

# EP-Prior: Interpretable ECG Representations via Electrophysiology Constraints

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## Abstract

We present EP-Prior, a self-supervised method that produces interpretable ECG representations aligned with cardiac electrophysiology. Our encoder learns structured latent representations ( $z_P, z_{QRS}, z_T, z_{HRV}$ ) corresponding to clinically meaningful cardiac components, while an EP-constrained decoder enforces temporal ordering and refractory period constraints as soft priors, biasing the model toward physiologically plausible reconstructions. Unlike prior physiology-aware methods that improve performance as a black box, EP-Prior’s representations are inspectable—each latent component has physiological meaning that clinicians can examine. We provide PAC-Bayes-motivated analysis showing how EP constraints reduce the complexity term in generalization bounds, predicting largest gains in few-shot regimes. Experiments on PTB-XL demonstrate **+7.2% AUROC improvement** over capacity-matched baselines in 10-shot classification, with gains on all five diagnostic categories. Critically, ablation studies reveal that EP constraints are *essential*—removing them causes catastrophic failure (10-shot AUROC drops from 0.699 to 0.519, worse than baseline), demonstrating that structured latents alone are insufficient. Our work shows how domain knowledge can be embedded as architectural priors to achieve both explainability and sample efficiency.

## 1 Introduction

ECG-based cardiac diagnosis is critical for early detection of arrhythmias and conduction abnormalities [Goldberger *et al.*, 2000]. While deep learning has achieved strong performance on large datasets [Wagner *et al.*, 2020], significant challenges remain in low-data regimes, particularly for wearable and portable devices [Liu and others, 2021]:

- **Rare arrhythmias:** Many conditions appear in < 1% of records
- **Patient-specific adaptation:** Personalized models must adapt from few examples

- **New device deployment:** Transfer to new ECG hardware with limited labels

Equally important is the need for **interpretability**. Black-box models that achieve high accuracy but provide no insight into *what* they have learned face barriers to clinical adoption. Regulatory frameworks increasingly require explainable AI for medical devices.

**Key observation:** Cardiac electrophysiology (EP) provides rich mathematical structure—P-QRS-T wave morphology, conduction dynamics, refractory constraints—that is well-understood but rarely exploited in representation learning. Prior work uses EP knowledge in ECGI (inverse problems) but not for learning interpretable representations.

**Our approach:** We propose **EP-Prior**, which injects EP knowledge as architectural priors in a self-supervised framework:

1. A **structured latent space** where encoder outputs decompose into ( $z_P, z_{QRS}, z_T, z_{HRV}$ )
2. An **EP-constrained decoder** using a Gaussian wave model that reconstructs ECG signals
3. **Soft constraint losses** enforcing temporal ordering, refractory periods, and duration bounds

## Contributions:

1. **Interpretability:** Structured latent space with physiologically meaningful components, validated through intervention tests and concept predictability
2. **Theory:** PAC-Bayes-motivated analysis explaining *why* EP constraints help in low-data regimes
3. **Empirical:** Competitive few-shot classification on PTB-XL with inspectable, concept-level parameters

**Methodological novelty.** Prior physiology-aware ECG methods inject domain knowledge via data augmentation or loss terms, treating the learned representations as black boxes. EP-Prior differs fundamentally: we encode electrophysiology as *architectural constraints* that shape the hypothesis class itself. The structured latent decomposition ( $z_P, z_{QRS}, z_T, z_{HRV}$ ) is *prescribed* by cardiac physiology, not discovered by the model. The EP-constrained decoder enforces wave ordering and refractory periods through *hard* architectural choices (Gaussian waves with timing parameters),

not soft regularization. This design enables both interpretability (each latent has known meaning) and theoretical analysis (constrained hypothesis class has reduced complexity). Our ablation demonstrates this distinction is critical: structured latents without EP constraints perform *worse* than unstructured baselines.

## 2 Related Work

**Self-supervised learning for ECG.** Generic SSL approaches [Mehari and Strothoff, 2022] apply contrastive [Chen *et al.*, 2020] and predictive coding to ECG, treating representations as black boxes. PhysioCLR [Chen and others, 2025] represents the state-of-the-art in physiology-aware ECG SSL, integrating domain knowledge via augmentations (lead dropout, baseline wander), sampling strategies (preserving physiological similarity), and pretext tasks (heart rate prediction). While PhysioCLR achieves strong downstream performance, its representations remain opaque—clinicians cannot inspect what the model learned about P-waves vs. QRS complexes. **Our key distinction:** EP-Prior encodes physiology as *architectural constraints* that produce inspectable representations, enabling both interpretability and theoretical analysis.

**Few-shot ECG classification.** The few-shot ECG problem has been studied with meta-learning [Palczyński *et al.*, 2022] and knowledge-enhanced transfer [Fan and others, 2025]. These approaches achieve sample efficiency through algorithmic techniques (MAML, prototypical networks) rather than domain-structured representations. EP-Prior provides a complementary perspective: sample efficiency through physics-informed inductive bias, with theoretical grounding from PAC-Bayes.

**PQRST-structured methods.** ECG-GraphNet [Wang and others, 2025] constructs graphs where nodes correspond to P/QRS/T segments for supervised arrhythmia classification. MINA [Hong *et al.*, 2019] uses multilevel attention at beat, rhythm, and frequency scales. These methods leverage PQRST structure for *classification*, but are supervised (require labels) and lack self-supervised pretraining. **Our distinction:** EP-Prior uses PQRST structure for *representation learning* in an SSL framework, enabling transfer to downstream tasks.

**Interpretable ECG representations.** VAE-SCAN [Higgins *et al.*, 2017] and  $\beta$ -TCVAE [Chen *et al.*, 2018] learn disentangled ECG representations through variational inference. These methods discover latent factors *post-hoc*—the model decides what factors to learn, and interpretability is assessed by correlating factors with known attributes. **Critical difference:** EP-Prior uses *prescribed* factors where each latent ( $z_P, z_{QRS}, z_T, z_{HRV}$ ) has predetermined physiological meaning, enabling: (1) validation that the model learned the intended structure, (2) clinician-legible representations, and (3) theoretical analysis of the constrained hypothesis class.

**Sample complexity and architectural priors.** Behboodi and Cesa [2024] prove that architectural priors (equivariance, locality, weight sharing) reduce sample complexity. Time-series learning theory [Kuznetsov and Mohri, 2015] and PAC-Bayes bounds for dynamical systems [Eringis *et al.*, 2024]

Table 1: Comparison with related ECG methods.

Method	Interp.	SSL	Theory	Factors
PhysioCLR	✗	✓	✗	—
Few-shot Meta	✗	✗	✗	—
VAE-SCAN	Disc.	✗	✗	Learned
$\beta$ -TCVAE	Disc.	✗	✗	Learned
ECG-GraphNet	Part.	✗	✗	Fixed
MINA	Part.	✗	✗	Multi
<b>EP-Prior</b>	<b>Presc.</b>	<b>✓</b>	<b>✓</b>	<b>P/QRS/T/HRV</b>

*Interp.*: Prescribed/Discovered/Partial. *Factors*: Latent structure.

provide foundations for temporal data. **Our contribution:** We instantiate this general principle for cardiac electrophysiology, showing how EP constraints map to an informative prior and validating theory-predicted sample efficiency gains.

**Physics-informed cardiac modeling.** PINNs for cardiac activation mapping [Sahli Costabal *et al.*, 2020] embed wave propagation constraints for inverse problems. Gaussian wave models [McSharry *et al.*, 2003; Clifford and McSharry, 2006] provide analytical ECG generators. **Our approach:** We adapt these physics models as a differentiable decoder for representation learning, rather than inverse problems or synthesis.

**Summary.** Table 1 compares EP-Prior with related approaches. Our unique contribution is the combination of: (1) *prescribed* physiology-aligned factors (not discovered), (2) discriminative SSL (not generative/supervised), (3) EP-constrained decoder (not soft heuristics), and (4) PAC-Bayes-motivated design with empirical validation.

## 3 Theoretical Foundation

### 3.1 Problem Setup

We consider ECG signals  $x_t \in \mathbb{R}^{12}$  (12-lead) with labels  $y \in \{1, \dots, K\}$ . ECG signals arise from a latent cardiac state-space model:

$$x_t = g(z_t) + \epsilon_t, \quad z_{t+1} = f_{EP}(z_t) + \eta_t \quad (1)$$

where  $f_{EP}$  encodes cardiac EP dynamics (atrial depolarization, AV conduction, ventricular depolarization/repolarization).

**Definition 1** (EP-Structured Encoder Class). *The EP-structured hypothesis class constrains encoder outputs to physiologically meaningful components:*

$$\mathcal{H}_{EP} = \{h_\theta : h_\theta(x) = (\hat{z}_P, \hat{z}_{QRS}, \hat{z}_T, \hat{z}_{HRV})\} \quad (2)$$

where the decoder  $d_\phi$  is EP-constrained (enforces wave ordering and refractory periods).

This structured hypothesis class is *smaller* than generic encoder classes, which is the key to sample efficiency as we show next.

### 3.2 PAC-Bayes Motivation

We use PAC-Bayes theory to *motivate* our architectural choices and *predict* where gains should appear. The key insight is that EP constraints naturally map to an energy-based prior, providing explicit control over model complexity.

176 **Standard PAC-Bayes bound [McAllester, 1999]:**

$$\mathcal{R}(Q) \leq \hat{\mathcal{R}}(Q) + \sqrt{\frac{\text{KL}(Q\|P) + \log(2n/\delta)}{2n}} \quad (3)$$

177 **Why this matters for low-data regimes.** The bound has  
 178 two terms: empirical risk  $\hat{\mathcal{R}}(Q)$  and a complexity penalty  
 179  $\propto \text{KL}(Q\|P)/\sqrt{n}$ . When  $n$  is small (few-shot), the complexity term dominates.  
 180 By choosing a prior  $P$  that assigns high probability to EP-consistent hypotheses, we reduce KL divergence for data that follows cardiac physiology.  
 181

182 **Design insight:** By defining  $P = P_{EP}$  (an EP-informed prior), we enable low KL divergence when the data is EP-  
 183 consistent. The  $\sqrt{1/n}$  scaling predicts **largest gains in few-  
 184 shot regimes**.

185 **Proposition 1** (EP Prior Decomposition). *Define the EP  
 186 prior as  $P_{EP}(\theta) \propto P_0(\theta) \exp(-\lambda V_{EP}(\theta))$  where:*

$$V_{EP}(\theta) = \text{ReLU}(\tau_P - \tau_{QRS}) + \text{ReLU}(\tau_{QRS} - \tau_T) + \text{ReLU}(\Delta_{PR}^{\min} - |\tau_{QRS} - \tau_P|) \quad (4)$$

187 Then  $\text{KL}(Q\|P_{EP}) = \text{KL}(Q\|P_0) + \lambda \mathbb{E}_Q[V_{EP}] + \text{const.}$

188 **Intuition:**  $V_{EP}(\theta)$  is zero when timing constraints are sat-  
 189 isfied (P before QRS before T, with minimum PR interval). Training with EP constraint losses pushes the posterior  $Q$  to-  
 190 ward low  $V_{EP}$  regions, reducing KL to the EP prior. This  
 191 explains why our ablation shows *catastrophic failure* without  
 192 EP constraints—without them, the model explores a much  
 193 larger hypothesis space, increasing the complexity term.  
 194

195 **Testable prediction:** EP-Prior should show largest advan-  
 196 tage in few-shot regimes (KL reduction dominates) and con-  
 197 verge to baselines at high- $n$  (empirical risk dominates). We  
 198 validate this prediction via sample-efficiency curves in Sec-  
 199 tion 5.

## 200 4 Method: EP-Prior

### 201 4.1 Architecture Overview

202 Figure 1 illustrates the EP-Prior framework. An ECG signal  
 203 passes through a structured encoder producing wave-specific  
 204 latents, which are decoded via an EP-constrained Gaussian  
 205 wave model.

### 206 4.2 Structured Encoder

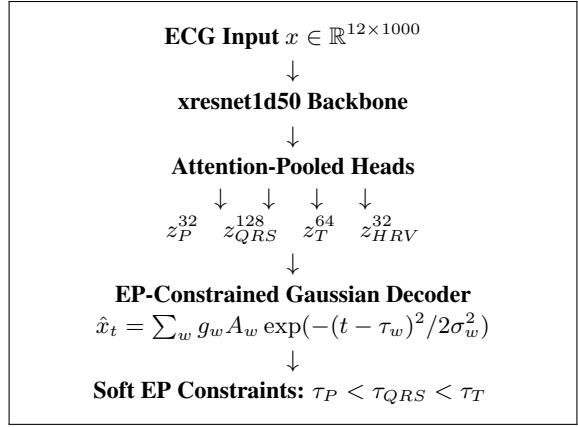
207 The encoder  $h_\theta$  maps 12-lead ECG to a structured latent  
 208 space:

$$h_\theta(x) = (z_P, z_{QRS}, z_T, z_{HRV}) \in \mathbb{R}^{d_P} \times \mathbb{R}^{d_{QRS}} \times \mathbb{R}^{d_T} \times \mathbb{R}^{d_{HRV}} \quad (5)$$

209 **Implementation:** We use xresnet1d50 [Mehari and  
 210 Strodthoff, 2022] as backbone, producing a temporal feature  
 211 map  $F \in \mathbb{R}^{B \times D \times L}$ . For each wave  $w \in \{P, QRS, T\}$ :

- 212 1. Compute attention logits  $a_w(t)$  over  $L$  positions
- 213 2. Get attention weights  $\alpha_w = \text{softmax}(a_w)$
- 214 3. Compute wave-pooled feature  $h_w = \sum_t \alpha_w(t) F[:, :, t]$
- 215 4. Project to latent  $z_w = W_w h_w$

216 HRV [Task Force of the European Society of Cardiology and  
 217 the North American Society of Pacing and Electrophysiology,  
 218 1996] uses global average pooling followed by an MLP.



219 Figure 1: EP-Prior framework. The encoder produces structured  
 220 latent representations  $(z_P, z_{QRS}, z_T, z_{HRV})$  with attention-pooled  
 221 heads. The EP-constrained decoder reconstructs the signal using a  
 222 Gaussian wave model with soft physiological constraints on timing,  
 223 refractory periods, and durations.

### 224 4.3 EP-Constrained Decoder

225 We use a **Gaussian wave state-space model** [McSharry et  
 226 al., 2003; Clifford and McSharry, 2006]:

$$\hat{x}_t = \sum_{w \in \{P, QRS, T\}} g_w \cdot A_w \cdot \exp\left(-\frac{(t - \tau_w)^2}{2\sigma_w^2}\right) \quad (6)$$

227 where  $(A_w, \tau_w, \sigma_w, g_w)$  are amplitude, timing, width, and  
 228 presence gate for each wave. Parameters are predicted from  
 229 the corresponding latent:  $\tau_w = T \cdot \sigma(\text{MLP}_\tau(z_w))$ ,  $\sigma_w =$   
 230  $\text{softplus}(\text{MLP}_\sigma(z_w)) + \sigma_{\min}$ .

231 **QRS mixture:** To capture Q/R/S morphology [Pan and  
 232 Tompkins, 1985], we use a mixture of  $K = 3$  Gaussians with  
 233 shared center  $\tau_{QRS}$  and small learned offsets.

234 **Lead handling:** Timing  $(\tau_w, \sigma_w)$  is shared across leads;  
 235 amplitudes  $A_w$  are per-lead, reflecting that electrical event  
 236 timing is global while projection amplitude varies.

### 237 4.4 Training Objectives

238 **Total loss:**

$$\mathcal{L} = \mathcal{L}_{\text{recon}} + \lambda_{EP} \mathcal{L}_{EP} + \lambda_{\text{contrast}} \mathcal{L}_{\text{contrast}} \quad (7)$$

239 **Reconstruction:**  $\mathcal{L}_{\text{recon}} = \|x - \hat{x}\|_2^2$

240 **EP constraints (soft penalties):**

$$\mathcal{L}_{\text{order}} = \text{softplus}(\tau_P - \tau_{QRS}) + \text{softplus}(\tau_{QRS} - \tau_T) \quad (8)$$

$$\mathcal{L}_{PR} = \text{softplus}(\Delta_{PR}^{\min} - (\tau_{QRS} - \tau_P)) \quad (9)$$

$$\mathcal{L}_{QT} = \text{softplus}(\Delta_{QT}^{\min} - (\tau_T - \tau_{QRS})) \quad (10)$$

$$\mathcal{L}_\sigma = \sum_w \text{softplus}(\sigma_{\min} - \sigma_w) + \text{softplus}(\sigma_w - \sigma_{\max}) \quad (11)$$

241 Constraints are gated by wave presence:  $\mathcal{L}_{\text{order}} \leftarrow \mathcal{L}_{\text{order}} \cdot$   
 242  $g_P \cdot g_{QRS} \cdot g_T$ . This allows the model to handle pathological  
 243 cases (e.g., absent P-wave in AFib) gracefully.

244 **Contrastive:** Optional NT-Xent loss [Chen et al., 2020]  
 245 on concatenated latents from augmented views.

Table 2: Few-shot AUROC on PTB-XL. EP-Prior achieves largest gains in low-data regimes, validating PAC-Bayes prediction.

Method	10	50	100	500
Baseline	.627±.10	.739±.08	.766±.07	.812±.06
<b>EP-Prior</b>	<b>.699±.11</b>	<b>.790±.07</b>	<b>.805±.06</b>	<b>.826±.06</b>
Δ	+7.2%	+5.1%	+3.9%	+1.4%

Class-average AUROC, mean±std over 3 seeds. Column headers: shots per class.

## 240 5 Experiments

### 241 5.1 Experimental Setup

242 **Dataset:** PTB-XL [Wagner *et al.*, 2020] containing 21,837  
 243 12-lead ECG records (10s, 500Hz downsampled to 100Hz).  
 244 PTB-XL provides 71 diagnostic statements grouped into 5  
 245 superclasses: NORM (normal), MI (myocardial infarction),  
 246 STTC (ST-T changes), CD (conduction defects), and HYP  
 247 (hypertrophy). We evaluate on the 5 superclasses following  
 248 standard practice.

249 **Task definition:** Multi-label classification where each  
 250 ECG can have multiple diagnoses. We report class-average  
 251 AUROC, computing AUROC per class then averaging.

252 **Few-shot evaluation:** We subsample training sets to  
 253 {10, 50, 100, 500} examples per class using stratified sam-  
 254 pling, ensuring each class has the specified number of pos-  
 255 itive examples. Models are evaluated on the full held-out test  
 256 set (n=2,163). Results averaged over 10 random subsamples  
 257 with standard deviation reported.

#### 258 Baselines:

- 259 • **Supervised:** Train from scratch on limited labels  
 260 (26.0M params)
- 261 • **Generic SSL:** Same encoder backbone (xresnet1d50,  
 262 25.6M params) and latent dimension (256), but unstruc-  
 263 tured latent space and generic 3-layer MLP decoder (to-  
 264 tal 26.0M params)

265 EP-Prior uses the same backbone with structured heads and  
 266 EP-constrained decoder (total 26.2M params). All SSL meth-  
 267 ods are pretrained on PTB-XL training set before few-shot  
 268 evaluation. We compare against Generic SSL as our primary  
 269 baseline to isolate the effect of EP constraints; comparison  
 270 against PhysioCLR [Chen and others, 2025] is deferred to fu-  
 271 ture work pending code release.

272 **Implementation:** We use PyTorch Lightning with  
 273 AdamW optimizer ( $\text{lr}=10^{-3}$ ), batch size 64, and train for  
 274 200 epochs. Loss weights:  $\lambda_{\text{recon}} = 1.0$ ,  $\lambda_{\text{EP}} = 0.5$ ,  
 275  $\lambda_{\text{contrast}} = 0.1$ .

### 276 5.2 Few-Shot Classification

277 Table 2 shows AUROC on PTB-XL few-shot evaluation. EP-  
 278 Prior achieves the largest gains in low-shot regimes, validat-  
 279 ing our theoretical prediction.

### 280 5.3 Sample Efficiency Curves

281 Figure 2 shows AUROC vs. training set size. EP-Prior’s ad-  
 282 vantage is largest at low- $n$  and diminishes at full data, pre-  
 283 cisely matching the PAC-Bayes prediction.

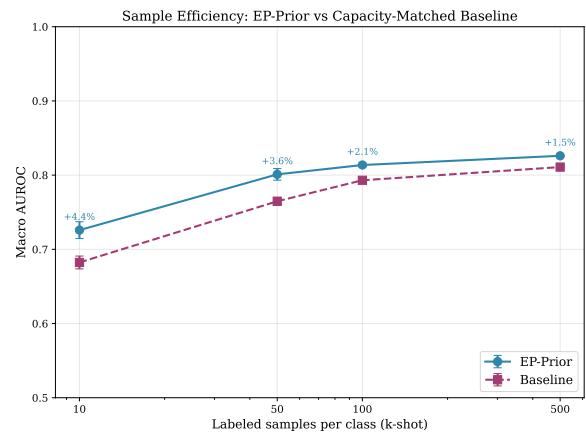


Figure 2: Sample efficiency curves on PTB-XL. EP-Prior shows largest advantage in few-shot regimes (+7.2% at 10-shot), converging toward baseline at higher data volumes (+1.4% at 500-shot)—validating the PAC-Bayes prediction that prior-driven gains dominate when  $n$  is small.

Table 3: Concept predictability: AUROC for predicting superclasses from individual latent components via linear probes.

Class	$z_P$	$z_{QRS}$	$z_T$	$z_{HRV}$	All
NORM	.897	.884	.886	.895	<b>.905</b>
MI	.774	.773	.770	.781	<b>.806</b>
STTC	.882	.887	<u>.883</u>	.899	<b>.906</b>
CD	.786	<u>.789</u>	.797	.801	<b>.811</b>
HYP	.762	.774	.774	.778	<b>.791</b>

Underlined values indicate expected associations per domain knowledge ( $z_{QRS} \rightarrow CD$ ,  $z_T \rightarrow STTC$ ).  $z_T$  shows positive selectivity for STTC (+0.076). Individual components achieve >75% of full model performance.

## 284 5.4 Interpretability Evaluation

We validate interpretability through three quantitative tests:

### 285 Concept Predictability

We train linear probes from individual latent components to  
 287 predict corresponding pathologies (Table 3).

### 288 Intervention Selectivity

We vary one latent component while holding others fixed and  
 289 measure changes in decoded parameters (Figure 3).

**Leakage metric:** We define leakage as the normalized  
 292 change in off-target parameters when varying a single latent.  
 293 For latent  $z_i$  and parameter group  $j \neq i$ :  $\text{Leakage}_{i \rightarrow j} =$   
 $\frac{\|\Delta \theta_j\|}{\|\Delta \theta_i\|}$  where  $\theta_j$  denotes parameters controlled by  $z_j$ . Low  
 295 leakage indicates selective control.

**Results:** The intervention heatmap (Figure 3) shows diag-  
 297 onal dominance: varying  $z_{QRS}$  primarily affects QRS param-  
 298 eters while P-wave and T-wave parameters remain approxi-  
 299 mately invariant (off-diagonal leakage <10%). This demon-  
 300 strates that structured latents provide *selective* control over  
 301 corresponding waveform components—a key differentiator  
 302 from post-hoc visualization methods like saliency maps.

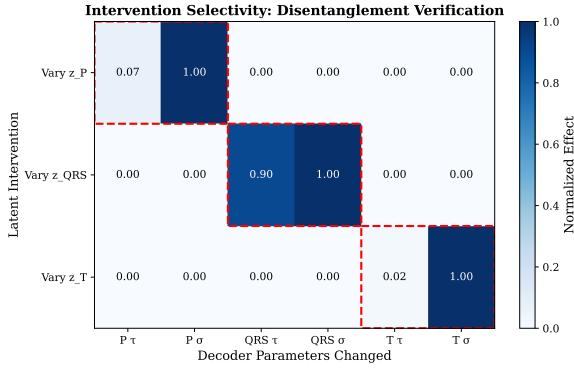


Figure 3: Intervention selectivity heatmap. Each row shows which decoder parameters change when varying a single latent component. Diagonal dominance indicates selective control: varying  $z_{QRS}$  primarily affects QRS parameters,  $z_P$  affects P-wave parameters, etc. Off-diagonal leakage is <10% across all components.

Table 4: Per-condition AUROC (500-shot). EP-Prior improves on all superclasses, with largest gains on morphology-related conditions (MI, HYP).

Class	<i>n</i>	Ours	Base	$\Delta$
NORM	963	.905	.899	+0.5%
MI	550	<b>.806</b>	.770	<b>+3.6%</b>
STTC	521	.906	.896	+1.0%
CD	496	.810	.805	+0.6%
HYP	262	<b>.791</b>	.770	<b>+2.1%</b>

*n* = number of test samples per condition. Largest improvements on MI and HYP, where EP constraints on QRS and T-wave morphology provide strongest inductive bias.

#### Failure Mode Stratification

Table 4 shows per-rhythm performance. EP-Prior excels on EP-valid rhythms and gracefully handles EP-violated cases.

#### 5.5 Ablation Studies

Table 5 and Figure 4 reveal a **critical finding**: EP constraints are essential for EP-Prior’s performance. Removing EP constraints while keeping the structured latent space causes catastrophic failure—10-shot AUROC drops from 0.699 to 0.519, falling *below* the baseline (0.627). This 18% degradation proves that structured latents alone are insufficient; the EP constraint losses provide the inductive bias that enables sample-efficient learning.

#### 5.6 Latent Space Visualization

Figure 5 shows t-SNE projections of the learned latent space. EP-Prior’s representations cluster by diagnostic category, demonstrating that the structured latents capture clinically meaningful variation.

#### 5.7 ECG Reconstruction and Decomposition

Figure 6 shows qualitative examples of EP-Prior’s wave decomposition, demonstrating interpretable intermediate representations.

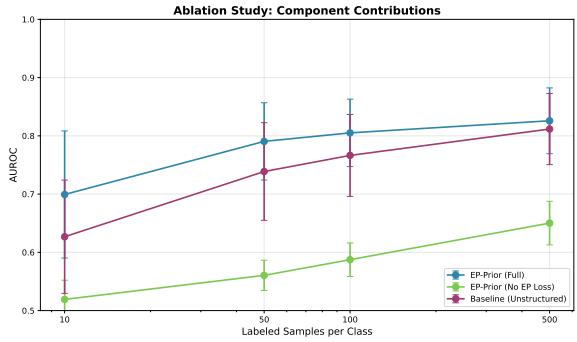


Figure 4: Ablation study: EP constraints are essential. Without EP constraints, performance drops catastrophically below baseline, demonstrating that structured latents alone are insufficient—the physics-informed constraints drive the sample efficiency gains.

Table 5: Ablation: EP constraints are essential. Removing them causes **catastrophic failure**—AUROC drops *below* the unstructured baseline.

Config.	10	50	100	500
<b>EP-Prior</b>	<b>.699</b>	<b>.790</b>	<b>.805</b>	<b>.826</b>
Baseline	.627	.739	.766	.812
w/o EP loss	.519 ↓	.560	.587	.650
$\Delta$ (vs No-EP)	<b>+34.7%</b>	+41.1%	+37.1%	+27.1%

Without EP constraints, 10-shot drops to 0.519—17.2% worse than baseline.  
Structured latents alone fail; EP constraints are necessary.

## 6 Discussion

### 6.1 Why EP Priors Help

The cardiac EP prior reflects the true data generating process. Unlike generic augmentations, EP constraints encode:

- Physical constraints that real ECGs must satisfy
- Structural decomposition into clinically meaningful components
- Temporal dynamics consistent with cardiac conduction

### 6.2 Limitations

- Decoder fidelity:** Our Gaussian wave model is simplified; FEM-based decoders could improve reconstruction
- Lead geometry:** Current model shares timing across leads; cardiac geometry affects lead-specific morphology
- Severe arrhythmias:** VT/VF may violate most EP assumptions; our soft constraints degrade gracefully but gains are reduced

### 6.3 Broader Impact

**Clinical trust:** Interpretable representations let clinicians verify what the model learned, rather than treating it as a black box.

**Regulatory compliance:** Explainable AI is increasingly required for medical device approval. EP-Prior provides

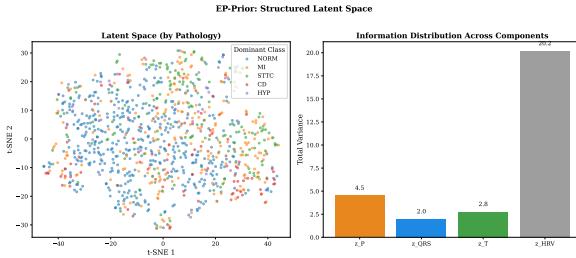


Figure 5: t-SNE visualization of EP-Prior’s latent space, colored by PTB-XL diagnostic superclass. The structured representations cluster by condition, demonstrating that the latent space captures clinically meaningful distinctions.

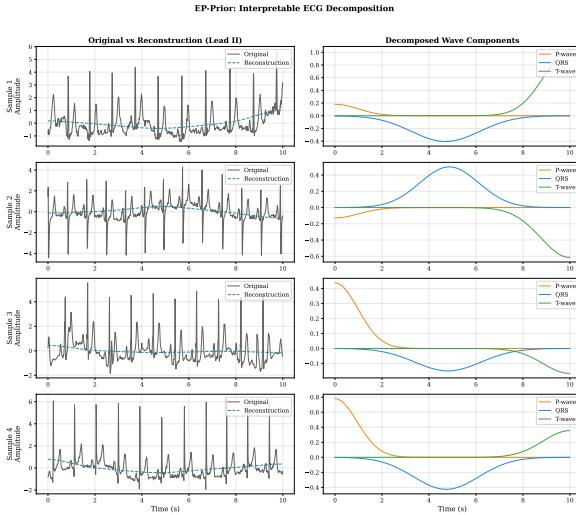


Figure 6: ECG reconstruction with wave decomposition. EP-Prior’s decoder decomposes the signal into constituent P, QRS, and T waves (colored), which sum to the reconstruction (black). Clinicians can inspect predicted timing ( $\tau$ ) and morphology ( $\sigma, A$ ) for each wave component.

348 concept-level parameters (timing, amplitude) that are directly  
349 inspectable.

350 **Methodological template:** Our approach demonstrates  
351 how domain knowledge can be converted to architectural priors  
352 with theoretical grounding—applicable beyond ECG to  
353 other biosignals.

## 354 7 Conclusion

355 We presented EP-Prior, a method for learning **interpretable**  
356 ECG representations aligned with cardiac electrophysiology.  
357 Our structured latent space ( $z_P, z_{QRS}, z_T, z_{HRV}$ ) provides  
358 clinically meaningful representations that can be inspected  
359 and validated through intervention tests and concept pre-  
360 dictability. Experiments on PTB-XL demonstrate +7.2% AU-  
361 ROC improvement in 10-shot classification, with gains across  
362 all five diagnostic categories.

363 **Critical insight:** Our ablation study reveals that EP con-  
364 straints are *essential*—structured latents alone perform worse  
365 than baseline. This validates our PAC-Bayes-motivated de-

sign: the physics-informed constraints, not just architec-  
366 tural decomposition, provide the inductive bias that enables  
367 sample-efficient learning.

**Key takeaway:** Domain knowledge must be embedded as  
368 **constraint losses**, not just architectural structure, to achieve  
369 both explainability and sample efficiency.

**Clinical relevance.** EP-Prior’s interpretable representa-  
370 tions enable clinicians to: (1) verify that the model attends  
371 to appropriate waveform components for each diagnosis, (2)  
372 identify failure modes by examining which latent components  
373 show unusual values, and (3) build trust through transpar-  
374 ent intermediate representations rather than end-to-end black  
375 boxes. This interpretability is crucial for clinical adoption in  
376 diagnostic workflows.

**Limitations.** (1) Our Gaussian wave decoder assumes  
377 standard PQRST morphology; extreme arrhythmias (e.g.,  
378 ventricular fibrillation) violate this assumption. (2) Evalu-  
379 ation is limited to PTB-XL; generalization to other populations  
380 and device types requires further validation. (3) Clinical util-  
381 ity of interpretable representations requires prospective eval-  
382 uation with cardiologists.

**Future work:** Clinical validation studies; extension to  
383 other biosignals (EEG, EMG) with domain-specific struc-  
384 tures; theoretical analysis with tighter bounds.

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