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Research Paper

Compensatory pauses after ventricular and atrial premature beats and heart rate turbulence in the MIT-BIH arrhythmia database

Chaulagain Dipak[1*], Arynbaev Zhanybai[2], Narymbetov Talantbek[3]
[1*]Department of Clinical Disciplines, Jalal-Abad International University, 720906, 24 Kurmanbek Str., Manas, Kyrgyzstan.
[2,3]Department of Natural and Humanities Disciplines, Jalal-Abad International University, 720906, 24 Kurmanbek Str., Manas, Kyrgyzstan.

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ABSTRACT

Background: The interval that follows a premature beat is one of the oldest diagnostic clues in electrocardiography: a premature ventricular contraction (PVC) is classically followed by a full compensatory pause, whereas an atrial premature beat (PAC) resets the sinus node and is followed by a less-than-compensatory pause. The same post-ectopic interval also drives heart rate turbulence (HRT), an autonomic marker of cardiovascular risk.
Methods: This study quantified these phenomena in the 48 two-channel ambulatory recordings of the MIT-BIH Arrhythmia Database, which contain 109,492 cardiologist-verified beat annotations from 47 subjects aged 23–89 years. For isolated premature beats preceded by stable sinus rhythm, the pause ratio, the sum of the coupling and post-ectopic intervals divided by twice the sinus cycle length, was calculated, together with HRT onset and slope and time-domain heart rate variability.
Results: Among 1,177 qualifying PVCs from 32 records, the median pause ratio was 0.997 (interquartile range 0.960–1.035), and 60.5% of PVCs met a ±5% definition of a full compensatory pause; among 366 PACs from 23 records, the median was 0.952 (0.916–0.987), and 48.6% of pauses were less than compensatory ($p < 0.001$). Earlier PACs produced shorter relative pauses (slope 0.57 per unit prematurity, $p < 0.001$). Only 14 records had at least three PVCs suitable for HRT analysis; turbulence slope was normal (> 2.5 ms per beat) in 12, but turbulence onset was abnormal (≥ 0%) in 9, so that only 4 records showed normal HRT. Turbulence slope correlated strongly with heart rate variability (SDNN, $\rho = 0.78$) and inversely with heart rate ($\rho = -0.66$).
Conclusions: The textbook rules hold on average, but with wide individual scatter: about four in ten PVCs and half of PACs violate them. Pause analysis should therefore be used as supporting rather than decisive evidence for the origin of a premature beat, and HRT is measurable in only a minority of short recordings.

Corresponding Author:
Chaulagain Dipak
Department of Clinical Disciplines, Jalal-Abad International University, 720906, 24 Kurmanbek Str., Manas, Kyrgyzstan.
Email: neurodipak@gmail.com

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1. INTRODUCTION

Premature beats are the most common arrhythmias encountered on ambulatory electrocardiograms. Premature ventricular contractions (PVCs) occur in a large proportion of healthy adults during 24-hour monitoring and are usually benign, but a high burden is associated with left ventricular dysfunction, heart failure and death [1], [2], [3]. Atrial premature beats (PACs) are similarly common and have been linked to atrial fibrillation and stroke. Distinguishing the two, and recognising aberrantly conducted PACs that mimic PVCs, is a daily task in electrocardiographic interpretation.

One of the oldest clues is the length of the pause that follows the premature beat. Because a PVC usually does not penetrate and reset the sinus node, the next sinus impulse arrives on schedule, and the interval from the beat before the PVC to the beat after it equals two sinus cycles, a full compensatory pause. A PAC, in contrast, depolarises the sinus node prematurely and resets its timing, so that the following pause is less than compensatory [4], [5]. These rules are taught to every medical student, yet their quantitative reliability in ambulatory recordings has rarely been reported with reproducible data.

The post-ectopic pause also has physiological consequences. The transient fall in arterial pressure caused by the premature beat and the compensatory pause triggers a baroreflex-mediated early acceleration followed by a late deceleration of sinus rhythm, a pattern termed heart rate turbulence (HRT) [6]. Its two components, turbulence onset (TO) and turbulence slope (TS), are reduced in patients with impaired autonomic function, and abnormal HRT is a strong independent predictor of mortality after myocardial infarction [6], [7], [8]. Heart rate variability (HRV) provides a complementary measure of autonomic modulation [9], [10].

The MIT-BIH Arrhythmia Database is the reference standard for arrhythmia analysis. Its 48 recordings were annotated beat by beat by cardiologists and have been freely available for decades [11], [12]. Although it is used mainly to test detection algorithms, it is also a rich source of clinical electrocardiographic data. This study used it to answer three questions: (i) how often do PVCs and PACs follow the textbook compensatory-pause rules; (ii) how does prematurity affect the pause; and (iii) how measurable is HRT in short ambulatory recordings, and how does it relate to heart rate and HRV?

2. METHODOLOGY

2.1 Data Source

The MIT-BIH Arrhythmia Database (version 1.0.0) contains 48 half-hour excerpts of two-channel ambulatory ECG recordings from 47 subjects studied by the Beth Israel Hospital Arrhythmia Laboratory between 1975 and 1979 [11]. The recordings were digitised at 360 samples per second with 11-bit resolution over a 10 mV range. The upper channel is a modified limb lead II (MLII) in most records, and the lower channel is usually V1. Each beat was annotated independently by two or more cardiologists, with disagreements resolved by consensus. Record headers include the subject's age, sex and medications. The database is openly available from PhysioNet under the Open Data Commons Attribution licence [12], [13]; a byte-identical copy whose checksums matched the original headers was used. Because the data are

anonymised and publicly released, no ethical approval was required. Representative ECG strips from the database are shown in Figure 1.



Figure 1. Excerpts (8 s, Lead MLII; Grid 0.5 MV × 0.2 s) of Real Recordings from the Database Illustrating Normal Sinus Rhythm, Ventricular Bigeminy, Atrial Fibrillation, Ventricular Flutter, Paced Rhythm and Atrial Premature Beats. Letters Above the Complexes are the Reference Beat Annotations (N, Normal; V, Ventricular Premature; A, Atrial Premature; R, Right Bundle Branch Block; /, Paced)

2.2 Beat Selection and Compensatory Pause

All beat annotations were read with the WFDB software package. Beats labelled N, L or R (normal or bundle-branch-block beats of sinus origin) were treated as sinus beats. PVCs were beats labelled V; PACs were beats labelled A, a, J or S. A premature beat was included in the pause analysis if it was preceded by at least four consecutive sinus beats and followed by a sinus beat, if the three sinus RR intervals before the coupling interval varied by less than 10% (coefficient of variation), and if the coupling interval was shorter than 92% of the mean preceding sinus RR interval. For each beat, the prematurity index (coupling interval divided by mean sinus RR), the post-ectopic interval and the pause ratio were calculated:

pause ratio = (coupling interval + post-ectopic interval) / (2 × mean sinus RR interval).

A ratio of 1 indicates a fully compensatory pause. Pauses were classified as full (0.95–1.05), less than compensatory (< 0.95) or more than compensatory (> 1.05); the ±5% band allows for normal beat-to-beat sinus variability.

2.3 Heart Rate Turbulence

HRT was calculated according to the international consensus [8]. A PVC qualified if it was preceded by at least two and followed by at least 15 sinus beats, if all these RR intervals lay between 300 and 2,000 ms and within 20% of their mean, if prematurity was at least 20% and if the post-ectopic interval was at least 120% of the preceding sinus intervals. Records with at least three qualifying PVCs were analysed. Turbulence onset was calculated from the record-averaged tachogram as $TO = [(RR_1 + RR_2) - (RR_{-2} + RR_{-1})] / (RR_{-2} + RR_{-1}) \times 100\%$, and turbulence slope as the maximum positive slope of a regression line fitted to any five consecutive RR intervals among the first 15 sinus intervals after the PVC. TO < 0% and TS > 2.5 ms per RR interval were considered normal; records were classified as HRT0 (both normal), HRT1 (one abnormal) or HRT2 (both abnormal) [7], [8].

2.4 Heart Rate Variability and Statistics

For each record without pacing or atrial fibrillation, normal-to-normal (NN) intervals between four consecutive sinus beats were extracted, and the standard deviation of NN intervals (SDNN) and the root mean square of successive differences (RMSSD) were calculated [9]. Because the recordings last 30 minutes, these values correspond to short-term rather than 24-hour HRV. PVC burden was expressed as the percentage of all beats. Pause ratios of PVCs and PACs were compared with the Mann–Whitney U test and, within records containing at least five of each, with the Wilcoxon signed-rank test. The relation between pause ratio and prematurity was estimated by linear regression with standard errors clustered by record. Spearman correlation was used for record-level associations. Analyses were performed in Python with the WFDB, NumPy, SciPy and statsmodels libraries.

3. RESULTS AND DISCUSSION

3.1 Recordings

Table 1 summarises the recordings. The median age was 67 years (range 23–89), 26 of the 48 records came from men, and the database contained 109,492 annotated beats, including 7,129 PVCs and 2,779 supraventricular premature beats. PVC burden was highly skewed: the median was 1.2% of beats, but 11 records had a burden of 10% or more, with a maximum of 33.6% (Figure 2b). Four records were predominantly paced and eight contained episodes of atrial fibrillation or flutter.

Table 1. Characteristics of the 48 Ambulatory ECG Recordings

Characteristic	Value
Records / subjects	48 / 47 (records 201 and 202 from one subject)
Age, median (range), years	67 (23–89); not recorded for 2
Men / women (records)	26 / 22
Recording	30 min, 2 channels, 360 Hz, 11-bit
Annotated beats	109,492
Normal / bundle-branch-block beats	75,054 / 15,333 (LBBB 8,074; RBBB 7,259)
Ventricular premature beats	7,129
Atrial, aberrant, nodal or supraventricular premature beats	2,779
Paced and pacemaker-fusion beats	8,010 (records 102, 104, 107, 217)
Records with atrial fibrillation or flutter	8
Median heart rate, bpm	74.6 (IQR 66.3–85.5)
PVC burden, median (IQR), % of beats	1.2 (0.04–7.3); maximum 33.6
Records with PVC burden ≥1% / ≥10%	24 / 11
Records listing a beta-blocker / digoxin	7 / 26

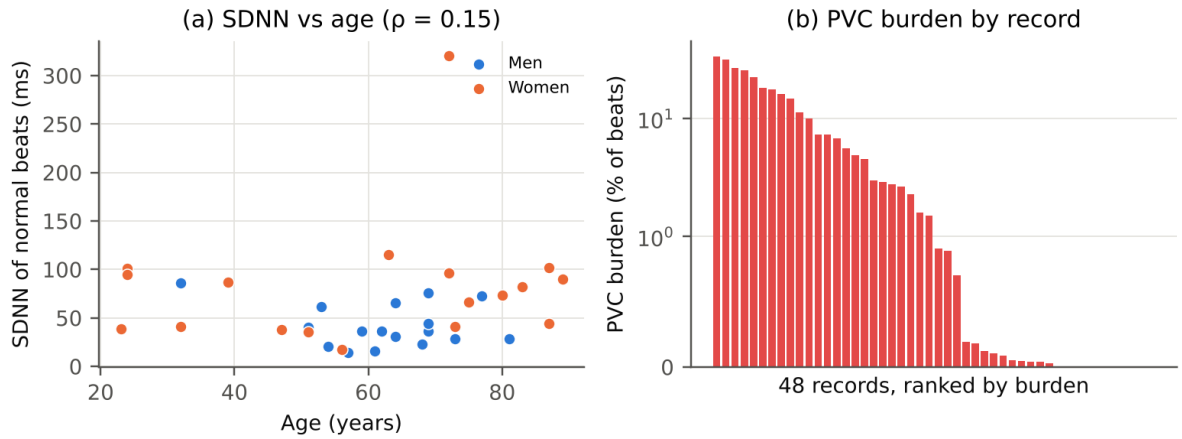


Figure 2. (A) Short-Term SDNN Against Age in Records Without Pacing or Atrial Fibrillation. (B) PVC Burden in the 48 Records, Ranked from Highest to Lowest (Logarithmic Scale Above 1%)

3.2 Compensatory Pauses

Figure 3 shows a typical example of each rule. In record 119, a PVC interrupted sinus rhythm with a coupling interval plus post-ectopic interval of 1.84 s against two sinus cycles of 1.81 s, a full compensatory pause. In record 209, an atrial premature beat was followed by a pause that made the two intervals sum to 1.20 s against two sinus cycles of 1.24 s (pause ratio 0.97), a shorter-than-compensatory pause, although one that falls within the ±5% band classed as full in the quantitative analysis; this example itself shows how narrow the margin between the two categories can be.

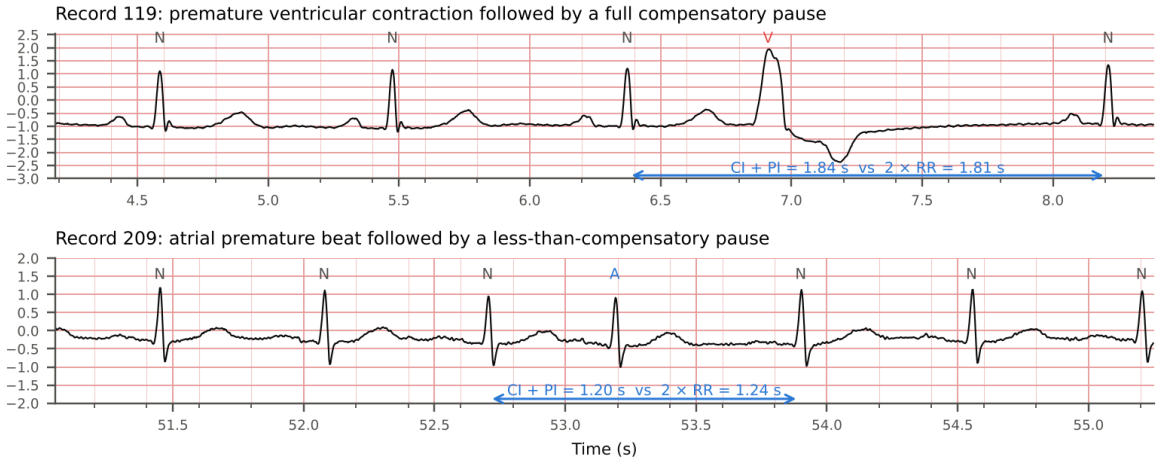


Figure 3. Measurement of the Post-ectopic Pause in Real Recordings. Top: a Premature Ventricular Contraction (V) Followed by a Full Compensatory Pause. Bottom: an Atrial Premature Beat (A) Followed by a Shorter-than-compensatory Pause (Ratio 0.97). CI, Coupling Interval; PI, Post-ectopic Interval; RR, Mean of the Preceding Sinus Cycles

Across the database, 1,177 PVCs from 32 records and 366 PACs from 23 records met the inclusion criteria (Table 2, Figure 4). PVCs were more premature than PACs (median prematurity index 0.66 vs 0.77). The median pause ratio was 0.997 for PVCs and 0.952 for PACs ($p < 0.001$). For PVCs, 60.5% of pauses were full, 21.8% less than compensatory and 17.8% more than compensatory; in 81% of records, the median PVC pause was full. For PACs, 46.7% of pauses fell within the full band and 48.6% were less than compensatory, whereas only 4.6% were longer. Relative to the sinus cycle, the post-ectopic interval was 1.32 times longer after PVCs and 1.13 times longer after PACs. In the five records with at least five beats of each type, the median ratio was lower for PACs than for PVCs in every case (0.961 vs 0.988; $p = 0.06$).

Table 2. Post-ectopic Pauses after Isolated Premature Ventricular and Atrial Beats

Beat Type	Beats	Records	Median Prematurity	Pause Ratio, Median (IQR)	Full, %	Less Than Compensatory, %	More Than Compensatory, %	Records with Full Median Ratio, %
PVC	1177	32	0.66	0.997 (0.960–1.035)	60.5	21.8	17.8	81
PAC	366	23	0.77	0.952 (0.916–0.987)	46.7	48.6	4.6	52

The pause depended on prematurity, particularly for PACs (Figure 4b). With every 0.1 increase in prematurity index (that is, a later beat), the pause ratio rose by 0.057 for PACs ($p < 0.001$) and by 0.029 for PVCs ($p = 0.006$). Early PACs thus produced clearly incomplete pauses, consistent with sinus node reset, whereas late PACs, which may arrive too late to penetrate the sinus node, produced near-complete pauses that cannot be distinguished from those of PVCs. More-than-compensatory pauses after PVCs, seen in about one in six cases, are compatible with retrograde concealed conduction or transient sinus slowing.

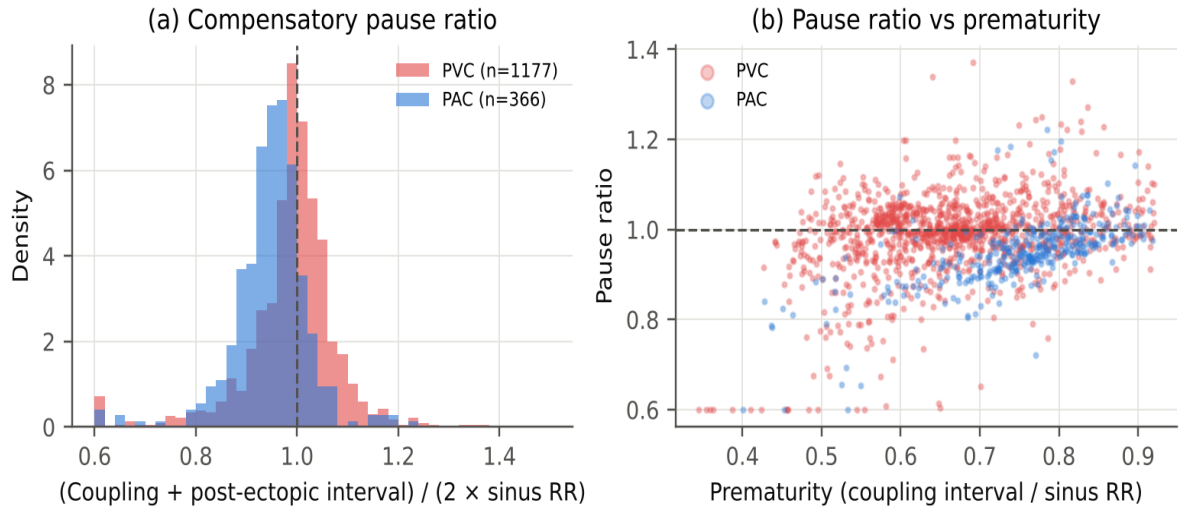


Figure 4. (A) Distribution of the Pause Ratio for Premature Ventricular Contractions (PVC) And Atrial Premature Beats (PAC); The Dashed Line Marks a Fully Compensatory Pause (Ratio 1). Values Outside 0.6–1.5 are shown at the Plot Limits. (B) Pause Ratio Against Prematurity of the Premature Beat

3.3 Heart Rate Turbulence

Only 14 records, contributing 185 PVCs, met the HRT requirements of at least three PVCs with stable surrounding sinus rhythm (Table 3, Figure 5). The median TO was +0.71% (interquartile range −0.43 to +1.84%), and the median TS was 6.2 ms per beat (2.8–12.4). TS was normal in 12 of the 14 records, but TO was abnormal (≥ 0%) in 9, so that 4 records were classified as HRT0, 9 as HRT1 and 1 as HRT2. The record-averaged tachograms (Figure 5a) show that the expected early acceleration was absent or small in most records, while the late deceleration was visible in many. TS correlated strongly and positively with SDNN ($\rho = 0.78$, $p = 0.001$) and negatively with heart rate ($\rho = -0.66$, $p = 0.010$), but not with age ($\rho = 0.16$, $p = 0.58$).

Table 3. Heart Rate Turbulence in the 14 Records with at Least Three Qualifying PVCs

Record	Age, Sex	PVCs	HR, Bpm	TO, %	TS, ms/beat	Category	Medication
105	73 F	32	85	+0.82	2.0	HRT2	digoxin
106	24 F	3	64	+3.42	15.6	HRT1	β-blocker
108	87 F	10	58	-3.99	17.9	HRT0	digoxin
109	64 M	4	84	+2.90	11.8	HRT1	
114	72 F	9	64	-0.29	7.9	HRT0	digoxin
116	68 M	39	80	-0.73	1.4	HRT1	
119	51 F	5	66	+3.74	6.0	HRT1	
124	77 M	3	53	+1.85	6.4	HRT1	digoxin
202	68 M	9	68	-2.94	12.6	HRT0	β-blocker, digoxin
205	59 M	13	89	+0.60	3.0	HRT1	digoxin
214	53 M	21	76	-0.48	4.9	HRT0	digoxin
215	81 M	21	112	+1.22	2.7	HRT1	
228	80 F	13	69	+1.81	16.9	HRT1	digoxin
234	56 F	3	91	+0.35	2.7	HRT1	

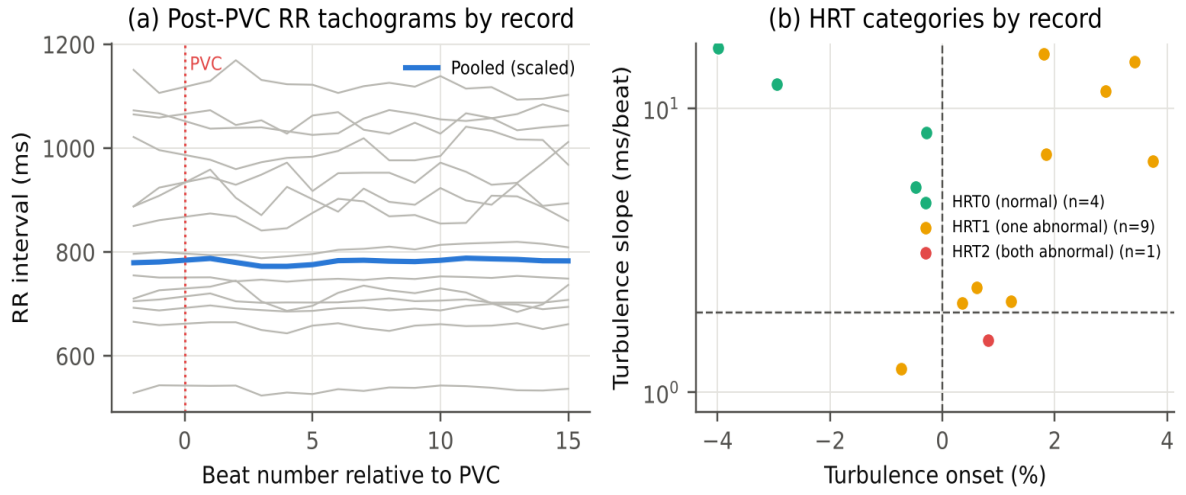


Figure 5. Heart Rate Turbulence. (a) Averaged RR Tachograms Around Qualifying PVCs in Each of the 14 Analysable Records (Grey) and the Pooled Tachogram (Blue), with the PVC at Beat 0. (b) Turbulence Onset and Slope by Record; Dashed Lines Mark the Normal Thresholds (TO < 0%, TS > 2.5 ms/beat)

3.4 Heart Rate Variability and PVC Burden

In 36 records without pacing or atrial fibrillation and with sufficient NN intervals, the median SDNN over 30 minutes was 44 ms (interquartile range 36–83) and the median RMSSD 32 ms (24–53). SDNN was not associated with age in this small, clinically heterogeneous sample ($\rho = 0.15$, $p = 0.39$), and values varied widely among older subjects (Figure 2a).

3.5 Discussion

Using the most widely studied annotated ECG database, this study quantified two classical electrocardiographic phenomena that are usually taught qualitatively. On average, the rules hold: PVCs were followed by a pause that almost exactly completed two sinus cycles, and PACs by a shorter pause. The variation around these averages was, however, large. Four in ten PVCs did not meet a ±5% definition of a full compensatory pause, and only about half of PACs produced a clearly incomplete pause.

These findings have practical implications for ECG interpretation. Wide-complex premature beats are sometimes aberrantly conducted PACs rather than PVCs, and the pause is often cited as a distinguishing clue [4], [5]. The present data show that the pause can support but not settle the distinction. A clearly less-

than-compensatory pause after an early premature beat favours an atrial origin, but a full pause is compatible with both, particularly for late PACs that arrive after the sinus node has already fired or that fail to reach it. Conversely, sinus arrhythmia, which changes the reference cycle length from beat to beat, and retrograde conduction of PVCs into the atria and sinus node can make PVC pauses incomplete or overcompensated [5]. Morphology of the P wave and QRS complex, coupling-interval consistency and the response of the following sinus beats remain essential.

The strong dependence of PAC pauses on prematurity agrees with the physiology of sinus node reset. An early PAC penetrates and resets the sinus node, so that the next sinus beat follows one sinus cycle (plus conduction time) after the PAC, whereas a late PAC may collide with the emerging sinus impulse and leave the sinus rhythm undisturbed. This continuum explains why late PACs behave like PVCs in pause analysis.

The HRT findings illustrate both the potential and the limitations of this marker. Most records showed preserved late deceleration (normal TS) but an absent early acceleration (TO ≥ 0%). This pattern is plausible in a largely elderly population, drawn from both inpatients and outpatients, with frequent structural heart disease and frequent use of rate-modifying drugs such as digoxin [11]. The strong correlation between TS and SDNN supports the interpretation of TS as a measure of vagal baroreflex function [8], and the inverse relationship with heart rate is consistent with the known rate dependence of HRT, which complicates comparisons between patients [8]. More importantly for practice, only 14 of 48 records yielded enough qualifying PVCs, because frequent ectopy, bigeminy, atrial fibrillation and pacing disrupt the sinus sequences that HRT requires. Validated HRT risk stratification relies on 24-hour recordings [6], [7], and the present data confirm that short recordings rarely suffice.

The short-term SDNN of 44 ms and RMSSD of 32 ms lie within the broad range expected for older adults with heart disease [10], [14]. The absence of an age gradient is not surprising in 36 heterogeneous records with differing medications and conduction disorders, and does not contradict the well-established decline of HRV with age in healthy populations [14]. The skewed distribution of PVC burden, with a quarter of records above 7%, reflects the deliberate selection of recordings containing clinically significant arrhythmias [11].

3.5.1 Methodological Considerations

The strengths of this study are its use of cardiologist-verified beat annotations rather than automated detection, its explicit and reproducible beat-selection rules, the open availability of the data, and the presentation of real ECG strips that readers can inspect.

The limitations reflect the database. The recordings are 30-minute excerpts from 1975–1979, mostly from older patients, about 60% of them inpatients, chosen to include unusual arrhythmias; they are not representative of a general population. Clinical information is limited to age, sex and medications, so the associations of HRT with disease and outcomes could not be examined. Only 14 records were suitable for HRT, so those results are descriptive. The PAC category combined atrial, aberrant, nodal and supraventricular premature beats, which may differ in their relationship to the sinus node. Finally, the ±5% definition of a full compensatory pause is a pragmatic choice; other thresholds would change the percentages but not the contrast between PVCs and PACs.

4. CONCLUSION

In 1,543 premature beats from the MIT-BIH Arrhythmia Database, PVCs were followed on average by a full compensatory pause and PACs by a less-than-compensatory pause, but about 40% of PVCs and half of PACs did not follow the textbook rule, and late PACs behaved like PVCs. The post-ectopic pause is therefore supporting rather than decisive evidence for the origin of a premature beat. Heart rate turbulence could be measured in fewer than a third of 30-minute recordings; where measurable, its slope tracked heart rate variability closely. Clinicians should interpret pauses in the context of prematurity and morphology, and HRT should be assessed from longer recordings.

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Author Contributions Statement

Name of Author	C	M	So	Va	Fo	I	R	D	O	E	Vi	Su	P	Fu
Chaulagain Dipak	✓	✓	✓	✓		✓	✓	✓	✓	✓	✓		✓	✓
Arynbaev Zhanybai	✓		✓	✓	✓	✓	✓		✓	✓		✓	✓	
Narymbetov Talantbek	✓	✓		✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓

C : **C**onceptualization
M : **M**ethodology
So : **So**ftware
Va : **Va**lidation
Fo : **Fo**rmal analysis
I : **I**nvestigation
R : **R**esources
D : **D**ata Curation
O : Writing - **O**riginal Draft
E : Writing - Review & **E**diting
Vi : **Vi**sualization
Su : **Su**pervision
P : **P**roject administration
Fu : **Fu**nding acquisition

Conflict of Interest Statement

The authors declare that there are no conflicts of interest regarding the publication of this article.

Informed Consent

Not applicable. This study analysed publicly available, de-identified data; no participants were recruited.

Ethical Approval

This study is a secondary analysis of anonymised electrocardiographic recordings that are publicly available from PhysioNet. No new data were collected from human participants, and ethical approval and informed consent were therefore not required.

Data Availability

The MIT-BIH Arrhythmia Database is openly available from PhysioNet (doi:10.13026/C2F305) under the Open Data Commons Attribution licence. ECG excerpts shown in Figure 1 and Figure 3 are drawn from it. The analysis code (Python, WFDB) is available from the corresponding author on reasonable request.

Use of AI Tools

Generative AI tools were used to assist with code development, statistical analysis and drafting of the manuscript. The authors reviewed and verified all analyses, results and text and take full responsibility for the content of this article.

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BIOGRAPHIES OF AUTHOR



Chaulagain Dipak, is an Associate Professor in the Department of Clinical Disciplines at Jalal-Abad International University, Manas, Kyrgyzstan, where the author teaches and conducts research in clinical medicine. For this article, Chaulagain Dipak was responsible for conceptualization, clinical and autonomic interpretation of heart rate turbulence and variability, writing – original draft. Email: neurodipak@gmail.com

	**Arynbaev Zhanybai**, is a Professor in the Department of Natural and Humanities Disciplines at Jalal-Abad International University, Manas, Kyrgyzstan, where the author teaches and conducts research in the natural sciences and humanities. For this article, Arynbaev Zhanybai was responsible for methodology, signal-processing and mathematical analysis of RR-interval series, supervision, writing – review and editing. Email: aryn-jan@mail.ru
	**Narymbetov Talantbek**, is an Associate Professor in the Department of Natural and Humanities Disciplines at Jalal-Abad International University, Manas, Kyrgyzstan, where the author teaches and conducts research in the natural sciences and humanities. For this article, Narymbetov Talantbek was responsible for software, data curation of beat annotations, formal analysis, visualization, writing – review and editing. Email: talant83@mail.ru