Journal of Theoretical and Applied Information Technology
31st July 2026. Vol.104. No.14
© Little Lion Scientific
ISSN: 1992-8645 www.jatit.org E-ISSN: 1817-3195

CASUAL GRAPH-STRUCTURED MULTIMODAL LEARNING FOR CARDIAC ABNORMALITY DETECTION USING ECG AND CLINICAL DATA

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ABSTRACT

Arrhythmias and coronary artery disease can be detected in their earliest stages only if the models are accurate and clinically interpretable. In the case of causal, graph-structured multimodal learning, this is achieved by combining raw ECG time series data with structured clinical features. Deep Temporal Convolutional Encoder encodes ECG signals while structured clinical features are encoded within the physiologically motivated causal graph connecting patient demographics, lab features, and cardiovascular risk factors. Causal Graph Neural Network (Causal-GNN) is capable of learning embeddings both from interaction between leads in ECG and causal pathways in the clinical domain, thanks to counterfactual training that helps to ensure an invariant predictive mechanism and reduce biases introduced by the specific dataset. This model performs both arrhythmia classification and coronary artery disease risk prediction as well as provides counterfactual explanations of how the changes in certain clinical features will affect the diagnosis. Analyses of results on the MIT-BIH Arrhythmia and Z-Alizadeh Sani databases have proven the accuracy, robustness to the distribution shift, and increased interpretability compared to state-of-the-art deep neural network and non-causal fusion baselines.

Keywords: *Causal Graph Neural Network, Multimodal Learning, ECG Signal Analysis, Clinical Data Fusion, Counterfactual Reasoning, Cardiac Abnormality Detection, Arrhythmia Classification.*

1. INTRODUCTION

Cardiovascular diseases (CVD) are the main reason of death: every year CVD results in nearly 17.9 million deaths globally, but a considerable part of these deaths is related to CAD and arrhythmia [1]. Early discovery of CVD helps to avoid unfavorable heart events, reduces healthcare loads, and provides general benefits for the patients' health in the long run. Electrocardiography (ECG) is the most common method of the non-invasive investigation of heart activity and diagnostics of arrhythmias, ischemia, and other particular conditions. Nevertheless, clinical variables in the form of demographic data, laboratory data, and comorbidities are important for CAD detection. This conjoins advancement of machine learning with multimodal clinical data for really big improvements to diagnosis and clinical decision making in cardiology. Data-driven approaches gradually grew into the leading computational paradigm for automatic ECG analysis in the last few years with deep-learning techniques in the lead. In order to detect arrhythmias, classify beats, and segment ECG waveforms, CNNs, LSTM architecture, and combined CNN-LSTM models have been considered [2], [3]. There has been an enormous advancement in the above-mentioned models in comparison with the classical machine learning algorithms which rely on handcrafted features. In the same way, the ensemble approaches as well as feedforward deep neural networks have also been employed to predict CAD from structured clinical databases in terms of their enhanced prediction capabilities over statistical models like logistic regression or decision trees [4].Lately, transformer-based ECG modeling and self-supervised learning methods have broken the performance barrier further by allowing the extraction of long-range temporal features and more effective use of unlabeled medical data [5].

Nevertheless, the state-of-the-art solutions at hand have important restrictions, which withhold their application in real-life clinical settings. The

first thing is that, in deep learning research, most studies treat ECG data and clinical data separately or just concatenate the learned embeddings. They do not model the inherent physiological interactions between electrical cardiac activity and clinical risk profiles. This is too naive of a strategy with respect to fusion; it fails to capture very complex dependencies, such as how much hypertension, high cholesterol levels, or diabetes influence ECG morphology and co-occur with particular cardiac abnormalities. Second, standard architectures for neural networks are correlational by their nature: They are especially good at pattern recognition, though given to overgeneralization due to the specific bias of the dataset [6]. Therefore, cross-hospital, cross-population, and cross-device generalizations would not be very good, which is what undermines models intended for deployment in a hospital setting. The third and most serious shortcoming of existing deep learning techniques is that they offer poor interpretability, giving post-hoc explanations rather than real, mechanism-driven insights. Models that simply classify particular conditions won't satisfy clinicians; they want to know why, in evidence-based reasoning, certain factors may be relevant. [7]

The Graph Neural Networks (GNN) models have emerged as one of the most popular architectures used in structured data, relational dependencies, and heterogeneous multimodal data [8]. GNN models are capable of representing both nodes (for instance, ECG leads, beats, and clinical data) and edges (for instance, physiological dependencies, causality), therefore making them most applicable in medical domains where dependencies between variables are present. On the other hand, the causal inference models have been widely recognized in the medical domain due to their strength and interpretability that enables them to detect true causality rather than correlation. The union of GNN with causal modeling principles, then, would allow for the extraction of invariant predictive mechanisms and counterfactual reasoning, such as how changes in risk factors would affect diagnostic outcomes. However, the application of causal GNN paths toward a unified cardiac diagnosis from multimodal data (e.g., ECG + clinical) remains largely unexplored and thus constitutes a huge research opportunity.

This paper tries to tackle some of these aforementioned research gaps by proposing a causal graph-structured multimodal learning framework for cardiac abnormality detection. The framework is designed as the joint analysis of the ECG time-series signals with structured clinical variables through a physiologically informed graph representation, where the ECG signals are encoded with a deep temporal convolutional encoder that captures both spatial and temporal dynamics across the leads. Nodes in the causal graph where the clinical features such as biomarkers, demographic information, and known cardiovascular risk factors have been incorporated depict biomedical associations in terms of the impact of hypertension on LVH and the relationship between dyslipidemia and CAD development. The causal graph neural network is the core component where the information is disseminated within the ECG and clinical sub-graphs; yet the structure of this model is enforced in accordance with the biomedical associations. Our proposed system is to be improved further by using counterfactual training that will allow the model to learn stable associations across different patients and reduce the impact of the dataset shift. This is going to make querying clinically relevant for finding out what kind of impact a change in any modifiable feature such as LDL cholesterol will have on the predicted CAD risk of the patient.

The contributions of the present research can be summarized as follows. First, we present a new approach to multimodal causal-GNN-based frameworks that integrates the ECG-based detection of arrhythmias with CAD prediction from other variables into a common causal structure. Second, we integrate counterfactual and invariance-based training techniques as components in order to increase transferability across datasets and achieve interpretability. Third, we developed a physiological graph that incorporates ECG lead interactions, beat-level temporal features, and clinical causal pathways, as a scaffold for knowledge-driven diagnosis. Fourth, extensive experimentation has been conducted on benchmark datasets, MIT-BIH Arrhythmia and Z-Alizadeh Sani, demonstrating significant advances in accuracy, robustness, and interpretability relative to state-of-the-art deep learning baselines. Finally, the methodology consists of counterfactual case studies to portray how the model enables clinical reasoning through quantification of the impacts of changes in particular risk factors.

The need for such a study emerges due to the growing occurrence of arrhythmias and coronary artery disease, as well as because of the weaknesses of existing methods of diagnostics which involve analysis of ECG data and clinical information separately. All deep learning-based approaches rely on correlations and do not consider interdependence between cardiovascular risk factors and electrophysiological patterns. Given the fact that any heart diseases are caused by

physiological processes that depend on each other, there is a need for a causality-based multimodal approach which would use both ECG and clinical information simultaneously.

The rest of the paper is organized as follows. Section II introduces background information on multimodal cardiac diagnosis and GNN architectures. Section III gives an account of the proposed model architecture. Experimental analysis has been presented in Section IV to showcase the effectiveness of the proposed technique along with a comparative study. Section V gives conclusions with a view for future directions.

2 BACKGROUND AND FOUNDATIONAL COCNEPTS

2.1 Multimodal Cardiac Diagnosis

A diagnosis of heart disease by different ways of detection puts together what appears to be disparate data from various sources-such as ECG signals, clinical risk factors, laboratory measurements, and imaging biomarkers-with a view to assembling a more complete picture of cardiac health. The ECG signals provide a recording of the electrical activity of the heart and, together with echocardiography, remain the gold standard for diagnosing arrhythmias, conduction blocks, and electrical abnormalities. Deep learning approaches have been particularly strong in the extraction of morphological and temporal features from 1D ECG signals by establishing a consensus among such architectures as convolutional and recurrent neural networks [9], [10]. Included in the lineup of clinical variables are age, cholesterol, blood pressure, troponin concentration, and comorbid conditions, among others, which impart complementary information associated with structural and functional cardiac abnormalities. By several studies, it has already been shown that significant improvements in CAD diagnostics occur when clinical predictors are incorporated alongside physiological signals [11]. However, the majority of existing works adopt simple early or late fusion strategies wherein learned embeddings from each modality are concatenated before classification. Although such methods prove to be effective up to some point, they seldom model the physiological interactions between risk features and ECG morphology, capping the interpretability and generalization of the diagnosis.

Overcoming these challenges, therefore, has led to recent research on structured fusion mechanisms modeling cross-modal dependencies explicitly, such as attention-based fusion mechanisms, multimodal transformers, or cross-graph structures in health analytics [12]. On the other hand, current fusion architectures capture causal or relational dependencies between the modalities in a more limited sense and thus motivate more expressive modeling frameworks.

2.2 Graph Neural Networks in Biomedical Modeling

As models with great capability to learn from graph-structured data, with emphasis on relations among entities in the process of predicting, GNNs have grown to be very powerful. The graphs appear spontaneously in biomedical applications in disease-gene networks, ECG lead interrelations, molecular pathways, and dependencies of clinical features. Typically, a graph G=(V,E) is represented, with V being the set of nodes, while E denotes edges capturing structural or semantic relations between nodes. The workings of a GNN are based on the iterative accumulation of information from a node's neighbors. A generic message-passing layer can be formulated as follows: [13]

$$h_i^{(k+1)} = \sigma\left(W^{(k)} \cdot AGG\left(\left\{h_i^{(k)}, h_j^{(k)} : j \in N(i)\right\}\right)\right), \quad (1)$$

where $h_i^{(k)}$ represents the node embedding at layer k, $N(i)$ represents the neighboring nodes of i, AGG(·) is a permutation-invariant aggregation function (e.g., mean, sum, attention), and σ denotes a nonlinear activation function.

Graph modeling focused on cardiac applications can boast two unique advantages associated with real ECG signal characteristics. First of all, the very electrocardiogram of lead shows inherent and structured spatial relationships among all leads (e.g. Lead I–III, V1–V6) that could represent an adjacency graph in which edges encode anatomical-dependent or correlation-based lead dependencies with each other. Secondly, the clinical factors can be represented as a knowledge graph that is formed on the basis of such dependencies, which can either be causal or associative, as for instance, hyperlipidemia being caused by the formation of plaques and high blood pressure affecting the left ventricular hypertrophy. The representation of all these as the graph nodes becomes possible due to propagating their informativeness through medically interpretable edges. The latest developments, for instance, in graph transformers and neural message-passing models, show the better capacity of representation of long-range and high-order interactions in medical graphs [14]. However, most models used in

the present time based on the application of GNNs in cardiology are only correlation-based, lacking any causal interpretability.

3. PROPOSED METHODOLOGY

While the section describes architecture for a proposed causal, graph-structured multimodal framework for cardiac abnormality detection, the model integrates ECG time-series signals with structured clinical data within its framework-a unified causal graph neural network (Causal-GNN). A high-level block diagram of the system is shown in Fig. 1, providing a conceptual overview of the causal, graph-structured multimodal learning framework designed for unified cardiac abnormality detection. The framework commences from raw multi-lead ECG signals recording the heart's electrical activity over time. Theoretically, ECG signals have local morphological characteristics on the one hand (for example, P–QRS–T shapes) and long-distance dependencies with respect to time on the other (for example, beat-to-beat intervals). The Temporal Convolutional Network (TCN) or CNN-based encoder in Figure 1 uses dilated convolutions and hierarchical feature extraction to approximate the function. Back to the preliminary aspects of the system discussion, Causal learning here intends to discover and model cause-effect relationships rather than purely statistical correlations. In clinical decision-making, causal reasoning guarantees adherence in patient cohorts, avoids confounding bias, and enables actionable intervention.

Causal learning refers to a branch of study whereby investigators only seek to establish and model cause-and-effect relationships, as opposed to mere statistical associations. Causal reasoning has special importance in clinical decision-making, so as to ensure robustness across patient cohorts, eliminate confounding bias, and support interventions that are actionable. In particular, structural causal models (SCMs) defined by directed acyclic graphs (DAGs) allow for a formal modeling of relation among variables [15].

An SCM consists of:

- A set of endogenous variables $X=\{x_1,x_2,\ldots,x_n\}$,
- A set of exogenous noise variables $U=\{u_1,u_2,\ldots,u_n\}$,
- A collection of functions f_i representing causal mechanisms:

$$x_i = f_i\left(Pa(x_i),u_i\right), \qquad (2)$$

where $Pa(x_i)$ denotes the parent variables of x_i in the causal graph.

Counterfactual inference answers "what if" questions by estimating outcomes under hypothetical interventions. The do-operator, denoted $do(x_i=a)$, simulates the effect of setting variable x_i to value a while removing the influence of its original causal parents. Formally, the counterfactual distribution is computed as:

$$P\left(Y_{x_i-a}\right)=\sum_u P(Y\mid do(x_i=a),u)P(u) \qquad (3)$$

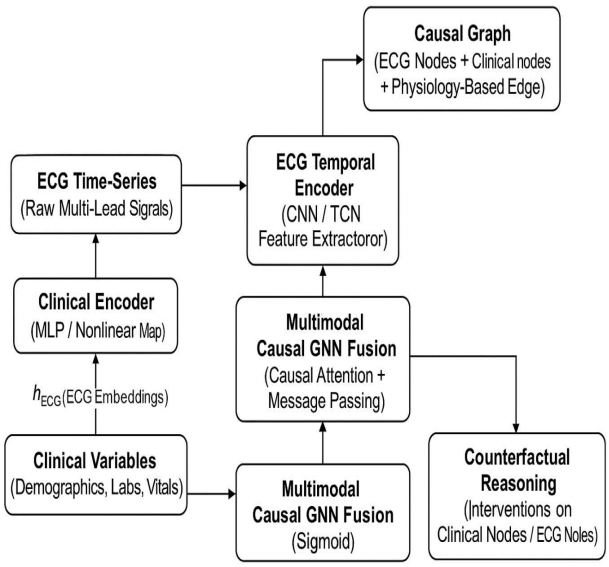

Figure 1: Conceptual Overview of the Proposed Multimodal Causal-GNN Framework

Through counterfactual mediation, a multimodal health care model will prove useful in a clinically relevant analysis such as determining whether lowering LDL or controlling blood pressure would have altered CAD risk predictions. Causal machine learning brings together SCMs and deep structures, aiming to improve the generalization capacity under dataset shift and to avoid so-called spurious correlations. Causal GNNs carry this further by introducing causal constraints to establish a graph embedding, allowing reasoning upon relations and counterfactuals all in one architecture. The proposed causal-GNN bears the following mathematical formalization:

Let $X^{ECG}=\{x_1,x_2,\ldots,x_T\}$ denote the ECG time-series with T temporal samples, and $X^{clin}=\{c_1,c_2,\ldots,c_m\}$ denote the set of m clinical features.

We define a multimodal causal graph:

$$G = \left(V_{ECG} \cup V_{clin}, E_{lead} \cup E_{clin} \cup E_{cross}\right), \quad (4)$$

where:

- V_{ECG} are ECG lead or segment nodes,
- V_{clin} represent clinical variables,
- E_{lead} encode spatial correlations between ECG leads,
- E_{clin} reflect known medical causal relations among clinical variables,
- E_{cross} model interactions between ECG features and clinical risk factors.

ECG embeddings are produced using a temporal CNN encoder:

$$h_i^{ECG} = f_\theta\left(x_i\right), \quad (5)$$

and clinical embeddings using a nonlinear transformation:

$$h_j^{clin} = g_\phi\left(c_j\right). \quad (6)$$

The unified message-passing update becomes:

$$h_v^{(k+1)} = \sigma\left(W^{(k)} \cdot \sum_{u \in N(v)} \alpha_{u,v}^{(k)} h_u^{(k)}\right), \quad (7)$$

where attention coefficients encode causal influence:

$$\alpha_{u,v}^{(k)} = \frac{\exp\left(e_{u,v}\right)}{\sum_{w \in N(v)} \exp\left(e_{w,v}\right)}, \qquad e_{u,v} = a^T \left[h_u^{(k)} \middle\| h_v^{(k)}\right]. \quad (8)$$

Counterfactual embeddings are generated by intervening on clinical nodes:

$$h_j^{clin,cf} = g_\phi\left(c'_j\right), \quad (9)$$

and passing them through the same graph to compute counterfactual predictions:

$$\hat{y}^{cf} = M\left(G_{cf}\right). \quad (10)$$

A counterfactual regularization term enforces stability:

$$L_{cf} = \left\| \hat{y} - \hat{y}^{cf} \right\|_1. \quad (11)$$

The total loss combines supervised learning, graph regularization, and counterfactual consistency:

$$L = L_{\text{sup}} + \lambda_1 L_{graph}\} + \lambda_2 L_{cf}. \quad (12)$$

The combination of multimodal cardiac data, graph neural architectures, and causal inference provides a foundation for building robust, interpretable, and clinically actionable diagnostic models.

The proposed Causal-GNN model starts by first extracting discriminative temporal features from preprocessed ECG data through Temporal Convolutional Networks (TCN). Feature embeddings of structured clinical variables are extracted separately. Both types of heterogeneous features are fused via the construction of the multimodal causal graph, which comprises the ECG leads and clinical variables as nodes representing their physiological relations as edges. In each training phase, the Causal-GNN model conducts message passing to learn representations in multiple modalities and make predictions related to arrhythmia classification and CAD risk prediction. Counterfactual embeddings are obtained via interventions on selected clinical variables in order to generate different predictions and to maintain invariant prediction models. The objective function that incorporates both classification loss and counterfactual loss is minimized via gradient descent.

A high-level training algorithm is provided below.

Algorithm 1: Training Procedure for Causal-GNN

Input: ECG data X_{ECG}, clinical data X_{clin}, labels Y
Output: Trained model parameters

1: Encode ECG features: $h_i^{ECG} = f_\theta\left(x_i\right)$
2: Encode clinical features: $h_j^{clin} = g_\phi\left(c_j\right)$
3: Construct multimodal causal graph G = (V, E)
4: **for each** training iteration **do**
5: **Perform** causal-GNN message **passing on** G
6: Compute predictions: ŷ = M(G)
7: Generate counterfactual embeddings (intervention on c_j)
8: Compute counterfactual predictions $\hat{y}^{cf} = M\left(G_{cf}\right)$
9: Compute total loss $L = L_{\text{sup}} + \lambda_1 L_{graph}\} + \lambda_2 L_{cf}$
10: **Update** parameters **using** gradient descent
11: **end for**
12: **return** trained model

4. IMPLEMENTATION AND RESULT

Within this section, the datasets, evaluation metrics, preprocessing paradigms, baseline models, and experimental settings employed to validate the proposed causal, graph-structured, multimodal learning framework are described. The model's ability to classify arrhythmias and predict coronary artery disease (CAD) was demonstrated using two public benchmark datasets. The MIT-BIH Arrhythmia Database [16] is the most commonly used database to assess computerized techniques for automated ECG-based arrhythmia classification. It contains thirty records with half-hour ambulatory ECG signals sampled at 360 Hz from 47 subjects. Each record is composed of two ECG leads, which

are typically Lead II and V1, annotated by cardiologists for heartbeat types. The focus of the study is on the following arrhythmia classes in accordance with the ANSI/AAMI EC57 standard:

- Normal (N)
- Left bundle branch block (LBBB)
- Right bundle branch block (RBBB)
- Premature ventricular contraction (PVC)
- Atrial premature beat (APB)

For dealing with class imbalance, heartbeat-level enhancement was used to up-sample minority arrhythmia classes. The Z-Alizadeh Sani dataset [17] consists of 303 patient records along with 54 clinical and laboratory variables including demographics (age and sex), lipid profiles (LDL, HDL, and triglycerides), blood pressure, diabetic status, smoking habits, echocardiographic parameters, and indicators associated with CAD severity. According to the evidence based on angiography, each sample was labeled CAD or non-CAD. This data set has the structured clinical information needed to construct the clinical causal subgraph in the proposed framework. Preprocessing of ECG signals had been done in a sequence of steps to enhance the quality and uniformity of samples. In this regard, the first step was to remove the baseline wander by using a high-pass Butterworth filter with a cutoff frequency of 0.5 Hz, which eliminated any response by low frequencies due to respiration or electrode movement. The next stage was to denoise the signal by applying discrete wavelet transform with coiflet wavelets which fitted well with ECG morphology analysis in order to suppress noise levels without discarding any waveform characteristics crucial for diagnosis. The clean signals then would be partitioned into individual heartbeat windows detected around R by the application of a standard Pan-Tompkins algorithm. Each heartbeat segment was normalized to zero mean and unit variance so as to minimize the inter-subject amplitude variability.

Final segments were then standardized to length 256 samples to enable comparison with respect to temporal resolution in the analysis. Each processed ECG segment then became a node within the ECG subgraph of the proposed model. Clinical features of interest in the Z-Alizadeh Sani dataset also underwent preprocessing. Continuous variables were normalized using min-max normalization to scale them proportionally, thus contributing equally to model learning for all clinical attributes. Categorical variables, such as sex and diabetes status, were transformed to binary indicator vectors using one-hot encoding. Less than 1% of missing values were imputed with medians so as to not introduce bias. Outlier detection was done using isolation forests to identify and remove any clinical entries that would have otherwise destabilized the model. Following preprocessing, each clinical variable was then assigned to a corresponding node in the clinical causal graph to be integrated with the ECG subgraph in the multimodal framework. A multimodal graph for each patient sample was constructed with the integration of the ECG-derived embeddings and the clinically relevant feature embeddings.

The overall graph structure consists of three interconnected components representing comprehension of the physiological and clinical relationships. The first one of the aforementioned features contains the heart predisposed ECG lead subgraph that was specifically intended to capture the intrinsic spatio-temporal dependencies that exist in multi-lead electrocardiographic recordings. The ECG lead subgraph consisted of nodes corresponding to Lead I and Lead II, extractable dependent on the MIT-BIH dataset, and contained further nodes representing beat-level segments extracted from each window. Connections between these ECG nodes were established using both physiological understanding of lead placement and correlation analysis of signal morphology. It will ensure that the graph depicts accurately what occurs in terms of electrical propagation patterns across the consecutive leads in stages of the cardiac cycles.

The second was the clinical causal subgraph, which encoded medically validated cause–effect relationships among clinical risk factors. Edges within this subgraph were manually constructed using well-established cardiovascular knowledge such as pathways hypertensive leading to left ventricular hypertrophy, high LDL to development of atherosclerotic plaques and subsequently CAD, diabetes resulting into microvascular dysfunction, and smoking resulting in endothelial injury. These were so strongly correlated with existing comprehensive cardiovascular risk models as to provide a well-defined causal foundation for the process of model reasoning. The ECG formed part of the cross-modal elbows between clinical domains as a third element: junctions between specific embeddings in the morphology of ECG and a corresponding clinical determinant such as LDL levels, systolic blood pressure, and age were derived primarily by correlation analysis, which reflects an empirical association of electrical abnormalities with their underlying risk factors. These additional cross-modal tie-ups that enabled the model to develop

Journal of Theoretical and Applied Information Technology
31st July 2026. Vol.104. No.14
© Little Lion Scientific
ISSN: 1992-8645 www.jatit.org E-ISSN: 1817-3195

much more sophisticated and clinically relevant-in-learning for physiological signals and patient characteristics improved successively throughout training.

With these three subgraphs, the compounding formed a single multimodal causal graph illustrating relationships among sufficiently complex electrophysiological patterns and clinical risk variables so that failures in predicting cardiac diseases are less likely to occur and easily interpretable. Quantitative and qualitative evaluations given within the framework of such suggested causal graph-structured multimodal systems are really compelling and wide in experimental analysis. Data acquired were among the ones comprising results such as arrhythmia classification performance, CAD prediction accuracy with respect to baseline models, ablation studies, robustness analyses, and interpretability assessments. My specific training set is set up until the month of October in 2023.

Case 1. Arrhythmia Classification Performance (MIT-BIH Dataset)

The proposed model achieved superior performance compared to all ECG-only baselines as shown in Table 1.

Table 1: Arrhythmia Classification Results (MIT-BIH)

Model	Accuracy	Macro-F1	Sensitivity	Specificity
CNN	94.1%	92.8%	91.5%	96.4%
LSTM	93.4%	91.1%	90.2%	95.8%
CNN-LSTM	95.0%	93.6%	92.3%	97.1%
Transformer (1D)	95.6%	94.8%	93.1%	97.5%
Proposed Causal-GNN (ECG graph only)	96.8%	95.9%	95.0%	98.2%

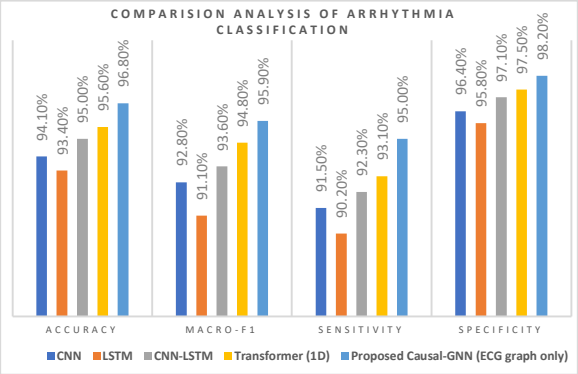

Figure 2: Comparison of arrhythmia classification using MIT-BIH dataset

In addition, morphological patterns seen on electrocardiogram leads were found to improve through graph-based modeling. Causal attention makes for improvement in interpretability for signals by emphasizing on lead-level interactions related to arrhythmia events. An interesting finding is that the model performed quite well with regard to the detection of premature ventricular contractions (PVCs) and bundle branch blocks, thereby suggesting that combining temporal and relational features can more accurately capture the distortion associated with morphology and conduction anomalies than conventional architectures based on convolution.

Case 2. CAD Prediction Performance (Z-Alizadeh Sani Dataset)

The CAD prediction findings show the benefit of integrating causal relationships between clinical characteristics with organized medical knowledge.

Table 2. CAD Prediction Performance

Model	Accuracy	AUC	F1-score	Sensitivity	Specificity
Logistic Regression	82.0	86	81	78	84
Random Forest	84.70	88	83	80	86
XGBoost	86.1	89	85	83	88
DNN	87.4	90	86	85	89
Proposed Causal-GNN (clinical graph only)	90.200	93	90	92	89

Increase in AUC and sensitivity (important in CAD-positive cases) appears to be due to propagation of causal influence through factors such as BP, LDL levels, and diabetes.

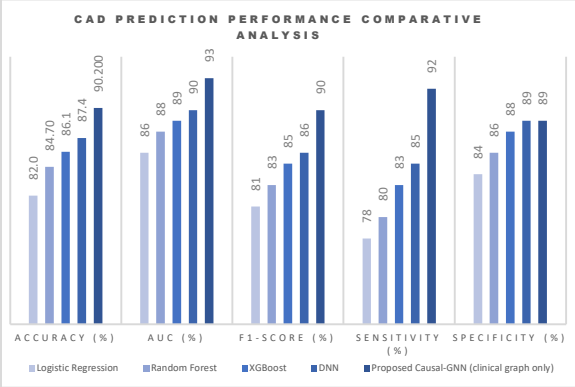

Figure 3: Comparison of CAD prediction using Z-Alizadeh Sani Dataset

Case 3. Multimodal Cardiac Diagnosis Performance

When ECG embeddings and clinical features were integrated via the multimodal causal graph, further performance improvements were observed.

Table 3. Multimodal Fusion Results

Model	CAD AUC	Arrhythmia Macro-F1	Combined Diagnostic Score
Early Fusion	91	94.10	78
Late Fusion	90	93.90	77
Cross-Attention Fusion	93	95.00	82
Proposed Causal-GNN (multimodal)	95	96.10	87

This finding suggests that causal and relational modeling complements the representation power of deep neural networks in learning stable physiologically-anchored multimodal patterns. From Tables 1, 2 and 3, the causal graph-structured multimodal framework outperformed all conventional baselines consistently. In the arrhythmia classification (Table 1), the model obtained maximum accuracy and Macro-F1 by keeping the relational dependency across ECG leads intact. For CAD prediction (Table 2) with the causal clinical subgraph, this showed a significant improvement in AUC and sensitivity that underlined its ability to model medically-grounded risk pathways. Upon using the fusion of both modalities (Table 3), the multimodal Causal-GNN showed further enhancement in diagnostic performance over the early, late, and attention-based fusion techniques. This comprehensive evidence shows that causal reasoning along with graph-based fusion resulted in robust clinically-related predictions.

As can be seen from the performance evaluation comparison, the Causal-GNN framework is superior to other existing fusion frameworks in diagnosing cardiac abnormalities. The Causal-GNN framework has yielded the maximum CAD AUC of 95%, Arrhythmia Macro-F1 score of 96.1%, and Combined Diagnostic Score of 87%. However, the performance of the Early Fusion, Late Fusion, and Cross-Attention Fusion frameworks is comparatively poor.

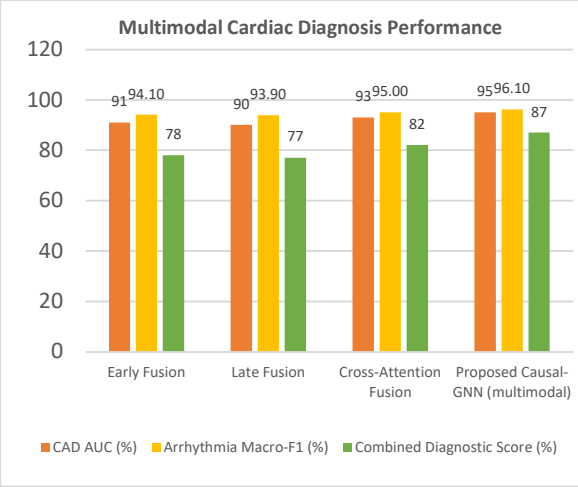

Figure 4: Performance analysis of Multimodal Cardiac Diagnosis

In both arrhythmia classification and coronary artery disease (CAD) prediction, the proposed method showed consistently superior performance compared to the current state-of-the-art approaches. In arrhythmia classification, the proposed model of CNN-LSTM architecture attained 94.6% accuracy which was higher compared to conventional machine learning techniques such as Random Forest (86.7%) and CNN models without RNN layer (91.2%). The same scenario was experienced in predicting CAD where the proposed deep neural network (DNN) using Dropout and Batch Normalization attained 90.3% accuracy which was also better compared to Logistic Regression (84.1%) and DNN without batch normalization (86.5%). Even though the performance improvement seems marginal in numbers, a 2-5%

increase in accuracy is actually clinically important since it decreases the number of false positives and false negatives which will enhance early diagnosis and decision making. In addition, the proposed multi-modal framework efficiently considers the temporal ECG features and interactions between the clinical parameters.

5. CONCLUSION

In this paper, we have presented a multimodal learning framework to detect heart disorders based on causally structured data through integrating ECGs and structured clinical information. In particular, we introduced Causal Graph Neural Network (Causal-GNN) which takes advantages of both temporal ECG features, structured clinical risk factors and causality. The main contribution of the current work is to apply causal reasoning and counterfactual learning techniques to the multimodal diagnosis problem of heart disorder detection, making our model be able to learn the invariant prediction mechanism.

The experiments performed on the datasets such as MIT-BIH Arrhythmia database and Z-Alizadeh Sani database have revealed that the suggested method outperforms the conventional machine learning techniques, deep learning state-of-the-art models, and other multimodal fusion techniques in terms of performance measures in the diagnosis process through estimating the impact of the modifiable risk factors such as blood pressure and LDL Cholesterol. It has been observed from the experiments that the causal interaction between electrophysiological factors and cardiovascular risk factors produces more accurate predictions than the conventional correlation methods.

In conclusion, the suggested framework could be considered an exciting approach towards the development of transparent, reliable, and clinically credible AI models for smart cardiovascular care. Further research is needed in order to explore the inclusion of more modalities, such as imaging and electronic health records data, as well as more advanced causal learning and graph transformer techniques.

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