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REVIEW ARTICLE

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Fetal heart rate variability and monitoring in gestational diabetes mellitus: a narrative review

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ABSTRACT

Objective: To evaluate evidence on fetal heart rate variability (HRV) in pregnancies complicated by gestational diabetes mellitus (GDM), focusing on cardiotocography (CTG), computerized CTG (cCTG), fetal electrocardiography (ECG), and magnetocardiography (MCG), and the influence of maternal glycemic control.

Study design: Narrative review synthesizing clinical and experimental studies retrieved from MEDLINE, EMBASE, and SCOPUS databases. Studies evaluating fetal HRV through CTG, cCTG, ECG, and MCG in GDM pregnancies were included.

Results: GDM is consistently associated with altered fetal HRV, particularly reduced short-term variation, elevated baseline fetal heart rate, and impaired autonomic balance. Advanced techniques such as ECG and MCG offer superior sensitivity compared to conventional CTG, especially in pregnancies with poor maternal glycemic control.

Conclusions: Fetal HRV assessment provides valuable insight into fetal well-being in GDM, suggesting potential clinical applications in antenatal monitoring. Standardization of methods and further prospective research are needed for clinical implementation.

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1. Introduction

Gestational diabetes mellitus (GDM) is one of the most common complications of pregnancy, with significant implications for both maternal and fetal health. It affects between 2% and 20% of pregnancies [1]. GDM is associated with higher risks of maternal hypertension, cesarean delivery, macrosomia, hypoglycemia, shoulder dystocia and stillbirth. Early identification and tight glycemic control are essential to reduce these risks [2]. Fetal heart rate (FHR) monitoring plays a central role in the antenatal surveillance of pregnancies complicated by GDM. Among the available tools, cardiotocography (CTG) and its computerized variant (cCTG) remain widely used to assess fetal well-being, particularly in women with suboptimal glycemic control or additional risk factors [3]. CTG simultaneously records the FHR and uterine activity, providing information on baseline heart rate, variability (HRV), accelerations, and decelerations, parameters that indirectly reflect fetal oxygenation and autonomic nervous system function [4]. Maternal hyperglycemia induces fetal hyperinsulinemia, which may contribute to increased oxygen consumption and impaired placental exchange. GDM is often accompanied by subclinical fetal compromise that CTG may help detecting [5,6]. However, CTG interpretation is subjective and inter-observer variability remains high. The optimal frequency and

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timing of CTG use in GDM are not clearly established. To overcome these limitations, cCTG has been developed as a more objective tool for analyzing fetal heart rate patterns. cCTG systems generate numerical outputs, such as short-term variation (STV), which quantify beat-to-beat fluctuations in FHR.

Recently innovative techniques such as fetal electrocardiography (ECG) and fetal magnetocardiography (MCG) offer a more accurate and physiologically grounded assessment of fetal HRV. Fetal ECG is a noninvasive modality that acquires fetal cardiac electrical signals allowing the direct measurement of R–R intervals and beat-to-beat heart rate dynamics, with high temporal resolution. Its advantages include portability, continuous monitoring potential and feasibility in outpatient settings; its effectiveness may be limited by maternal obesity, fetal position and signal contamination from maternal heart activity [7,8]. Fetal MCG measures the magnetic fields generated by the electrical activity of the fetal heart using superconducting quantum interference devices (SQUIDs). These highly sensitive sensors are capable of capturing extremely weak biomagnetic signals produced by the fetal myocardium, without requiring any direct contact with the maternal abdomen or fetal skin. This noninvasive nature makes MCG particularly safe and well tolerated, while also avoiding common artifacts associated with poor electrode coupling in ECG recordings [9]. Fetal ECG and MCG represent advanced tools for detecting early signs of fetal autonomic dysfunction, especially in high-risk pregnancies such as those complicated by GDM.

Aim of this narrative review is to describe how maternal glycemic control, in pregnancies complicated by GDM, may influence fetal HRV parameters assessed through CTG, cCTG, fetal ECG and MCG, according to current evidence.

2. Materials and methods

A systematic literature search was performed using the main search engines (MEDLINE, EMBASE, and SCOPUS) with all possible combinations of the following keywords: "gestational diabetes," "fetal monitoring," "cardiotocography," "cCTG," "FHR," "HRV," "fetal ECG," "magnetocardiography" and "maternal glycemic control." No language restrictions were applied to ensure comprehensive identification of relevant studies. Additional references were identified through backward citation tracking from key articles included in the initial search results. The inclusion criteria were:

- Study population: Pregnant women with GDM, regardless of the diagnostic criteria employed.
- Study design: Observational studies (prospective cohort, retrospective cohort, case-control), interventional studies, and experimental studies explicitly evaluating fetal HRV parameters or other FHR analyses.
- Monitoring modalities: Studies utilizing conventional CTG, cCTG, fetal ECG or MCG.
- Outcomes: Studies reporting quantitative or qualitative analyses of fetal HRV and their association with maternal metabolic parameters, glycemic status, pregnancy outcomes.

Studies were excluded if they met one or more of the following conditions:

- Animal studies or basic science research without clinical fetal HRV assessment.
- Studies involving non-diabetic pregnancies or mixed populations without clearly reported GDM subgroup analyses.
- Abstracts, editorials, letters to editors, or case reports

Relevant data from each selected article were extracted, including:

- First author, year of publication, and journal name.
- Study design, sample size, geographic setting, and methodology.
- Type of fetal monitoring employed (CTG, cCTG, ECG, MCG).
- Main outcomes regarding FHR variability parameters.
- Association between maternal glycemic status (HbA1c, insulin therapy) and fetal autonomic function.

Eight studies fulfilled all criteria; the selection process is detailed in Figure 1.

Due to significant methodological variability and heterogeneity of HRV metrics, a narrative synthesis was chosen over a formal meta-analysis. Extracted data were organized into summary tables to facilitate comparative assessment and interpretation of findings across different methodologies and studies.

3. Results

The literature search identified several key studies evaluating fetal HRV parameters using various monitoring methods (cCTG, ECG, MCG) in pregnancies complicated by GDM. We included 8 studies in this narrative review, differing in methodology, design, and settings, as summarized in Table 1.

3.1. cCTG monitoring in GDM pregnancies

We included four studies investigating cCTG) variations in pregnancies complicated by GDM.

Lobmaier et al. [10] conducted a prospective case-control applying an innovative signal processing technique known as phase-rectified signal averaging (PRSA) to assess fetal autonomic function. PRSA allows the detection of subtle periodic modulations within noisy and non-stationary signals like the FHR, providing two distinct metrics: average acceleration capacity (AAC) and average deceleration capacity (ADC), which separately reflect the sympathetic and parasympathetic components of the autonomic nervous system. The study demonstrated that fetuses of diabetic mothers

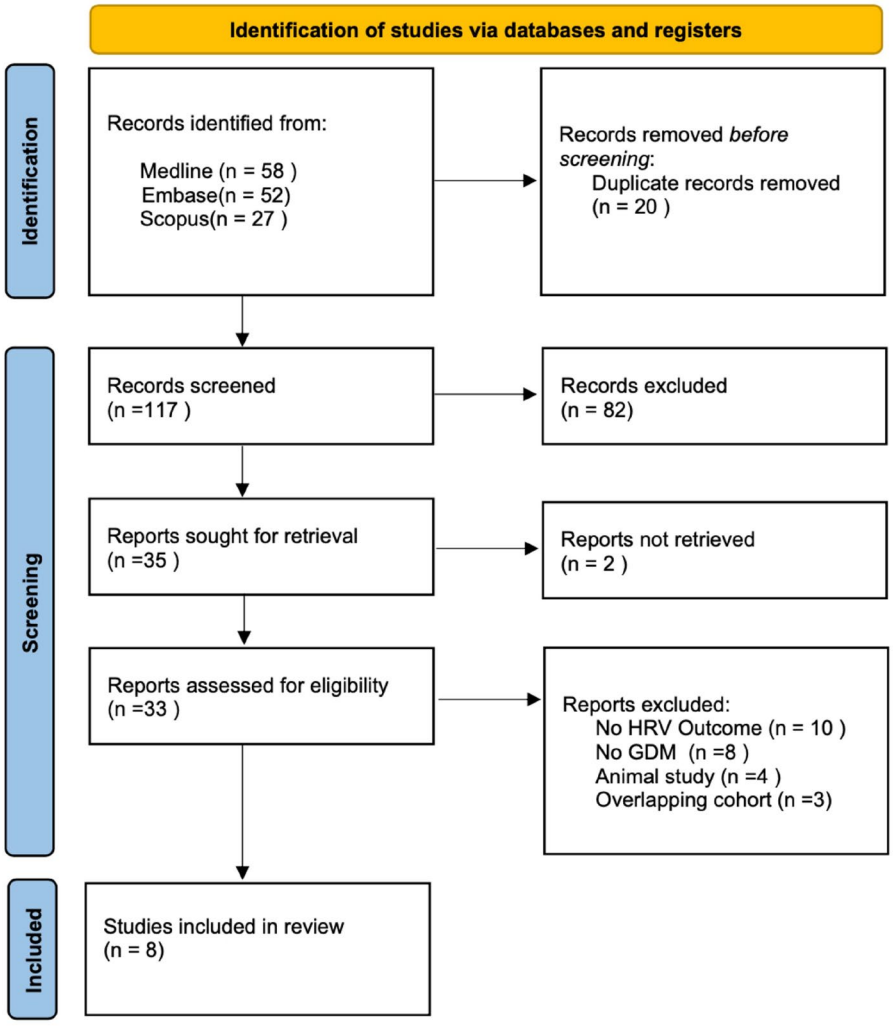

Figure 1. Flowchart of included study.

Table 1. Summary of studies on fetal HRV and monitoring in GDM pregnancies.

First author (year)	Sample size	Study design	Method	Setting	Main findings
Lobmaier (2018) [10]	116 participants (58 GDM, 58 controls) between 32 and 40w + 6d	Prospective case–control	cCTG (PRSA technique)	Germany, tertiary care	Increased fetal autonomic activity detected with PRSA; superior sensitivity compared to standard STV metrics.
Ruozi-Berretta (2004) [11]	73 participants (27 diabetic, 46 controls) > 37 weeks	Prospective cohort	cCTG	Italy, tertiary care	Significantly reduced STV and accelerations in diabetic pregnancies; lack of correlation between STV and fetal acid-base status in diabetic cases.
Buscicchio (2010) [12]	600 total (100 GDM on diet therapy, 100 GDM on insulin therapy, 100 control) between 35 and 36 weeks.	Comparative cohort	cCTG	Italy, tertiary care	Reduced STV, increased baseline fetal heart rate, prolonged episodes of low variability, indicating impaired fetal reactivity.
Gragnano (2022) [13]	298 participants (77 GDM, 105 controls, 89 DM I, 27 DM II)	Retrospective cohort	cCTG	Italy, tertiary care	Reduced HRV parameters, reflecting impaired autonomic regulation in GDM pregnancies.
Chivers (2024) [14]	96 participants (38 GDM, 58 controls) >20 weeks.	Prospective multicenter observational	Fetal ECG	UK, multicenter tertiary care	Higher fetal HRV indices negatively correlated with maternal HbA1c and BMI, indicating maternal metabolic impact on fetal autonomic function.
Fehlert (2016) [15]	49 (13 GDM, 36 controls) > weeks	Prospective observational	MCG	Germany, research center	HRV decreased significantly during oral glucose tolerance tests in GDM fetuses, indicating altered autonomic responses to acute glycemic variations.
Zöllkau (2021) [16]	19 GDM pregnancies monthly monitored at 27,31,35 and 39 weeks	Observational	MCG	Germany, research center	Maternal glucose levels correlated with fetal sympathetic activity; increased vagal modulation linked to improved metabolic control.
Mercado (2022) [17]	40 (28 diabetic, 12 controls) between 28 and 39 weeks	Observational	MCG	Germany and USA, multicenter research	Altered fetal HRV and impaired heart rate-movement coupling observed in pregestational diabetes, indicating disrupted sympathovagal balance.

exhibited significantly lower AAC and ADC values compared to healthy controls, indicating impaired autonomic regulation. Interestingly, conventional STV failed to detect these differences, underscoring the potential superiority of PRSA-based approaches in identifying early autonomic dysfunction in GDM pregnancies. No significant differences in PRSA-derived parameters were observed over time within the GDM group.

Ruozi-Berretta et al. [11] showed a significant reduction in STV and the number of FHR accelerations among diabetic pregnancies. However, they noted that STV did not correlate with umbilical blood gas parameters (pH and pCO_2) in diabetic cases, contrary to what was observed in controls. This finding raises concerns about the applicability of conventional cCTG criteria in assessing fetal well-being in diabetic pregnancies.

Buscicchio et al. [12] reported a significantly higher baseline FHR, longer episodes of low variability, and reduced STV in GDM pregnancies. These alterations suggest that even diet-controlled GDM can subtly affect fetal autonomic regulation, emphasizing the clinical utility of computerized analysis in refining antenatal surveillance strategies.

Finally, Gragnano et al. [13] found a consistent reduction in STV and other variability indices, reflecting impaired autonomic control. Their findings support the broader evidence that metabolic disturbances in GDM pregnancies can significantly impact fetal neurocardiac function, advocating for enhanced monitoring protocols in this population.

3.2. Fetal ECG and MCG variation in GDM pregnancies

In addition to cCTG studies, we found four studies that employed more advanced techniques, such as fetal ECG and fetal MCG to explore fetal HRV alterations in pregnancies complicated by GDM.

Chivers et al. [14] conducted a prospective multicenter observational study in which fetal ECG was recorded overnight using a noninvasive abdominal device. The authors found that, during quiet sleep (1 F behavioral state), fetuses of GDM mothers exhibited higher SDNN compared to controls. Moreover, a negative correlation was observed between fetal HRV and maternal body mass index (BMI) and HbA1c levels, while FHR positively correlated with worsening maternal metabolic control. These findings suggest that even moderate disturbances in maternal glycemic status can impact fetal autonomic development.

Fehlert et al. [15] used fetal MCG to monitor changes in fetal HRV during an oral glucose tolerance test (OGTT). The results demonstrated that while maternal HRV remained unaffected, fetuses of GDM mothers showed significant reductions in HRV parameters, including SDNN and spectral power in both low- and high-frequency bands, particularly during the metabolic challenge of the OGTT. These alterations indicate that GDM pregnancies may impair the fetal autonomic nervous system's ability to respond to acute metabolic stress.

Zöllkau et al. [16] performed longitudinal fetal MCG assessments and observed increased fetal vagal tone in GDM pregnancies, although sympathetic activation was positively associated with higher maternal glucose levels. Moreover, maternal glucose levels correlated with increased birthweight z-scores, suggesting a link between autonomic regulation and fetal growth patterns in the context of GDM.

Mercado et al. [17] investigated the effects of pregestational diabetes using fetal MCG recordings in a cohort that included both Type 1 and Type 2 diabetic mothers. Their findings showed alterations in the fetal sympathovagal balance, with an increased very-low-frequency/low-frequency (VLF/LF) ratio and reduced coupling between FHR accelerations and body movements. These disruptions were more pronounced in fetuses of mothers with poor glycemic control, reinforcing the concept that maternal metabolic status has a measurable impact on fetal autonomic nervous system development.

Together, these studies consistently highlight that both GDM and pregestational diabetes are associated with measurable alterations in fetal autonomic regulation., as detected by various fetal monitoring techniques. Advanced monitoring techniques such as fetal ECG and MCG provide more detailed physiological insights than conventional methods and may enhance early detection of subtle fetal compromise.

4. Discussion

This narrative review confirms that pregnancies complicated by GDM exhibit consistent alterations in fetal HRV, particularly characterized by reduced STV, diminished accelerations, and elevated baseline FHR. Supporting this hypothesis, Sirico et al. [18] demonstrated that as early as 11–14 weeks the baseline FHR is higher in pregnancies complicated by pre-gestational diabetes (161 vs. 158 bpm; $p = 0.02$), a difference that remains independent of maternal BMI, age and gestational age, indicating that sympatho-vagal imbalance may begin very early in gestation. These autonomic disturbances correlate strongly with poor maternal metabolic control, including elevated HbA1c levels, higher BMI, and insulin therapy requirements. Advanced fetal monitoring techniques, such as cCTG, fetal ECG, and fetal MCG, demonstrated superior sensitivity in detecting these subtle autonomic alterations compared to conventional CTG [3,4].

These findings are consistent with well-established evidence demonstrating that chronic hyperinsulinemia can impair autonomic balance by suppressing parasympathetic activity and stimulating sympathetic tone. This mechanism may operate in the fetus exposed to maternal hyperglycemia, leading to increased baseline FHR and reduced variability. Such autonomic imbalance is thus a plausible physiological consequence of chronic fetal hyperinsulinemia rather than an unexpected finding [19–21].

The findings of our review are further supported by recent literature; Sharifi-Heris et al. highlighted the growing role of HRV as a biomarker of autonomic dysfunction in high-risk pregnancies, although they emphasized the need for standardization in both maternal and fetal HRV assessment [22]. Similarly, the study by Zöllkau et al. [16] corroborated our observation that GDM can alter fetal autonomic regulation even in pregnancies with well-controlled glycemia, showing increased vagal

modulation but a concomitant rise in sympathetic activation associated with higher maternal glucose levels. These results are consistent with our findings that impaired maternal glycemic control negatively influences fetal autonomic maturation.

Additionally, the study by Ursem et al. focused on insulin-dependent diabetes mellitus and revealed that fetuses exposed to maternal diabetes displayed increased FHR variability and higher umbilical artery peak systolic velocity early in gestation [23]. These physiological adaptations may indicate underlying cardiovascular stress and align with the subtle HRV disturbances we identified across various studies using cCTG and MCG modalities.

Advanced signal processing methods, such PRSA applied in cCTG analysis [10], and high-resolution fetal ECG/MCG recordings [14–17], have proven critical for detecting fine-grained autonomic imbalances that traditional CTG may miss. Our findings regarding the superior performance of these technologies are strongly supported by these recent investigations, which demonstrated that fetal autonomic responses to acute glycemic changes and maternal metabolic status can be sensitively captured using advanced monitoring modalities.

Importantly, the consistent association between altered fetal HRV and maternal metabolic parameters across studies highlights HRV not merely as a surrogate marker of fetal well-being, but potentially as an early indicator of adverse intrauterine programming. Our review aligns with growing evidence suggesting that autonomic dysfunction detected antenatally may predispose the offspring to later cardiovascular and metabolic disorders [1,16], reinforcing the clinical significance of early HRV monitoring in pregnancies complicated by GDM.

Nevertheless, methodological heterogeneity remains a significant limitation. As observed both in previous systematic reviews [18] and in the studies included in our review, variations in diagnostic criteria for GDM, monitoring protocols, HRV parameter definitions, and population characteristics complicate direct comparisons. Most studies were observational and conducted in tertiary centers, which may limit the generalizability of results to broader clinical settings. Furthermore, inconsistencies in gestational age at monitoring and recording durations (e.g. 6 min to overnight recordings) pose challenges for standardizing HRV interpretation across different studies.

Despite these challenges, the strength and consistency of the findings across multiple methodologies and populations support the clinical relevance of HRV monitoring. Integrating cCTG-derived PRSA parameters, fetal ECG, and fetal MCG into routine antenatal care could enable early identification of fetuses at increased risk of autonomic dysfunction. Our review supports the notion that maternal glycemic optimization remains paramount, but that adjunctive fetal surveillance strategies based on HRV analysis may provide added value, particularly in high-risk GDM cohorts.

Interestingly, while reduced FHRV is frequently documented during the antenatal period in GDM pregnancies, these fetuses may paradoxically exhibit increased variability during labor, often manifesting as the "ZigZag" pattern described by Tarvonen et al. [24]. This apparent discrepancy likely reflects transient autonomic instability or acute hypoxic stress occurring during contractions, potentially exacerbated by fetal macrosomia and elevated metabolic demands. This phenomenon underscores the dynamic nature of fetal autonomic responses to labor-related stress in diabetic pregnancies.

According to the recommendations of the Italian Society of Gynecology and Obstetrics (SIGO), antenatal cardiotocographic monitoring in diabetic pregnancies should begin at 36 weeks of gestation in the absence of maternal or fetal complications and adequate glycemic control [25]. However, in the presence of pregestational diabetes, poorly controlled GDM or hypertension, FGR or polyhydramnios, surveillance should be initiated earlier, from 32 weeks. SIGO further emphasizes that the frequency and timing of surveillance should be individualized according to the clinical picture and the presence of additional risk factors.

These recommendations are consistent with those of the American College of Obstetricians and Gynecologists (ACOG) [26].

Cardiotocography (CTG) or cCTG remains the first-line method, with integration of the biophysical profile and Doppler velocimetry. Reduced STV or elevated baseline FHR in GDM should not be interpreted in isolation but as part of a comprehensive assessment including maternal glycemic control, fetal growth, and Doppler findings.

From a practical standpoint, reduced FHR variability or elevated FHR in fetuses of mothers with GDM should prompt clinicians to reassess maternal metabolic control, including glycemic levels and insulin therapy adequacy. When variability remains persistently low despite maternal metabolic optimization, additional testing is warranted—such as a repeat cCTG after accounting for fetal behavioral state, biophysical profile, or Doppler velocimetry. In cases of persistent tachycardia, suspected arrhythmia, or evidence of myocardial hypertrophy, referral for fetal echocardiography is recommended to exclude cardiomyopathies or cardiac rhythm abnormalities [27,28].

Future research should focus on prospective, multicenter studies employing standardized HRV acquisition and interpretation protocols, and should assess whether HRV-guided interventions can reduce perinatal morbidity. Technological advancements in wearable fetal monitoring devices may further facilitate real-time, noninvasive HRV assessment, paving the way for precision medicine approaches in obstetric care.

In conclusion, the synthesis of available evidence strongly corroborates our review findings: maternal dysglycemia significantly impairs fetal autonomic development, and advanced HRV analysis represents a promising tool for early detection of fetal compromise. Efforts toward standardization and clinical integration of these monitoring techniques are critical to translating research into practice and improving outcomes in pregnancies affected by gestational diabetes.

4.1. Methodological considerations and limitations

The reviewed studies exhibit notable heterogeneity in methodologies, including differences in study design (prospective vs retrospective), sample sizes, diagnostic criteria for GDM, and HRV measurement techniques. The majority of studies were observational and conducted in specialized tertiary care settings or research-oriented environments, potentially introducing selection bias and limiting the generalizability of findings to broader clinical populations.

Moreover, significant variability exists in how fetal HRV parameters are defined and interpreted across studies. Although computerized CTG, fetal ECG, and magnetocardiography (MCG) offer more objective assessments compared to traditional CTG, lack of standardized thresholds and uniform reporting of HRV metrics complicates direct comparisons between studies. This methodological inconsistency poses a substantial challenge in translating research findings into standardized clinical practice guidelines.

4.2. Clinical implications and future research

Despite methodological limitations, the consistent association between altered fetal HRV and poor maternal metabolic profiles underscores the clinical potential of advanced fetal monitoring techniques. Incorporating cCTG or ECG, MCG into routine antenatal surveillance protocols for GDM pregnancies could enable earlier identification of fetal autonomic disturbances.

In conclusion, while current evidence strongly supports the clinical relevance of HRV monitoring in GDM, significant methodological and practical challenges remain. Addressing these gaps through rigorous, standardized, and clinically oriented research will ultimately determine the optimal role of HRV analysis in routine antenatal care, potentially enhancing fetal safety and maternal health outcomes.

5. Conclusions

Fetal monitoring in pregnancies complicated by GDM remains an evolving and clinically important field. The integration of HRV analysis, particularly through cCTG, fetal ECG and MCG, provides valuable insights into fetal autonomic function and allows earlier identification of fetal compromise.

In pregnancies affected by GDM, reduced HRV or STV and elevated baseline FHR likely reflect an autonomic imbalance linked to maternal metabolic status.

A reduction in STV or an isolated increase in FHR should not in itself prompt delivery but rather trigger a comprehensive reassessment of maternal glycemic control and overall fetal condition. Current

evidence clearly demonstrates that poor maternal metabolic control adversely affects fetal HRV, underscoring the need for multidisciplinary and individualized management.

Future research should prioritize large-scale randomized trials, harmonization initiatives, and technological innovation to facilitate the translation of these promising monitoring methods into routine clinical practice.

Taken together, these data suggest that reduced HRV in GDM fetuses represents a physiological adaptation to chronic hyperinsulinemia and altered autonomic control, which may precede later intrapartum instability. Clinicians should, therefore, interpret HRV findings in the context of maternal metabolic status and consider targeted investigations when abnormalities persist or worsen.

Ultimately, integrating HRV monitoring into prenatal surveillance strategies could significantly enhance individualized clinical management, improve timing of interventions, and contribute to better maternal and neonatal outcomes in pregnancies affected by gestational diabetes.

Author contributions

CRediT: **Laura Sarno**: Conceptualization, Writing – original draft, Writing – review & editing; **Chiara Murolo**: Conceptualization, Data curation, Formal analysis, Methodology, Writing – original draft, Writing – review & editing; **Stefano Raffaele Giannubilo**: Software, Validation, Visualization; **Andrea Ciavattini**: Formal analysis, Investigation; **Maria Liberata Meccariello**: Data curation, Formal analysis, Validation; **Pietro D'Alessandro**: Supervision; **Mariarosaria Motta**: Methodology, Writing – review & editing; **Sara Iannantuoni**: Writing – review & editing; **Francesco D'Antonio**: Supervision; **Martina Derme**: Methodology, Supervision; **Marco Miceli**: Supervision; **Maurizio Guida**: Supervision, Validation, Visualization; **Giuseppe Maria Maruotti**: Conceptualization, Supervision, Visualization, Writing – original draft.

Ethical approval

Not required for narrative review.

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Data availability statement

No new datasets were generated or analyzed for this narrative review. All evidence summarized is drawn from the published studies cited in the References. The authors will make available, upon reasonable request, the data-extraction sheets and any additional materials supporting the synthesis, in accordance with Taylor & Francis' *Share Upon Reasonable Request* data policy.

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