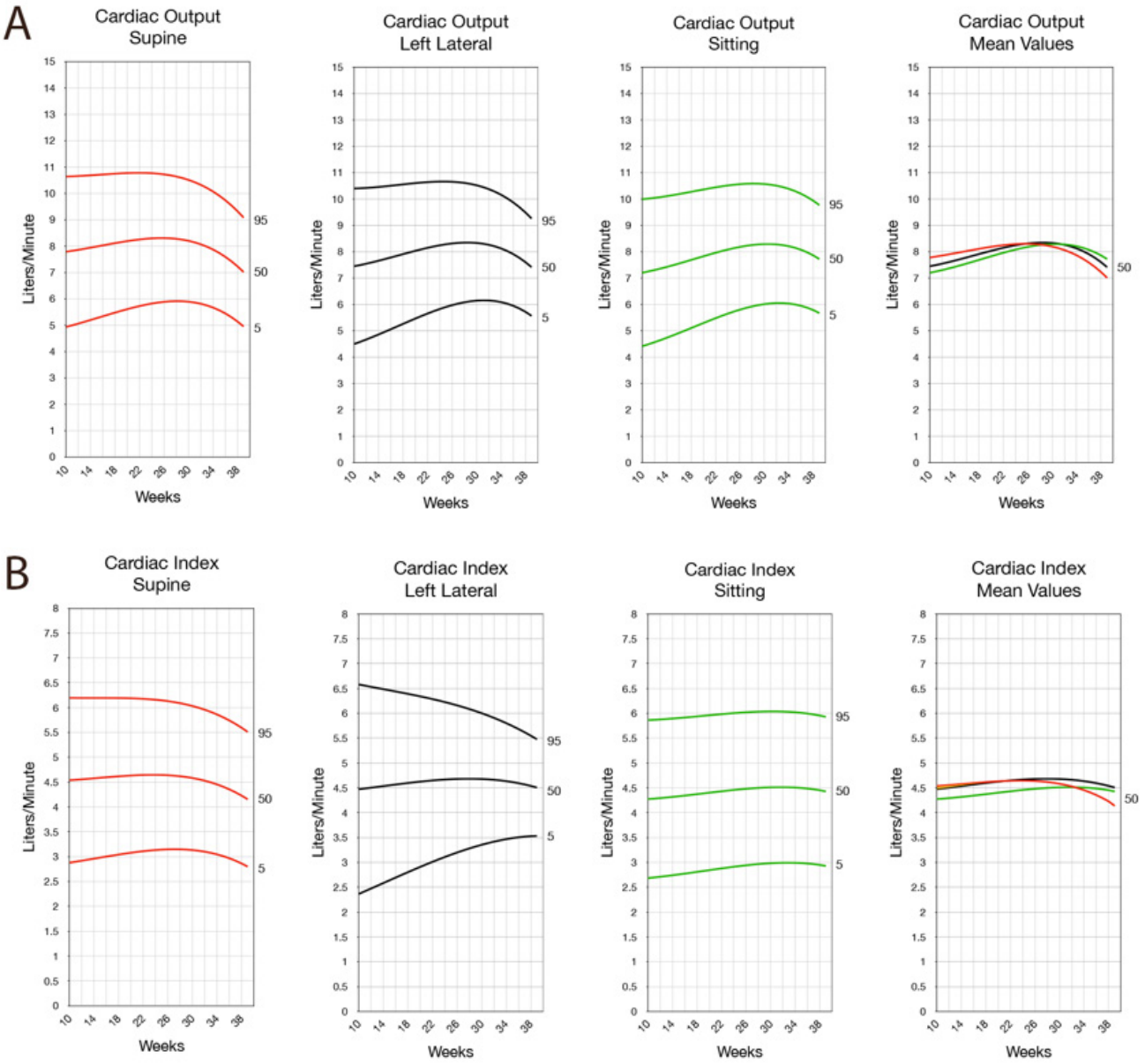


Figure 8 Reference intervals of the 5th, 50th, and 95th percentiles for the cardiac output (A) and cardiac index (B) in the supine, left lateral, and sitting positions. When comparing the cardiac output (A) and cardiac index (B) in the supine and sitting positions with the left lateral position the mean values were not significantly different.

Total peripheral vascular resistance

Total peripheral vascular resistance, calculated via both PRU and dynes/sec/cm^5, decreased until the early third trimester and then increased until term (Figure 9A & 9B). TPVR was lowest in the left lateral position, highest in the sitting position, with the supine position intermediate. These differences were statistically significant ($p < 0.001$, Table 4).

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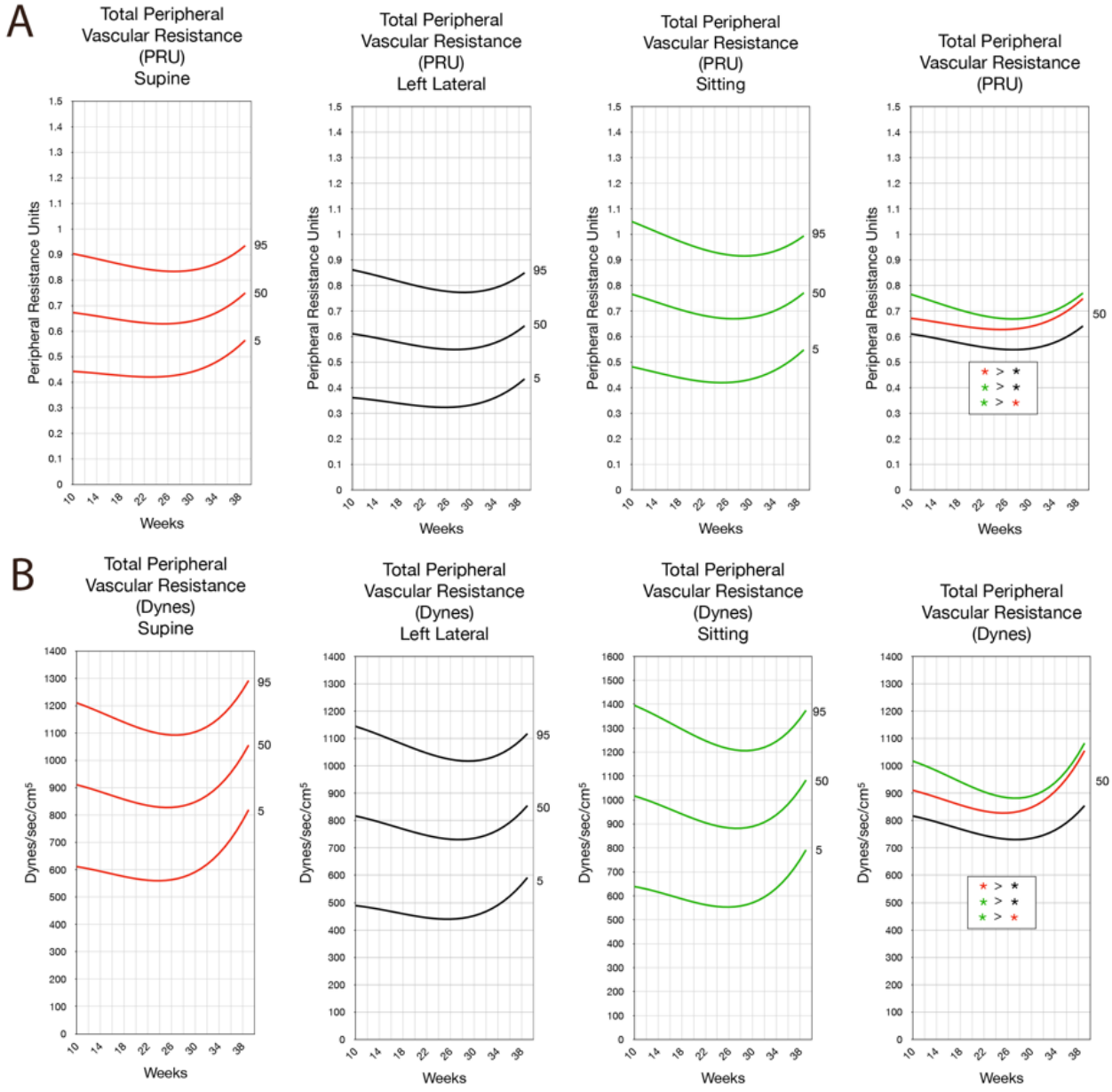


Figure 9 Reference intervals of the 5th, 50th, and 95th percentiles for the total peripheral vascular resistance measured in peripheral resistance units (A) and Dynes/sec/cm^5 (B) in the supine, left lateral, and sitting positions. When comparing the total peripheral vascular resistance, the values were lower in the left lateral position than the supine (yellow shaded area) and sitting positions throughout pregnancy. The image that summarizes the supine, left lateral and supine graphs contains a colored * that represents the supine (red), left lateral (black) and sitting (green) mean results that are significantly different ($P<0.05$).

Discussion

Principal findings

This study describes reference intervals for maternal cardiovascular parameters measured using the Vicorder® device from 10 to 39 weeks of gestation. Irrespective of maternal position, brachial and aortic mean arterial pressure and total peripheral vascular resistance followed a U-shaped trend: decreasing to a nadir in the late second or early third trimester, then increasing toward term. Heart rate changed in the opposite direction, increasing through mid-gestation and decreasing near term. Stroke volume and stroke volume index declined steadily, while cardiac output and cardiac index increased until mid-gestation before decreasing near term. Positional differences revealed that the left lateral position was associated with significantly lower mean arterial pressure and total peripheral vascular resistance, and higher stroke volume and stroke volume index, though cardiac output and cardiac index did not differ significantly across positions. These findings suggest that despite similar cardiac output across positions, reduced total peripheral vascular resistance in the left lateral position may enhance end-organ perfusion.

Comparison with existing literature

Our results align with previous studies using other devices (e.g., USCOM®, NICOM®) that report similar gestational trends in cardiac

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output, heart rate, and total peripheral vascular resistance.[38,40,42,59,60] For instance, di Pasquo et al.[61] classified hypertensive pregnant patients into hemodynamic phenotypes using USCOM measurements.[61] They demonstrated that aligning antihypertensive treatment with these profiles reduced the incidence of severe hypertension before delivery (6% vs. 19.4%, p=0.02). This underscores the value of understanding individual cardiovascular profiles to inform therapy. Our findings also support prior research indicating that altered cardiac output and total peripheral vascular resistance may precede clinical manifestations of preeclampsia and fetal growth restriction. Therefore, coupling assessment of the maternal hemodynamic profile with the appropriate antihypertensive medication appears to decrease the severity of hypertension before delivery which has important clinical implications since the latter is associated with significant perinatal and maternal morbidities and mortality.[62–73]

Changes in cardiac output and total peripheral vascular resistance prior to the manifestation of gestational hypertension, preeclampsia, and fetal growth restriction

Previous studies have suggested different manifestations of cardiac output and total peripheral vascular resistance in patients with gestational hypertension, preeclampsia, and preeclampsia with fetal growth restriction (Table 1).[74,75] In patients who develop preeclampsia the cardiac output may be increased with decreased total peripheral vascular resistance, while those with preeclampsia and fetal growth restriction the cardiac output may be decreased and total peripheral vascular resistance increased.[76,77] In addition to profiling cardiac function at the time of diagnosis of gestational hypertension or preeclampsia, studies have also reported that an increased total peripheral vascular resistance and decreased cardiac output occur in the 1st trimester, prior to the development of preeclampsia.[6,8,26,43,48] Therefore, routine measurements of these cardiovascular parameters could be facilitated using the Vicorder® device because of its ease of use.

Clinical implications

The availability of position-specific reference intervals may aid clinicians in interpreting maternal hemodynamic data more precisely. Total peripheral vascular resistance values may be important in differentiating hypertensive subtypes and guiding individualized treatment. The Vicorder® device's simplicity allows for incorporation into routine prenatal care. A practical calculator tool (Supplement 5) and instructional video (Supplement 6) were developed to facilitate bedside use. These tools enable clinicians to calculate Z-scores and percentiles, visualize gestational trends, and support evidence-based clinical decision-making.

Research implications

This study highlights the potential role of hemodynamic profiling in the management of hypertensive disorders of pregnancy. Future research should investigate the clinical utility of treatment selection based on maternal cardiac output and total peripheral vascular resistance measurements. Longitudinal studies using the Vicorder® device could determine whether early deviations from normal hemodynamic trajectories predict later adverse outcomes.

Strengths and limitations

Strengths of the study include its large sample size, standardized measurement protocol, and generation of gestational age–specific reference intervals for three maternal positions. The Vicorder® device offers a practical, non-invasive, and widely accessible method for cardiovascular assessment. However, the study did not include direct comparisons with other hemodynamic devices. As recommended by the International Working Group on Maternal Hemodynamics,[42] device-specific calibration remains essential due to technological variability.

Conclusion

This study provides gestational age-specific reference intervals and Z-score equations for maternal cardiovascular parameters from 10 to 39 weeks of gestation, stratified by maternal position. These reference values, obtained using a simple brachial cuff-based system, may aid in identifying abnormal cardiovascular adaptation in pregnancy. Incorporating these parameters into clinical practice may improve assessment and management of gestational hypertension and preeclampsia, and facilitate personalized antihypertensive therapy based on maternal hemodynamic profiles.

Acknowledgments

I would like to thank the following individuals who assisted with this study: Idella Houston, Angelica Hernandez, Rosio Anzora, and Guillermina Torres, and Cecilia Castorena.

Funding

None.

Conflicts of interest

The author has no conflict of interest.

References

1. Vasapollo B, Novelli GP, Maellaro F, et al. Maternal cardiovascular profile is altered in the preclinical phase of normotensive early and late intrauterine growth restriction. *Am J Obstet Gynecol*. 2025;232(3):312.e1–312.e21.
2. Schirwani-Hartl N, Tschanun L, Palmrich P, et al. The impact of COVID-19 during pregnancy on maternal hemodynamic function, angiogenic markers and neonatal outcome. *Viruses*. 2024;16(6):868.
3. Mangos JG, Crocker S, Flood M, et al. Use of the USCOM® noninvasive cardiac output measurement system to predict the development of pre-eclampsia in hypertensive pregnancies. *Hypertens Pregnancy*. 2024;43(1):2310607.
4. Lubrano C, Parisi F, Coco C, et al. Associations between maternal nutritional status, hemodynamic parameters, and delivery outcomes in low-risk pregnancies: a prospective observational study. *Nutrients*. 2024;16(2):183.
5. Farsetti D, Pometti F, Novelli GP, et al. Longitudinal maternal hemodynamic evaluation in uncomplicated twin pregnancies according to chorionicity: physiological cardiovascular dysfunction in monochorionic twin pregnancy. *Ultrasound Obstet Gynecol*. 2024;63(2):198–205.
6. Valensise H, Farsetti D, Pometti F, et al. The cardiac-fetal-placental unit: fetal umbilical vein flow rate is linked to the maternal cardiac profile in fetal growth restriction. *Am J Obstet Gynecol*. 2023;228(2):222.e1–222.e12.
7. Palmrich P, Haase N, Sugulle M, et al. Maternal haemodynamics in Hypertensive Disorders of Pregnancy under antihypertensive therapy (HyperDiP): study protocol for a prospective observational case-control study. *BMJ Open*. 2023;13(6):e065444.
8. Montaguti E, Cofano M, Diglio J, et al. The prediction of hypertensive disorders by maternal hemodynamic assessment in the first trimester of pregnancy. *J Matern Fetal Neonatal Med*. 2023;36(1):2198063.

9. Ackerman-Banks CM, Bhinder J, Eder M, et al. Continuous non-invasive hemodynamic monitoring in early onset severe preeclampsia. *Pregnancy Hypertens*. 2023;34:27–32.
10. Ornaghi S, Caricati A, Di Martino DD, et al. Non-invasive maternal hemodynamic assessment to classify high-risk pregnancies complicated by fetal growth restriction. *Front Clin Diabetes Healthc*. 2022;3:851971.
11. Montaguti E, Di Donna G, Youssef A, et al. Hypertensive disorders and maternal hemodynamic changes in pregnancy: monitoring by USCOM(®) device. *J Med Ultrason*. 2022;49(3):405–413.
12. Montaguti E, Di Donna G, Pilu G. Usefulness of USCOM® evaluation in women with chronic hypertension who developed severe preeclampsia with low platelets and elevated liver enzymes. *J Matern Fetal Neonatal Med*. 2022;35(25):4942–4945.
13. Mecacci F, Ottanelli S, Vannuccini S, et al. Maternal hemodynamic changes in gestational diabetes: a prospective case-control study. *Arch Gynecol Obstet*. 2022;306(2):357–363.
14. Mappa I, Luviso M, Tartaglia S, et al. Maternal cardiovascular function in the prediction of fetal distress in labor: a prospective cohort study. *J Matern Fetal Neonatal Med*. 2022;35(25):7139–7145.
15. Farsetti D, Vasapollo B, Pometti F, et al. Maternal hemodynamics for the identification of early fetal growth restriction in normotensive pregnancies. *Placenta*. 2022;129:12–14.
16. Perry H, Binder J, Gutierrez J, et al. Maternal haemodynamic function differs in pre-eclampsia when it is associated with a small-for-gestational--age newborn: a prospective cohort study. *BJOG*. 2021;128(2):167–175.
17. Yin O, Woods A, Koos B, et al. Central hemodynamics are associated with fetal outcomes in pregnancies of advanced maternal age. *Pregnancy Hypertens*. 2020;19:67–73.
18. Phillips RA, Ma Z, Kong B, et al. Maternal hypertension, advanced Doppler haemodynamics and therapeutic precision: principles and illustrative cases. *Curr Hypertens Rep*. 2020;22(7):49.
19. Perry H, Lehmann H, Mantovani E, et al. Are maternal hemodynamic indices markers of fetal growth restriction in pregnancies with a small-for--gestational-age fetus? *Ultrasound Obstet Gynecol*. 2020;55(2):210–216.
20. Kalafat E, Barratt I, Nawaz A, et al. Maternal cardiovascular function and risk of intrapartum fetal compromise in women undergoing induction of labor: pilot study. *Ultrasound Obstet Gynecol*. 2020;56(2):233–239.
21. Verburg PE, Roberts CT, McBean E, et al. Peripheral maternal haemodynamics across pregnancy in hypertensive disorders of pregnancy. *Pregnancy Hypertens*. 2019;16:89–96.
22. Perry H, Lehmann H, Mantovani E, et al. Correlation between central and uterine hemodynamics in hypertensive disorders of pregnancy. *Ultrasound Obstet Gynecol*. 2019;54(1):58–63.
23. Valensise H, Tiralongo GM, Pisani I, et al. Maternal hemodynamics early in labor: a possible link with obstetric risk? *Ultrasound Obstet Gynecol*. 2018;51(4):509–513.
24. Vinayagam D, Gutierrez J, Binder J, et al. Impaired maternal hemodynamics in morbidly obese women: a case-control study. *Ultrasound Obstet Gynecol*. 2017;50(6):761–765.
25. Monteith C, McSweeney L, Breatnach CR, et al. Non-invasive cardiac output monitoring (NICOM(®)) can predict the evolution of uteroplacental disease-Results of the prospective HANDLE study. *Eur J Obstet Gynecol Reprod Biol*. 2017;216:116–124.
26. Gagliardi G, Tiralongo GM, LoPresti D, et al. Screening for pre-eclampsia in the first trimester: role of maternal hemodynamics and bioimpedance in non-obese patients. *Ultrasound Obstet Gynecol*. 2017;50(5):584–588.
27. Valensise H, Farsetti D, Lo Presti D, et al. Preterm delivery and elevated maternal total vascular resistance: signs of suboptimal cardiovascular adaptation to pregnancy? *Ultrasound Obstet Gynecol*. 2016;48(4):491–495.
28. Stott D, Bolten M, Salman M, et al. Maternal demographics and hemodynamics for the prediction of fetal growth restriction at booking, in pregnancies at high risk for placental insufficiency. *Acta Obstet Gynecol Scand*. 2016;95(3):329–338.
29. Khalil A, Goodyear G, Joseph E, et al. PP097. Cardiac output and systemic vascular resistance in normal pregnancy and in control non-pregnant women. *Pregnancy Hypertens*. 2012;2(3):292–293.
30. Vasapollo B, Novelli GP, Farsetti D, et al. Maternal peripheral vascular resistance at mid gestation in chronic hypertension as a predictor of fetal growth restriction. *J Matern Fetal Neonatal Med*. 2022;35(25):9834–9836.
31. Di Martino DD, Ferrazzi E, Garbin M, et al. Multivariable evaluation of maternal hemodynamic profile in pregnancy complicated by fetal growth restriction: prospective study. *Ultrasound Obstet Gynecol*. 2019;54(6):732–739.
32. Vasapollo B, Novelli GP, Gagliardi G, et al. Medical treatment of early-onset mild gestational hypertension reduces total peripheral vascular resistance and influences maternal and fetal complications. *Ultrasound Obstet Gynecol*. 2012;40(3):325–331.
33. Novelli GP, Vasapollo B, Gagliardi G, et al. Left ventricular midwall mechanics at 24 weeks' gestation in high-risk normotensive pregnant women: relationship to placenta-related complications of pregnancy. *Ultrasound Obstet Gynecol*. 2012;39(4):430–437.
34. Valensise H, Vasapollo B, Novelli GP, et al. Maternal total vascular resistance and concentric geometry: a key to identify uncomplicated gestational hypertension. *BJOG*. 2006;113(9):1044–1052.
35. Vasapollo B, Valensise H, Novelli GP, et al. Abnormal maternal cardiac function precedes the clinical manifestation of fetal growth restriction. *Ultrasound Obstet Gynecol*. 2004;24(1):23–29.
36. Vasapollo B, Valensise H, Novelli GP, et al. Abnormal maternal cardiac function and morphology in pregnancies complicated by intrauterine fetal growth restriction. *Ultrasound Obstet Gynecol*. 2002;20(5):452–457.
37. Szczepkowska A, Lausegger S, Papastefanou I, et al. Maternal vascular indices and hemodynamic parameters at 36 weeks' gestation in gestational and pre-existing diabetes mellitus. *Ultrasound Obstet Gynecol*. 2024;64(5):597–603.
38. Vinayagam D, Thilaganathan B, Stirrup O, et al. Maternal hemodynamics in normal pregnancy: reference ranges and role of maternal characteristics. *Ultrasound Obstet Gynecol*. 2018;51(5):665–671.
39. Vinayagam D, Patey O, Thilaganathan B, et al. Cardiac output assessment in pregnancy: comparison of two automated monitors with echocardiography. *Ultrasound Obstet Gynecol*. 2017;49(1):32–38.
40. DeVore GR, Polanco B. Assessing maternal cardiac function by obstetricians: technique and reference ranges. *Am J Obstet Gynecol*. 2023;229(2):155.e1–155.e18.
41. Lihme F, Basit S, Persson LG, et al. Reference ranges for third-trimester maternal cardiovascular function parameters measured in normotensive pregnant women using a non-invasive cardiac output monitor: a study based on data from the prospective PEACH cohort study. *BJOG*. 2024;131(4):463–471.
42. Bijl RC, Valensise H, Novelli GP, et al. Methods and considerations concerning cardiac output measurement in pregnant women: recommendations of the International Working Group on Maternal Hemodynamics. *Ultrasound Obstet Gynecol*. 2019;54(1):35–50.
43. Montaguti E, Youssef A, Cavalera M, et al. Maternal hemodynamic assessment by USCOM(®) device in the first trimester of pregnancy. *J Matern Fetal Neonatal Med*. 2022;35(25):5580–5586.
44. Gutierrez Henares J, Gutierrez Henares R, Perry H, Khalil A, et al. Maternal cardiovascular potential and kinetic energy indices in pre-eclamptic and small-for-gestational-age pregnancies. *Ultrasound Obstet Gynecol*. 2022;59(5):613–618.

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45. Mulder E, Basit S, Oben J, et al. Accuracy and precision of USCOM versus transthoracic echocardiography before and during pregnancy. *Pregnancy Hypertens*. 2019;17:138–143.

46. Mangos JG, Pettit F, Preece R, et al. Repeatability of USCOM®-measured cardiac output in normotensive non-pregnant and pregnant women. *Pregnancy Hypertens*. 2018;12:71–74.

47. Vinayagam D, Bowe S, Sheehan E, et al. Non-invasive haemodynamic monitoring in pregnancy: a comparative study using ultrasound and bioreactance. *Fetal Diagn Ther*. 2017;41(4):273–282.

48. Tiralongo GM, Lo Presti D, Pisani I, et al. Assessment of total vascular resistance and total body water in normotensive women during the first trimester of pregnancy. a key for the prevention of preeclampsia. *Pregnancy Hypertens*. 2015;5(2):193–197.

49. McNamara H, Barclay P, Sharma V. Accuracy and precision of the ultrasound cardiac output monitor (USCOM 1A) in pregnancy: comparison with three-dimensional transthoracic echocardiography. *Br J Anaesth*. 2014;113(4):669–676.

50. van der Graaf AM, Zeeman GG, Groen H, et al. Non-invasive assessment of maternal hemodynamics in early pregnancy. *Pregnancy Hypertens*. 2013;3(4):261–269.

51. Kager CC, Dekker GA, Stam MC. Measurement of cardiac output in normal pregnancy by a non-invasive two-dimensional independent Doppler device. *Aust N Z J Obstet Gynaecol*. 2009;49(2):142–144.

52. Lo Presti D, Scala RL, Tiralongo GM, et al. PP065. Assessment of total vascular resistance and total body water in normotensive women during the first trimester of pregnancy in order to predict hypertensive complications. *Pregnancy Hypertens*. 2013;3(2):90–91.

53. Lo Presti D, Scala RL, Tiralongo GM, et al. PP064. Total vascular resistances in early pregnancy: A key to understand abnormal cardiovascular adaptation associated with spontaneous abortion. *Pregnancy Hypertens*. 2013;3(2):90.

54. Royston P. Constructing time-specific reference ranges. *Stat Med*. 1991;10(5):675–690.

55. Silverwood RJ, Cole TJ. Statistical methods for constructing gestational age-related reference intervals and centile charts for fetal size. *Ultrasound Obstet Gynecol*. 2007;29(1):6–13.

56. Altman DG, Chitty LS. Design and analysis of studies to derive charts of fetal size. *Ultrasound Obstet Gynecol*. 1993;3(6):378–384.

57. Altman DG, Chitty LS. Charts of fetal size: 1. Methodology. *Br J Obstet Gynaecol*. 1994;101(1):29–34.

58. Klabunde R. Vascular function. In: Klabunde R, editor. *Cardiovascular physiology concepts*. 3rd ed. Wolters Kluwer; 2022:112:chap 5.

59. Meah VL, Cockcroft JR, Backx K, et al. Cardiac output and related haemodynamics during pregnancy: a series of meta-analyses. *Heart*. 2016;102(7):518–526.

60. Mahendru AA, Everett TR, Wilkinson IB, et al. A longitudinal study of maternal cardiovascular function from preconception to the postpartum period. *J Hypertens*. 2014;32(4):849–856.

61. di Pasquo E, Giannubilo SR, Valentini B, et al. The "Preeclampsia and Hypertension Target Treatment" study: a multicenter prospective study to evaluate the effectiveness of the antihypertensive therapy based on maternal hemodynamic findings. *Am J Obstet Gynecol MFM*. 2024;6(5):101368.

62. Lee R, Brandt JS, Joseph KS, et al. Pregnancy-associated mortality due to cardiovascular disease: impact of hypertensive disorders of pregnancy. *Paediatr Perinat Epidemiol*. 2024;38(3):204–215.

63. Steegers EA, von Dadelszen P, Duvekot JJ, et al. Pre-eclampsia. *Lancet*. 2010;376(9741):631–644.

64. Ford ND, Cox S, Ko JY, et al. Hypertensive disorders in pregnancy and mortality at delivery hospitalization - United States, 2017-2019. *MMWR Morb Mortal Wkly Rep*. 2022;71(17):585–591.

65. Magee LA, Smith GN, Bloch C, et al. Guideline No. 426: hypertensive disorders of pregnancy: diagnosis, prediction, prevention, and management. *J Obstet Gynaecol Can*. 2022;44(5):547–571.e1.

66. Venkatesh KK, Perak AM, Wu J, et al. Impact of hypertensive disorders of pregnancy and gestational diabetes mellitus on offspring cardiovascular health in early adolescence. *Am J Obstet Gynecol*. 2025;232(2):218.e1–218.e12.

67. Borders A, Keenan-Devlin L, Oh EH, et al. Reducing severe maternal morbidity for birthing persons with severe hypertension through a statewide quality improvement initiative. *Am J Obstet Gynecol*. 2025;232(1):128.e1–128.e9.

68. Joseph KS, Lisonkova S, Boutin A, et al. Maternal mortality in the United States: are the high and rising rates due to changes in obstetrical factors, maternal medical conditions, or maternal mortality surveillance? *Am J Obstet Gynecol*. 2024;230(4):440.e1–440.e13.

69. Harper LM, Kuo HC, Boggess K, et al. Blood pressure control in pregnant patients with chronic hypertension and diabetes: should <130/80 be the target? *Am J Obstet Gynecol*. 2025;232(5):482.e1-482.e8.

70. Carey C, Mulcahy E, McCarthy FP, et al. Hypertensive disorders of pregnancy and the risk of maternal dementia: a systematic review and meta-analysis. *Am J Obstet Gynecol*. 2024;231(2):196–210.

71. Brohan MP, Daly FP, Kelly L, et al. Hypertensive disorders of pregnancy and long-term risk of maternal stroke-a systematic review and meta-analysis. *Am J Obstet Gynecol*. 2023;229(3):248–268.

72. Alers RJ, Ghossein-Doha C, Canjels LPW, et al. Attenuated cognitive functioning decades after preeclampsia. *Am J Obstet Gynecol*. 2023;229(3):294.e1–294.e14.

73. Ackerman-Banks CM, Lipkind HS, Palmsten K, et al. Association between hypertensive disorders of pregnancy and cardiovascular diseases within 24 months after delivery. *Am J Obstet Gynecol*. 2023;229(1):65.e1–65.e15.

74. Masini G, Foo LF, Tay J, et al. Preeclampsia has two phenotypes which require different treatment strategies. *Am J Obstet Gynecol*. 2022;226(2s):S1006–S1018.

75. Gyselaers W. Hemodynamic pathways of gestational hypertension and preeclampsia. *Am J Obstet Gynecol*. 2022;226(2s):S988–S1005.

76. Maseliene T, Zukiene G, Laurinaviciene A, et al. Alterations in maternal cardiovascular parameters and their impact on uterine and fetal circulation in hypertensive pregnancies and fetal growth restriction. *Int J Cardiol Cardiovasc Risk Prev*. 2024;22:200316.

77. Tay J, Foo L, Masini G, et al. Early and late preeclampsia are characterized by high cardiac output, but in the presence of fetal growth restriction, cardiac output is low: insights from a prospective study. *Am J Obstet Gynecol*. 2018;218(5):517.e1–517.e12.