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Trimester-Specific Hemodynamic and Blood Volume Adaptations in Pregnancy: A Systematic Review of Normal and High-Risk Populations

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ABSTRACT

Pregnancy involves significant cardiovascular adaptations, including increased blood volume, cardiac output (CO), and reduced systemic vascular resistance (SVR), which are essential to support fetal development. While these changes are well established, the timing, magnitude, and variability across maternal risk profiles remain inconsistently reported. **Objectives:** To synthesize original research from 2015 to 2025 examining trimester-specific changes in maternal hemodynamic and blood volume, with a focus on both normal and high-risk pregnancies. **Methods:** Systematic searches were conducted in PubMed, Science Direct, Scopus, and Wiley Online Library. Inclusion criteria encompassed original English-language studies involving human pregnancies that assessed maternal blood volume or cardiovascular parameters using validated methods. Extracted data were synthesized across four domains: study characteristics, trimester-wise trends, methodology, and quality. **Results:** Nine studies met the inclusion criteria. Most reported a rise in CO and plasma volume from early to mid-pregnancy, alongside a reduction in SVR. In contrast, high-risk groups, including those with fetal growth restriction (FGR), preeclampsia (PE), and obesity, demonstrated impaired adaptation, marked by persistently high SVR and reduced stroke volume. **Conclusions:** It was concluded that trimester-specific hemodynamic adaptation is essential for healthy pregnancy progression. Deviations in high-risk populations may serve as early markers of complications. Incorporating non-invasive cardiovascular monitoring into routine prenatal care may improve risk stratification and outcomes.

INTRODUCTION

Pregnancy induces a series of profound physiological changes to support fetal growth and maternal adaptation [1]. Among the most critical are cardiovascular adjustments, including increased cardiac output (CO), plasma volume expansion, and reduced systemic vascular resistance (SVR), which ensure adequate uteroplacental perfusion. These changes begin as early as the first trimester and peak during mid-pregnancy, allowing for enhanced nutrient and oxygen delivery to the fetus [2, 3]. Normal gestation is typically characterized by a 40–50% increase in maternal blood volume and up to a 50% rise in CO by the second trimester, accompanied by a drop in SVR and arterial blood pressure [4, 5]. These adaptations are not only vital for fetal development but also serve as indicators of maternal cardiovascular health. When these physiological shifts are impaired or absent, the risk of

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pregnancy complications increases. Conditions such as preeclampsia (PE), fetal growth restriction (FGR), and maternal obesity are often associated with reduced CO, persistently elevated SVR, and abnormal vascular remodelling [6, 7]. Despite these established principles, there is inconsistency in the literature regarding the precise timing, magnitude, and clinical relevance of hemodynamic changes across trimesters. Many earlier studies focused predominantly on the second or third trimesters, often overlooking early cardiovascular markers detectable in the first trimester [8-10]. Furthermore, methodological variability in measurement tools ranging from Doppler ultrasound to NICOM (non-invasive cardiac output monitoring) and MRI complicates cross-study comparison. As a result, there is a lack of consolidated data detailing normative versus pathological trends in maternal hemodynamic across different risk groups and gestational stages [11]. In recent years, the use of advanced, non-invasive cardiovascular monitoring has become more widely available in both clinical and research settings. These tools have enabled earlier and more precise tracking of maternal cardiovascular function, highlighting the need to re-evaluate and synthesize current evidence. Clinical guidelines from the American College of Obstetricians and Gynaecologists (ACOG) and the World Health Organization (WHO) increasingly emphasize the importance of early maternal risk stratification and individualized antenatal care [12, 13]. Understanding how hemodynamic profiles differ by trimester and maternal risk status is central to this approach.

Pregnancy is associated with substantial cardiovascular and blood volume adaptations that are essential for maintaining maternal health and supporting fetal growth. However, the timing, magnitude, and progression of these hemodynamic changes across different trimesters remain inconsistently reported, particularly among high-risk pregnancies such as preeclampsia, fetal growth restriction, and obesity. Existing studies also vary considerably in methodology and measurement techniques, limiting direct comparison and clinical application. Therefore, this systematic review aimed to synthesize current evidence on trimester-specific maternal hemodynamic and blood volume adaptations in both normal and high-risk pregnancies and to identify patterns that may support early risk stratification and improved prenatal care.

METHODS

This systematic review followed the PRISMA 2020 guidelines. Its objective was to synthesize original research examining maternal hemodynamic and blood volume changes during pregnancy across different gestational stages and population risk profiles. Studies were included if they met the following criteria: Study Type: Original, peer-reviewed research articles (cohort, cross-sectional, or observational studies). Population: Pregnant women at any gestational age, including both healthy and high-risk groups (with hypertension, FGR, or obesity). Outcomes: Reported maternal hemodynamic parameters such as CO, SV, SVR, blood pressure (BP), or plasma volume. Methods: Used validated measurement techniques (NICOM bioreactance, Doppler ultrasound, impedance cardiography, MRI, or tonometry). Language and Date: Published in English between January 1, 2015 and March 30, 2025. Exclusion criteria included: non-original articles (reviews, editorials, case reports), animal studies, studies not reporting maternal outcomes, or those lacking trimester-specific data. A comprehensive search was conducted in PubMed, Scopus, Science Direct, Wiley Online Library, Google Scholar, and reference lists. The final search was completed in March 2025 using Me-SH terms and keywords such as "maternal blood volume," "hemodynamic adaptation," and "cardiac output," with Boolean operators to refine results. Search results were imported into EndNote and duplicates removed. Two reviewers independently screened titles and abstracts, followed by full-text reviews for eligibility. Discrepancies were resolved through discussion or adjudicated by a third reviewer. Inter-rater agreement was achieved through consensus. Discrepancies in interpretation were reviewed and resolved by a third investigator. Study quality was evaluated using a modified version of the Joanna Briggs Institute (JBI) checklist for cohort and observational studies. Two reviewers independently assessed five key domains: (1) clear inclusion criteria, (2) validity of exposure measurement, (3) validity of outcome measurement, (4) confounder identification and control, and (5) adequacy of follow-up. "Partial" scores were assigned when criteria were only partly met for example, when studies had small pilot samples limiting generalizability, or when key confounders such as BMI or parity were not adjusted for. Final quality ratings (high, moderate) were reached through reviewer consensus. A total of 186 records were initially retrieved; after screening, nine studies met all inclusion criteria and were included in the final review. The selection process is illustrated in the PRISMA 2020 flow diagram. Extracted variables included: Author and publication year. Country and study design. Sample size and gestational window. Measurement methods. Maternal characteristics (age, BMI, comorbidities) and reported hemodynamic outcomes and key findings (Figure 1).

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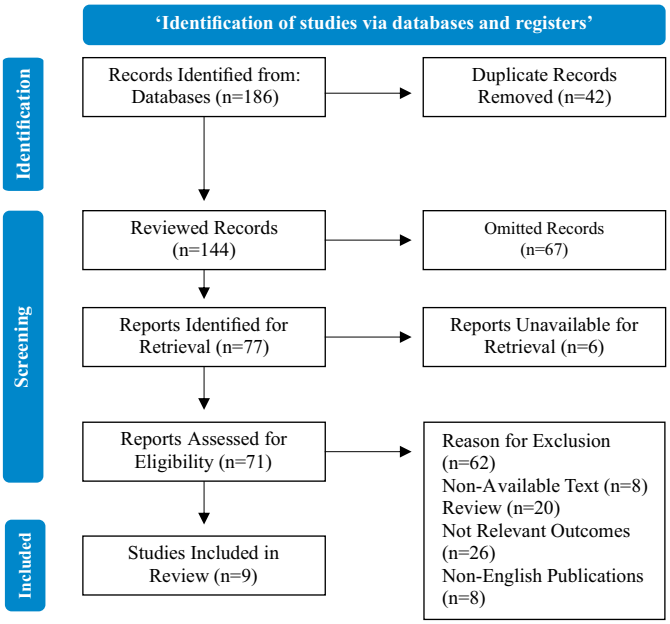

Figure 1: PRISMA 2020 Flow Diagram Illustrating the Process of Study Identification, Screening, and Inclusion

RESULTS

This systematic review included nine original studies published between 2015 and 2025 that examined maternal hemodynamic and blood volume changes during pregnancy. CO showed a consistent increase from the first to the second trimester across most studies, typically peaking mid-gestation. For example, Ling *et al.*, reported a rise in CO from approximately 5.2 L/min in early pregnancy to 6.4 L/min in the second trimester among younger women, accompanied by a significant reduction in systemic vascular resistance (SVR) [14]. In women with fetal growth restriction (FGR), Stott *et al.*, observed persistently low CO and elevated SVR throughout the second and third trimesters, indicating poor cardiovascular adaptation [15]. Several studies, including those by O'Callaghan *et al.*, and Iacobaeus *et al.*, identified a biphasic adaptation pattern [16, 17]. During early pregnancy, vascular resistance declined, along with a drop in central blood pressure.By the third trimester, these trends began to reverse slightly, with some studies reporting a modest increase in SVR and a reduction in stroke volume (SV). Measurement methods varied but remained non-invasive and clinically validated. Bioreactance via NICOM was most frequently used, while others applied MRI, Doppler ultrasound, and pulse wave analysis. Despite differences in methodology, trends were largely consistent. Studies focusing on high-risk groups, such as Pisani *et al.*, on hypertensive pregnancies and Patel *et al.*, on obesity, showed altered hemodynamic characterized by higher SVR and reduced vascular compliance [18, 19]. Geographic representation spanned Europe, North America, and Scandinavia. However, early gestational data (first trimester) were underrepresented in some studies, with several initiating follow-ups only from the second trimester. Furthermore, only a few studies, such as Gragasin *et al.*, explored relationships between maternal hemodynamics and fetal or placental outcomes [11]. Findings summarize the design, population, gestational window, and key findings of these studies (Table 1).

Table 1: Summary of Included Studies on Maternal Hemodynamic and Blood Volume Adaptations (2015–2025)

Sr. No.	References	Country	Study Design	Sample Size	Gestational Window	Methods	Population	Key Hemodynamic Findings
1	[11]	Canada	Cross-sectional	182	Term only	HemoCue Hb201+	Healthy term pregnancies	Cord Hb ↑ with BW:PW ratio; maternal Hb not associated
2	[14]	UK	Prospective longitudinal	1,789	11–13, 20–22, 34–37 weeks	NICOM	Healthy, stratified by age/BMI	↑CO, ↓SVR in younger women; age influences adaptation
3	[15]	UK	Prospective longitudinal	140	20–36 weeks	NICOM	FGR vs. AGA pregnancies	FGR group had sustained ↓CO, ↑SVR
4	[16]	Ireland	Prospective longitudinal	100	14, 24, 36 weeks	Tonometry	Normotensive singleton pregnancies	↓Central BP mid-pregnancy; CO peak at 24 weeks
5	[17]	Sweden	Prospective longitudinal	55	10–40 weeks	US, FMD	Healthy singleton pregnancies	↑FMD early, ↓FMD near term; biphasic vascular function
6	[18]	Belgium	Prospective observational	120	12–40 weeks	Non-invasive monitor	Normotensive, GHTN, PE	↑SVR, ↓CO in PE; distinct hypertensive profiles
7	[19]	Denmark	Prospective observational	115 (65 obese, 50 controls)	14, 24, 36 weeks	NICOM	Obese vs. normal BMI	Obese group: ↑CO, ↑SVR, ↓vascular compliance
8	[20]	USA	Pilot study	14 pregnant, 14 non-pregnant, 9 postpartum	Mean 26 ± 7 weeks	Quantitative MRI	Healthy and PE	↓Vascular reactivity; more dysfunction in PE postpartum

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9	[21]	Belgium	Data synthesis	Not stated	Late 1st to 3rd trimester	Impedance, Doppler	Aggregated prospective data	↑Venous capacity, redistribution essential for adaptation

↑ = Increase; ↓ = Decrease; CO = Cardiac Output; SV = Stroke Volume; SVR = Systemic Vascular Resistance; FGR = Fetal Growth Restriction; PE = Preeclampsia; GHTN = Gestational Hypertension; BMI = Body Mass Index; FMD = Flow-Mediated Dilation; BP = Blood Pressure; NICOM = Non-Invasive Cardiac Output Monitoring; US = Ultrasound; MRI = Magnetic Resonance Imaging; BW:PW = Birth Weight to Placental Weight Ratio

Findings present a critical appraisal of the included studies using a modified Joanna Briggs Institute (JBI) checklist for cohort and observational designs. Most studies demonstrated high methodological rigor, particularly in clear inclusion criteria, valid outcome measurements, and adequate follow-up protocols. Studies by Ling *et al.*, Stott *et al.*, and Pisani *et al.*, received "High" quality ratings due to longitudinal follow-up, well-defined populations, and effective control for confounders [14, 15, 18]. In contrast, Gragasin *et al.*, and Langham *et al.*, were rated Moderate [11, 20]. Langham *et al.*, was a small pilot study, which limited generalizability and statistical power, while Gragasin *et al.*, used a cross-sectional design and did not adjust for potential confounders such as body mass index (BMI) or parity [11]. Greenspan., although methodologically structured, synthesized data from earlier prospective studies rather than collecting new participant-level data [21]. Therefore, while the measurement tools used were valid, the absence of original data collection reduced their empirical strength and led to a "Moderate" rating. Partial ratings in the table indicate criteria that were only partially met. For example, a study may have used validated tools and defined outcomes well but lacked comprehensive control of confounders or had limitations in design (pilot nature, small sample size, or reliance on aggregated data). These nuances were considered when assigning overall quality scores (Table 2).

Table 2: Quality Assessment of Included Studies (Modified JBI Checklist)

Sr. No.	References	Inclusion Criteria Clear	Valid Exposure Measurement	Confounder Control	Adequate Follow-up	Overall Quality
1	[11]	Yes	Yes	Partial (no confounder control)	Not applicable	Moderate
2	[14]	Yes	Yes	Yes	Yes	High
3	[15]	Yes	Yes	Yes	Yes	High
4	[16]	Yes	Yes	Yes	Yes	High
5	[18]	Yes	Yes	Yes	Yes	High
6	[17]	Yes	Yes	Yes	Yes	High
7	[19]	Yes	Yes	Yes	Yes	High
8	[20]	Yes	Yes	Partial (pilot study, small N)	Yes	Moderate
9	[21]	Partial (synthesis data)	Yes	Yes	Not applicable	Moderate

"Partial" = Criteria partially met (pilot design, absence of confounder adjustment, or reliance on synthesized data). "Not applicable" = Study design (e.g., cross-sectional or secondary analysis) did not involve longitudinal follow-up. All studies used validated measurement tools, but external validity was limited in smaller or non-comparative studies.

Results highlight the trimester-specific cardiovascular adaptations during pregnancy across the included studies. A biphasic pattern is evident in most healthy pregnancies: the first trimester is marked by systemic vasodilation, reflected in reduced systemic vascular resistance (SVR) and blood pressure, while CO begins to rise. These changes intensify during the second trimester, when CO typically peaks, and vascular compliance improves further to meet the increasing metabolic demands of the developing fetus. This physiologic adaptation supports placental perfusion and fetal oxygenation. In the third trimester, some of these trends begin to reverse. Several studies reported a plateau or slight increase in SVR and a reduction or stabilization in CO and flow-mediated dilation (FMD), suggesting a compensatory phase as the cardiovascular system adjusts to late gestational load. For example, Ling *et al.*, documented a peak CO of approximately 6.4 L/min in the second trimester with a modest rise in SVR approaching term [14]. In contrast, pregnancies complicated by FGR, obesity, or PE exhibited abnormal hemodynamic patterns throughout gestation. Studies by Stott *et al.* and Pisani *et al.*, consistently reported reduced CO and persistently elevated SVR in these high-risk groups [15, 18]. Similarly, Patel *et al.*, noted that obese women maintained high SVR and impaired vascular compliance across all trimesters, indicating suboptimal adaptation [19]. Overall, the table underscores that healthy pregnancies exhibit dynamic, gestation-specific hemodynamic regulation, while deviations in high-risk pregnancies reflect underlying pathophysiology that may predispose to adverse outcomes (Table 3).

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Table 3: Hemodynamic Trends by Trimester Across Included Studies

Sr. No.	Study	Early Pregnancy (1st Trimester)	Mid-Pregnancy (2nd Trimester)	Late Pregnancy (3rd Trimester)
1	[11]	–	–	Cord Hb ↑ with BW: PW ratio; maternal Hb uncorrelated
2	[14]	↑CO (~5.2 L/min), ↓SVR in younger women	↑↑CO (~6.4 L/min), SVR stable	CO maintained, slight ↑SVR
3	[15]	–	↓CO, ↑SVR in FGR	Sustained ↓CO, ↑SVR in FGR
4	[16]	↓cfPWV, ↓brachial BP	Peak CO (~6.0 L/min)	↑BP, CO returned to baseline
5	[17]	↑FMD, ↓central BP	BP plateaued	↓FMD, ↑central BP
6	[18]	–	Profiles varied by HTN type	PE: ↑SVR (~1800 dyn: s/cm^5), ↓CO
7	[19]	↑CO, ↑SVR in obese group	Persistent ↑SVR	↑SVR, ↓vascular compliance
8	[20]	–	↓Vascular reactivity in pregnancy group	Compared to the postpartum group
9	[21]	↑Venous capacity	Redistribution of blood volume	↑Uterine venous impedance

↑ = Increase; ↓ = Decrease; CO = Cardiac Output; SVR = Systemic Vascular Resistance; FMD = Flow-Mediated Dilation; BP = Blood Pressure; cfPWV = Carotid-Femoral Pulse Wave Velocity; PE = Preeclampsia; HTN = Hypertension; FGR = Fetal Growth Restriction; Hb = Hemoglobin; BW:PW = Birth Weight to Placental Weight Ratio. "–" = Not reported in that trimester

The study summarises the methodological diversity among the included studies in terms of design, sample size, population type, measurement tools, and gestational coverage. Most studies employed prospective longitudinal designs, allowing repeated assessments across gestation and enhancing temporal validity. The inclusion of high-quality monitoring methods such as bioreactance-based NICOM, Doppler ultrasound, and pulse wave analysis ensured accurate, non-invasive evaluation of cardiovascular parameters. Sample sizes varied considerably, ranging from 37 participants in Langham *et al.*, MRI pilot study to over 1,700 in Ling *et al.*, population-based cohort. This heterogeneity impacted the generalizability and statistical power of findings [20, 14]. While most studies focused on healthy pregnancies, several included high-risk groups such as women with PE [18], FGR [15], or obesity [19], offering valuable comparisons. Gestational timing also varied. Some studies followed participants throughout pregnancy, while others Gragasin *et al.*, were limited to term-only assessments, missing temporal trends [11]. Studies beginning in the second trimester may have overlooked early hemodynamic changes critical for risk prediction. Overall, despite differences in design and timing, the inclusion of diverse populations and validated tools strengthens the review's clinical relevance, though it poses challenges for direct comparison (Table 4).

Table 4: Methodological and Population Characteristics of Included Studies

References	Study Design	Population	Sample Size	Measurement Method	Gestational Window
[11]	Cross-sectional	Healthy term pregnancies	182	Hemoglobin analyzer (HemoCue)	Term only (delivery)
[14]	Prospective longitudinal	Healthy, age-stratified pregnant women	1,789	NICOM (bioreactance)	11–37 weeks
[15]	Prospective longitudinal	FGR vs. AGA pregnancies	140	NICOM (bioreactance)	20–36 weeks
[16]	Prospective longitudinal	Normotensive pregnancies	100	Applanation tonometry, cfPWV	14, 24, 36 weeks
[18]	Prospective observational	Normotensive, GHTN, PE	120	Non-invasive CO monitor	12–40 weeks
[17]	Prospective longitudinal	Healthy singleton pregnancies	55	Flow-mediated dilation, pulse wave	10–40 weeks
[19]	Prospective observational	Obese vs. normal BMI	115 (65+50)	NICOM	14, 24, 36 weeks
[20]	Prospective pilot study	Pregnant, non-pregnant, postpartum	37 (14+14+9)	Quantitative MRI (vascular function)	~26 weeks (mean GA)
[21]	Synthesis of earlier studies	Aggregated from multiple sources	Not stated	Impedance cardiography, Doppler	1st–3rd trimester

DISCUSSION

This review synthesizes findings from nine studies on maternal hemodynamic and blood volume changes, highlighting normal adaptations and deviations in high-risk pregnancies. In healthy women, rising cardiac output and declining SVR in early to mid-pregnancy were consistently observed, supporting adequate placental perfusion and fetal growth [22-24]. A key trend observed was the biphasic nature of adaptation. CO tends to rise early and peak mid-gestation, while SVR decreases and then stabilizes or slightly rebounds by the third trimester. This pattern was supported by Aguree S and A.D. Gernand [25], who reported plasma volume increases of up to 48% by the third trimester, and by Lopes van Balen *et al.*, who described two phases of endothelial remodelling: rapid

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early expansion and later stabilization [26]. The discussion of high-risk pregnancies further underscores the importance of these adaptive processes. Studies focusing on women with fetal growth restriction (FGR), preeclampsia (PE), or obesity consistently reported persistently low CO and elevated SVR indicative of impaired cardiovascular remodelling [27, 28]. These findings were supported by previous research, including work by Ducas *et al.*, and Valensise *et al.*, which found that such deviations often precede clinical symptoms and may serve as early markers of poor placental function [29, 30]. These deviations also have clinical implications. The presence of abnormal maternal hemodynamics may be an early indicator of placental insufficiency and increased perinatal risk. Non-invasive methods like bioreactance monitoring and Doppler ultrasound, as suggested by Loreto *et al.*, and Ornaghi *et al.*, offer potential for earlier detection and more tailored prenatal care [27, 28]. Maternal characteristics, including age and obesity status, were also shown to influence cardiovascular adaptation. Ling *et al.*, found that younger women had more favourable CO and SVR profiles [14], while Patel *et al.*, and Kennedy *et al.*, reported that obesity leads to elevated SVR and reduced vascular compliance [19, 31]. These findings suggest that maternal anthropometry and metabolic profile should be factored into antenatal risk assessment. Although measurement modalities varied, ranging from NICOM and Doppler to MRI, the consistency of physiological trends supports their reliability. Langham *et al.*, [20] MRI-based vascular data reinforced earlier work by Duvekot and Peeters [32], who emphasized the interplay between vascular tone and cardiac function as central to pregnancy adaptation. Many studies lacked early first-trimester data, missing a crucial window where maladaptation may begin. Reijnders *et al.*, Mecacci *et al.*, and Warren et al., emphasized that elevated uterine artery resistance early in pregnancy can predict later complications [33-35].

This review is limited by the small number of eligible studies, heterogeneity in study designs, populations, and hemodynamic assessment methods, which restricted direct comparison across studies. Additionally, several studies lacked first-trimester data and included relatively small sample sizes, reducing the generalizability of findings. Future research should adopt standardized measurement protocols, include larger multicenter longitudinal cohorts beginning in early pregnancy, and evaluate the relationship between maternal hemodynamic profiles and maternal-fetal outcomes. Such studies may facilitate the development of personalized monitoring strategies and earlier identification of pregnancy-related complications.

CONCLUSIONS

This review confirms that pregnancy requires dynamic, trimester-specific cardiovascular adaptation characterized by increased cardiac output and reduced vascular resistance, which is crucial for fetal development. In high-risk pregnancies (fetal growth restriction (FGR), preeclampsia (PE), and obesity), these adaptations are often impaired and may signal early complications. Routine use of non-invasive hemodynamic monitoring could aid in early risk detection. Future studies should standardize measurements, begin assessments in the first trimester, and link maternal profiles with perinatal outcomes to support personalized care.

Authors' Contribution

Conceptualization: NF, MU
Methodology: SA, OK, MA, MU
Formal analysis: OK, NF
Writing and Drafting: AJ, SA, OK, MA, MU
Review and Editing: AJ, SA, OK, MA, MU

All authors approved the final manuscript and take responsibility for the integrity of the work

Conflicts of Interest

All the authors declare no conflict of interest.

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