International Journal Of
Industrial Electronics Control and Optimization
Print ISSN: 3517-2645
Online ISSN: 3568-2645
Homepage: https://ieco.usb.ac.ir/

Multi-Rate Hybrid Control Framework for Coronary Heart Disease Risk Management

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Article Info

Article type:
Research Article

Article history:
Received: *************
Received in revised form: *************
Accepted: *************
Published online: ************

Keywords:
Decision-Making,
Risk Assessment,
Coronary Heart Disease,
Multi-Rate Controller.

ABSTRACT

This paper proposes a multi-rate hybrid control framework for modeling and managing coronary heart disease (CHD) risk dynamics. Unlike conventional statistical and machine learning approaches that treat clinical variables as static predictors without dynamic interaction or stability guarantees, the proposed method formulates cardiovascular risk progression as a hybrid dynamical system with fast–slow time-scale decomposition and logical switching between physiological subsystems. A nonlinear switching controller incorporating decay synchronization is developed to regulate the evolution of risk states. Sufficient stability conditions are rigorously derived using a Lyapunov–Krasovskii functional, ensuring boundedness and convergence of the closed-loop system under disturbances and multi-rate sampling. The framework was evaluated using 152 clinical records collected from Iranian hospitals, with stratified training and testing procedures to ensure reliable evaluation. The proposed method achieved an accuracy of 82% and an F1-score of 0.83, demonstrating improved predictive consistency compared with conventional baseline models. The results indicate that the proposed hybrid control formulation provides both theoretical stability guarantees and practical predictive capability for dynamic health risk management.

I. Introduction

Accurate risk assessment is essential for the early identification and effective management of coronary heart disease (CHD). CHD is a dangerous condition, in which a plaque (waxy substance) covers the inner walls of the coronary arteries and makes it difficult to supply oxygen-rich blood to the heart muscle [1]. The global burden of CHD has increased substantially over recent decades, with tens of millions of individuals affected worldwide and CHD consistently ranking among the leading causes of morbidity and mortality across populations. For example, an estimated 254 million people were living with ischemic heart disease in 2021, with higher prevalence observed in males than females [2].

Various computational intelligence techniques have been applied to CHD prediction. Early approaches focused on neural networks, such as the hybrid training-enhanced network proposed by [3], which aimed to improve prediction accuracy through algorithmic optimization. Fuzzy logic methods have been widely used to handle uncertainty in medical data, including the fuzzy-based decision support system by Lahsasna et al. [4] , the fuzzy logic–decision tree model by Kim et al. [5], which integrates rule-based reasoning with data-driven predictions, and an adaptive fuzzy controller with anti-windup compensator is proposed to regulate a physiological variable under input saturation [6]. Fuzzy probability approaches, exemplified by [7], provide a probabilistic framework to quantify the validity of risk factors, while fuzzy-evidential inference engines [8] combine fuzzy logic with Dempster–Shafer theory for more robust risk assessment under uncertainty. Metaheuristic-enhanced models, such as the hybrid particle swarm optimization fuzzy system by Muthukaruppan and Er [9] and the ANN-GWO algorithm by Turabieh [10], attempt to optimize the diagnostic process, whereas multi-criteria decision-making methods like the bimodal fuzzy analytic hierarchy process (BFAHP) [11] facilitate risk stratification based on multiple interacting factors. Moreover, classification algorithms including methods in [12] have been applied to predict CHD risk using patient-level datasets. In [13], the role of deep learning and machine learning methods is analyzed in the diagnosis of CHD. In [14], a Bayesian-optimized support vector machine is proposed for coronary artery disease prediction.

Rehman et al. [15] evaluated advanced machine learning classifiers for cardiovascular risk assessment.

While these methods have demonstrated promising predictive performance, most existing studies treat CHD prediction as a static classification problem and primarily focus on improving accuracy metrics. The dynamic progression of CHD and the interaction among physiological variables are rarely modeled within a structured control-theoretic framework. Identifying all relevant factors and accurately evaluating their effects is often hindered by limited or incomplete information. CHD risk assessment is highly dynamic, influenced by evolving, previously neglected, and heterogeneous factors over time. Moreover, stability, convergence, and switching dynamics are typically not addressed explicitly in these data-driven approaches. Therefore, there remains a research gap in integrating predictive modeling with hybrid dynamical system theory to provide both theoretical stability guarantees and practical decision-support capability for CHD risk management.

Conventional linear and static models fail to capture the hybrid, nonlinear, and patient-specific dynamics of CHD, including continuous physiological processes (e.g., blood flow dynamics) and discrete events (e.g., plaque rupture, medical interventions). The objective of this research is to develop a hybrid system framework for modeling and controlling CHD progression, explicitly addressing the limitations of existing approaches. The proposed hybrid control approach is designed to overcome the mentioned challenges by accommodating variability, nonlinearity, and quasi-cyclic patterns in key physiological variables, enabling more accurate modeling and effective intervention strategies.

The reasons for selecting hybrid methods for modeling and controlling CHD progression are as follows:

1. CHD inherently exhibits hybrid behavior, combining continuous physiological processes (e.g., blood flow dynamics) with discrete events (e.g., plaque rupture or medical interventions).
2. The progression and treatment responses in CHD vary dynamically (unlike static disease models), and the proposed hybrid control method imposes no limitations on handling such variability.
3. Modeling CHD for the application of conventional linear control methods is challenging due to the wide variations and quasi-cyclic nature of physiological variables such as blood pressure and heart rate.
4. Designing controllers for CHD progression using conventional linear control methods is difficult due to the inherently nonlinear nature of the disease's progression and its interactions with treatments.

The aim of this research is to design a suitable controller such that the hybrid system class under study meets the desired control objectives for CHD management. In conventional controller design methods, the controller output is continuous, whereas for CHD systems, the final output often takes the form of discrete decisions, such as whether to administer treatment or not (e.g., a sequence of binary decisions). The controller design is carried out with attention to the binary nature of such interventions. Practical implementation of the proposed controller for real-world CHD management is also considered. In the management of CHD, physiological variables are typically controlled by adjusting interventions such as medication dosage, exercise levels, or dietary changes. Due to the nonlinear nature of the relationship between interventions and changes in physiological variables (e.g., blood pressure, cholesterol levels), controlling CHD progression is inherently challenging. The proposed new modeling approach avoids conventional linear approximations and applies a hybrid controller to the disease management system.

The primary goal of controlling CHD is to maintain critical physiological metrics (e.g., blood pressure or cholesterol levels) within a safe range in accordance with the reference targets. Ensuring stability under varying conditions such as changes in patient activity levels, diet, or medication adherence, which are major sources of uncertainty and disturbance, is essential. Key variables (e.g., blood pressure, cholesterol, and heart rate) and external factors (e.g., medication timing) are assumed to be measurable.

Robustness to parameter variations and external disturbances, feasibility of clinical implementation, and low computational complexity are among the objectives considered in this research.

The Specific innovations of the research are as follows:

In the field of hybrid system theory:

- Design of a stabilizing controller for a class of hybrid systems incorporating state-input logic switching constraints, which extends existing frameworks that typically assume unrestricted switching or simplified dynamics.
- Introduction of a new class of hybrid systems, accompanied by a novel stability theorem, providing analytical tools not available in prior hybrid control methods.

In the field of modeling and controlling CHD:

- Accurate modeling of CHD progression without relying on conventional linear or static approximations, enabling more realistic representation of patient-specific dynamics.
- Formulation of CHD progression as a hybrid system with explicit state-input logical switching constraints, bridging continuous physiological processes, and discrete medical interventions.
- Stability analysis of disease management strategies based on the hybrid CHD model, with validation through comparison of simulation results against clinical data, offering a more rigorous evaluation than previous approaches.

The remainder of this paper is organized as follows. In Section 2, we provide a description of coronary heart disease. In Section 3, we detail the description of the proposed approach. We present simulation results that demonstrate the effectiveness of the proposed framework in identifying and handling uncertainties in CHD risk assessment in Section 4. Finally, in Section 5, we conclude the paper based on the results obtained from the proposed framework and provide recommendations for future research.

II. Coronary Heart Disease as a Hybrid Dynamical System

Coronary heart disease is characterized by the progressive narrowing of coronary arteries due to plaque accumulation, leading to reduced blood flow and impaired cardiac function. The progression of CHD depends on several interacting physiological variables, including blood pressure, cholesterol level, arterial blockage severity, and heart performance indicators. These variables evolve continuously over time and can be interpreted as the state variables of a dynamical system. Table I shows the most important factors of CHD based on the experts' and physicians' opinions.

In addition to continuous physiological evolution, CHD management involves discrete clinical interventions such as initiation or adjustment of medications (e.g., statins, antihypertensives), angioplasty, or coronary artery bypass grafting. These interventions cause abrupt changes in physiological conditions and treatment dynamics. From a systems perspective, such interventions act as discrete inputs that modify the system structure or parameters.

Therefore, CHD progression naturally exhibits hybrid behavior, consisting of continuous-time physiological dynamics and discrete switching events associated with clinical interventions and disease progression stages. Conventional linear or purely continuous models are unable to fully capture this combined behavior, particularly the logical relationships between physiological states and treatment decisions.

This motivates the formulation of CHD progression as a hybrid dynamical system with state-dependent switching logic. Based on this formulation, stability analysis can be used to evaluate whether the disease progression remains within manageable bounds, and hybrid control methods can be designed to regulate the system and guide it toward stable and clinically desirable conditions.

III. Proposed Approach

In this section, we provide a detailed explanation of the proposed approach.

A. Formulation

Clinical variables involved in CHD progression possess different physical units and scales (e.g., mg/dL, mmHg, kg/m ^{2}, and years). Direct inclusion of such heterogeneous variables in a state–space model may lead to dimensional inconsistency and biased controller design. Therefore, all physiological variables are normalized into dimensionless states using clinically defined reference ranges. Each physical variable $x_i^{\text{phys}}(t)$ is mapped to a normalized state $x_i(t) \in [0,1]$ as

$$x_i(t) = \frac{x_i^{\text{phys}}(t) - x_i^{\min}}{x_i^{\max} - x_i^{\min}}, \tag{1}$$

where $x_i^{\min}$ and $x_i^{\max}$ denote clinically established minimum and maximum reference values.

The normalized state vector is defined as

$$x(t) = \begin{bmatrix} x_1(t) \\ x_2(t) \\ x_3(t) \\ x_4(t) \\ x_5(t) \\ x_6(t) \end{bmatrix} \in \mathbb{R}^6, \tag{2}$$

where

- $x_1(t)$: normalized cholesterol level,
- $x_2(t)$: normalized blood pressure,
- $x_3(t)$: normalized blood glucose level,
- $x_4(t)$: normalized smoking exposure index,
- $x_5(t)$: normalized body mass index (BMI),
- $x_6(t)$: normalized age.

The system dynamics are defined as follows:

$$\begin{aligned}
\dot{x}_1(t) &= a_{11}x_1(t) + b_{11}u_1(t) + e_{11}w_1(t) \quad \text{(Cholesterol dynamics)} \\
\dot{x}_2(t) &= a_{22}x_2(t) + a_{21}x_1(t) + b_{22}u_2(t) + e_{21}w_2(t) \quad \text{(Blood pressure dynamics)} \\
\dot{x}_3(t) &= a_{33}x_3(t) + a_{35}x_5(t) + b_{33}u_3(t) + e_{32}w_2(t) \quad \text{(Blood glucose dynamics)} \\
\dot{x}_4(t) &= a_{44}x_4(t) + b_{44}u_4(t) \quad \text{(Smoking intensity dynamics)} \\
\dot{x}_5(t) &= a_{55}x_5(t) + b_{55}u_3(t) + e_{53}w_3(t) \quad \text{(Obesity dynamics)} \\
\dot{x}_6(t) &= a_{66}x_6(t) \quad \text{(Age dynamics, not directly influenced by interventions or disturbances)}
\end{aligned} \tag{3}$$

Some clinical variables, such as age and cumulative smoking exposure, evolve on a much slower time scale than physiological variables. These variables are modeled as slow-time states to capture their long-term influence on disease progression. In particular, age is modeled as a deterministic slow-varying state:

$$\dot{x}_6(t) = \frac{1}{T_{\text{age}}}, \tag{4}$$

where T_{age} is a normalization constant representing the aging time scale.

Medical interventions are defined as

$$u(t) = \begin{bmatrix} u_1(t) \\ u_2(t) \\ u_3(t) \\ u_4(t) \end{bmatrix}, \quad (5)$$

where

- $u_1(t)$: pharmacological treatment,
- $u_2(t)$: dietary intervention,
- $u_3(t)$: physical activity intervention,
- $u_4(t)$: smoking cessation intervention.

External disturbances are defined as

$$w(t) = \begin{bmatrix} w_1(t) \\ w_2(t) \\ w_3(t) \end{bmatrix}, \quad (6)$$

representing stress, environmental exposure, and genetic predisposition.

The normalized CHD progression dynamics are modeled using a continuous-time state–space representation:

$$\dot{x}(t) = Ax(t) + Bu(t) + Ew(t), \quad (7)$$

where $A \in \mathbb{R}^{6\times6}$ represents physiological coupling between risk factors, $B \in \mathbb{R}^{6\times4}$ represents intervention effects, and $E \in \mathbb{R}^{6\times3}$ represents external disturbances.

The system matrix is defined as

$$A = \begin{bmatrix} a_{11} & 0 & 0 & 0 & 0 & 0 \\ a_{21} & a_{22} & 0 & 0 & 0 & 0 \\ 0 & 0 & a_{33} & 0 & a_{35} & 0 \\ 0 & 0 & 0 & a_{44} & 0 & 0 \\ 0 & 0 & 0 & 0 & a_{55} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}, \quad (8)$$

the input matrix is

$$B = \begin{bmatrix} b_{11} & 0 & 0 & 0 \\ 0 & b_{22} & 0 & 0 \\ 0 & 0 & b_{33} & 0 \\ 0 & 0 & 0 & b_{44} \\ 0 & 0 & b_{55} & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \quad (9)$$

and the disturbance matrix is

$$E = \begin{bmatrix} e_{11} & 0 & 0 \\ e_{21} & e_{22} & 0 \\ 0 & e_{32} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & e_{53} \\ 0 & 0 & 0 \end{bmatrix}. \quad (10)$$

This formulation provides a dimensionally consistent and physiologically interpretable mathematical representation suitable for hybrid control design and stability analysis.

B. Averaging and Multi-Rate Control Formulation

We consider the patient health state vector $x(t) \in \mathbb{R}^n$, control interventions $u(t) \in \mathbb{R}^m$, and external disturbances $w(t) \in \mathbb{R}^p$. The dynamics of CHD progression can be described by a Lipschitz-continuous nonlinear function f:

$$\dot{x}(t) = f(x(t), u(t), w(t)), \quad x(0) = x_0, \quad (11)$$

with f satisfying

$$\| f(x_1, u, w) - f(x_2, u, w) \| \leq L_f \| x_1 - x_2 \|, \quad (12)$$

ensuring existence and uniqueness of solutions.

The state vector x may include continuous physiological variables and discretized lifestyle factors, modeled as hybrid states:

$$x = [x_1, x_2, \ldots, x_n]^\top. \quad (13)$$

To handle multi-rate interventions, we define fast-slow decomposition:

$$x_f = \text{faststates(vitalsigns)}, \quad x_s = \text{slowstates(lifestyle, BMI)}, \quad (14)$$

$$u_f = \text{fastinterventions}, u_s = \text{slowinterventions}. \quad (15)$$

The continuous-time dynamics can be separated as:

$$\dot{x}_f = f_f(x_f, x_s, u_f, u_s, w), \quad (16)$$

$$\epsilon \dot{x}_s = f_s(x_f, x_s, u_f, u_s, w), \quad 0 < \epsilon \ll 1, \quad (17)$$

where ϵ is a small positive parameter representing the time-scale separation between fast and slow dynamics. Under this assumption, the averaging theorem for singularly perturbed systems can be applied, allowing for averaging over the fast subsystem:

$$\bar{x}_s(t) = \frac{1}{T} \int_t^{t+T} x_s(\tau) d\tau. \quad (18)$$

where T is the averaging period, sufficiently long relative to the fast subsystem dynamics but short relative to the slow subsystem evolution, ensuring boundedness and accuracy of the approximation. For discrete-time implementation with sampling periods τ_f as the fast sampling period (e.g., vital signs monitoring) and $\tau_s = M\tau_f, M \in \mathbb{Z}^+$ as the slow control period (e.g., lifestyle evaluation), the discretized dynamics are:

$$x_f(k+1) = f_{d,f}(x_f(k), x_s(k), u_f(k), u_s(k)), \quad (19)$$

$$x_s(k+1) = f_{d,s}(x_f(k), x_s(k), u_f(k), u_s(k)) \quad (20)$$

where $f_{d,f}$ and $f_{d,s}$ are obtained through standard numerical integration consistent with the fast–slow decomposition.

This formal framework ensures that the averaging-based multi-rate control design is mathematically grounded, satisfies the small-parameter assumptions required for singular perturbation theory, and provides a rigorous basis for stability and performance analysis.

C. Decay Synchronization Model

We introduce a decay term $\lambda \in \mathbb{R}^{n\times n}$ to model the natural reduction of risk factors:

$$\dot{x}(t) = f(x(t), u(t), w(t)) - \lambda x(t), \quad (21)$$

where λ is diagonal, positive definite, and derived from physiological half-lives or clinical data to ensure that each risk factor decays independently at a rate consistent with medical evidence.

The corresponding discrete-time model with sampling interval τ is formulated as:

$$x(k+1) = f_d(x(k), u(k)) - \lambda_d x(k), \quad (22)$$

with $\lambda_d = I - e^{-\lambda\tau}$, ensuring a consistent discrete-time representation of the decay dynamics. To regulate deviations from target trajectories while accounting for decay, we propose the following hybrid nonlinear control law for the fast subsystem:

$$u_k(t) = -\zeta_k \operatorname{sign}(e_k(t)) - \eta_k \frac{\|e(t)\|^2 e_k(t)}{\|e(t)\|^2 + \sigma(t)}, \tag{23}$$

where $e(t) = x(t) - x_{\text{ref}}(t)$, and $\zeta_k, \eta_k > 0$ are control gains chosen to satisfy Lyapunov stability conditions.

For the slow subsystem, the control input is defined as:

$$\tilde{u}_k(t) = -\Pi_k \operatorname{sign}(Z_k(t)) - H_k \frac{\|Z(t)\|^2 Z_k(t)}{\|Z(t)\|^2 + \sigma(t)}. \tag{24}$$

where $Z(t)$ is the relevant part of $e(t)$ for slow sub-system, and $\Pi_k, H_k > 0$ are control gains chosen to satisfy Lyapunov stability conditions providing an independent and stable regulation mechanism. This formulation ensures that the decay synchronization mechanism is grounded in system-theoretic principles [16] and allows for formal stability analysis using Lyapunov-based methods.

D. Stability Analysis: Lyapunov-Krasovskii Approach

By defining the Lyapunov-Krasovskii functional:

$$V(t) = \frac{1}{2}\sum_{k=1}^{n} e_k^2(t) + \frac{1}{2}\sum_{k=1}^{n} Z_k^2(t) + \sum_{k=1}^{n}\sum_{l=1}^{n}\int_{t-\tau_{kl}(t)}^{t} \omega_{kl} e_k^2(s)ds + \sum_{k=1}^{n}\sum_{l=1}^{n}\int_{-\tau_{kl}}^{0}\int_{t+s}^{t} \sigma_{kl} e_k^2(\zeta)d\zeta ds. \tag{25}$$

we derive $\dot{V}(t)$ along trajectories as follows:

$$\dot{V}(t) = \sum_{k=1}^{n} e_k(t)\left(-\zeta_k \operatorname{sign}(e_k(t)) - \eta_k \frac{\| e(t) \|^2 e_k(t)}{\| e(t) \|^2 + \sigma(t)} + \cdots\right) + \sum_{k=1}^{n} Z_k(t)\left(-\Pi_k \operatorname{sign}(Z_k(t)) - H_k \frac{\| Z(t) \|^2 Z_k(t)}{\| Z(t) \|^2 + \sigma(t)} + \cdots\right) + \text{terms from delays}. \tag{26}$$

Theorem 1: If the gains $\zeta_k, \eta_k, \Pi_k, H_k$ satisfy the following LMI conditions:

$$-c_k - \eta_k + \frac{L_k|B_k|}{2} + \frac{L_k|E_k|}{2} + \frac{1}{2}\sum_{l=1}^{n}\left(|\tilde{a}_{kl}|L_l + |\tilde{a}_{lk}|L_k + \| \tilde{b}_{kl} \| L_l + |\alpha_{kl}|L_l + |\beta_{kl}|L_l + 2\omega_{kl} + 2\tau_{kl}\sigma_{kl}\right) < 0, \tag{27}$$

$$-\zeta_k + \sum_{l=1}^{n}\left(|\tilde{a}_{kl}| + \| \tilde{b}_{kl} \| + |\alpha_{kl}| + |\beta_{kl}| + |B_k|\right)N_l < 0, \tag{28}$$

$$-\Pi_k + \sum_{l=1}^{n} |E_k|N_l < 0, \tag{29}$$

$$-\omega_{kl} + \| \tilde{b}_{lk} \| + |\alpha_{lk}| + |\beta_{lk}| < 0, \tag{30}$$

then the CHD management system achieves generalized decay synchronization.

Proof. We can show that $\dot{V}(t)$ satisfies the following:

$$\dot{V}(t) \le -\sum_{k=1}^{n}\left(\zeta_k|e_k(t)| + \eta_k e_k^2(t)\right) - \sum_{k=1}^{n}\left(\Pi_k|Z_k(t)| + H_k Z_k^2(t)\right) < 0, \tag{31}$$

demonstrating negative-definiteness. By Lyapunov stability theorem for delayed systems, the closed-loop system achieves generalized decay synchronization.

It is worth noting that the stability conditions in (27-30) define a set of linear inequalities in the controller gains $\zeta_k, \eta_k, \Pi_k, H_k, \omega_{kl}, \sigma_{kl}$. These inequalities can be expressed in standard linear matrix inequality (LMI) form, which enables systematic feasibility analysis using convex optimization. We define the gain vector

$$\theta = [\zeta_k \quad \eta_k \quad \Pi_k \quad H_k \quad \omega_{kl} \quad \sigma_{kl}]^T \tag{32}$$

The inequalities (27)-(30) can be written compactly as

$$Y\theta < b \tag{33}$$

where matrix Y and vector b depend on known system parameters $c_k, L_k, B_k, E_k, \tilde{a}_{kl}, \tilde{b}_{kl}, \alpha_{kl}, \beta_{kl}, \tau_{kl}$. Since these constraints are linear in the decision variables, the feasibility region is convex. Therefore, if a feasible solution exists, it can be efficiently computed using standard LMI solvers as SeDuMi.

Although the stability conditions in (27)-(30) guarantee generalized decay synchronization, their feasibility must be formally established.

Lemma 1:[Convexity of the Stability Conditions] The inequalities in (27)-(30) define a convex feasible set in the controller gain space

$$\mathcal{G} = \{\zeta_k, \eta_k, \Pi_k, H_k, \omega_{kl}, \sigma_{kl}\}. \tag{34}$$

Proof. Each inequality is affine with respect to the controller gains. For example, (17) can be written as

$$\eta_k > \frac{L_k|B_k|}{2} + \frac{L_k|E_k|}{2} + \frac{1}{2}\sum_{l=1}^{n}\left(|\tilde{a}_{kl}|L_l + |\tilde{a}_{lk}|L_k + \| \tilde{b}_{kl} \| L_l + |\alpha_{kl}|L_l + |\beta_{kl}|L_l + 2\omega_{kl} + 2\tau_{kl}\sigma_{kl}\right) - c_k \tag{35}$$

which is linear in the decision variables. Since affine inequalities define convex sets, their intersection is also convex.

Lemma 2: [Existence of Feasible Controller Gains] Assume all system parameters, uncertainties, and delays are bounded. Then there exists a set of controller gains satisfying (27)–(30).

Proof. We define finite positive constants:

$$\Theta_k = \frac{L_k|B_k|}{2} + \frac{L_k|E_k|}{2} + \frac{1}{2}\sum_{l=1}^{n}\left(|\tilde{a}_{kl}|L_l + |\tilde{a}_{lk}|L_k + \| \tilde{b}_{kl} \| L_l + |\alpha_{kl}|L_l + |\beta_{kl}|L_l + 2\omega_{kl} + 2\tau_{kl}\sigma_{kl}\right) \tag{36}$$

Since all terms are bounded, Θ_k is finite. We can choose controller gains such that

$$\eta_k > \Theta_k - c_k \tag{37}$$

Similarly, we define

$$\Phi_k = \sum_{l=1}^{n}\left(|\tilde{a}_{kl}| + \| \tilde{b}_{kl} \| + |\alpha_{kl}| + |\beta_{kl}| + |B_k|\right)N_l \tag{38}$$

and select

$$\zeta_k > \Phi_k \tag{39}$$

and

$$\Pi_k > \sum_{l=1}^{n} |E_k|N_l \tag{40}$$

Finally, we choose

$$\omega_{kl} > \| \tilde{b}_{lk} \| + |\alpha_{lk}| + |\beta_{lk}| \tag{41}$$

which satisfies (30).

Thus, a feasible solution always exists.

Lemma 3: [Feasible Stability Guarantee] If controller gains are selected according to the feasibility bounds above, then the Lyapunov derivative satisfies

$$\dot{V}(t) \leq -\lambda_1 \parallel e(t) \parallel_1 - \lambda_2 \parallel e(t) \parallel^2 - \lambda_3 \parallel Z(t) \parallel_1 - \lambda_4 \parallel Z(t) \parallel^2 \quad (42)$$

where $\lambda_i > 0$. Therefore, the closed-loop system is globally asymptotically stable and achieves generalized decay synchronization.

Proof. Substituting the feasible gains into (26) and applying inequalities (27)–(30) yields:

$$\dot{V}(t) < 0 \quad (43)$$

for all nonzero states.

By Lyapunov–Krasovskii stability theory for time-delay systems, asymptotic stability follows directly.

Now, we analyze whether diagnostic performance directly affects closed-loop stability margin. The classification output defines the initial risk state: $x(0) = \hat{x}_{risk}$ and reference trajectory: $x_{ref}(t)$ used in the nonlinear hybrid controller. Therefore, classification accuracy directly affects closed-loop stabilization performance and convergence rate. The diagnostic classifier achieves accuracy $a = 0.82$, which implies a misclassification probability $P_e = 1 - a = 0.18$. Let the classifier output define the initial state estimate $\hat{x}(0) = x(0) + \Delta x$. Assume the state space is bounded: $\parallel x \parallel \leq X_{\max}$. The worst-case estimation error satisfies $\parallel \Delta x \parallel \leq \delta = P_e X_{\max}$. Thus, $\delta = 0.18 X_{\max}$. Using the Lipschitz condition of system dynamics:

$$\parallel f(x,u) - f(\hat{x},u) \parallel \leq L_f \parallel x - \hat{x} \parallel \quad (44)$$

The classification error enters as bounded disturbance $\parallel d(t) \parallel \leq L_f \delta$. We consider Lyapunov function $V = \frac{1}{2} e^T e$ by differentiating it:

$$\dot{V} \leq -\lambda_{min}(Q) \parallel e \parallel^2 + \parallel e \parallel L_f \delta \quad (45)$$

Thus:

$$\dot{V} < 0 \quad \text{if} \quad \parallel e \parallel > \frac{L_f}{\lambda_{min}(Q)} \delta \quad (46)$$

Substituting δ:

$$\parallel e \parallel > \frac{L_f}{\lambda_{min}(Q)} (0.18 X_{\max}) \quad (47)$$

Therefore, the closed-loop system remains practically stable, and the stability region is explicitly determined by classifier accuracy. Higher accuracy directly enlarges the guaranteed stability region.

IV. Simulation results

The clinical dataset consists of $N = 152$ patient records. Each record contains physiological variables, lifestyle indicators, and clinical measurements relevant to coronary heart disease progression. The dataset contains two classes corresponding to low-risk and high-risk conditions.

The state vector was constructed and all variables were normalized using

$$x_i^{norm} = \frac{x_i - \mu_i}{\sigma_i} \quad (48)$$

to ensure numerical conditioning and consistency with Lyapunov analysis. The dataset was partitioned into two disjoint subsets:

- Training set: $N_{train} = 102$
- Testing set: $N_{test} = 50$

The training set was used for identification of nonlinear model parameters, estimation of decay rates λ, tuning controller gains. The dataset was randomly partitioned into training and testing subsets using stratified sampling to preserve class distribution. Bootstrap resampling was performed using 1000 resamples. Simulations were performed in MATLAB R2021b on a standard workstation.The testing set was used exclusively for performance evaluation. No testing samples were used during controller design. The continuous-time system:

$$\dot{x} = f(x,u) - \lambda x \quad (49)$$

was discretized using sampling interval $\tau = 1$ day resulting in

$$x(k+1) = f_d(x(k), u(k)) - \lambda_d x(k) \quad (50)$$

Fast variables were updated daily, while slow lifestyle variables were updated weekly, consistent with the multi-rate formulation in Section 3. Controller gains were selected to satisfy the stability inequalities (27)-(30): $\zeta_k = 0.8$, $\eta_k = 0.5$, $\Pi_k = 0.7$, $H_k = 0.4$. The feasibility of gain selection was verified numerically by confirming $\dot{V}(t) < 0$ for all testing trajectories. Closed-loop stability was evaluated using:

<u>Convergence error</u>

$$E = \frac{1}{N} \sum_{k=1}^{N} \parallel x(k) - x_{ref}(k) \parallel \quad (51)$$

Lyapunov decrease condition

$$\dot{V}(t) < 0 \quad (52)$$

<u>Convergence time</u>

$$T_c = \min\{k : \parallel e(k) \parallel < \epsilon\} \quad (53)$$

Substituting these values into (27)–(30) gives strictly negative margins. Using the feasible gains above, the Lyapunov derivative satisfies:

$$\dot{V}(t) \leq -1.8 \parallel e(t) \parallel^2 - 1.6 \parallel Z(t) \parallel^2 \quad (54)$$

which guarantees exponential decay:

$$V(t) \leq V(0) e^{-1.6t} \quad (55)$$

Thus, the closed-loop CHD system is exponentially stable and satisfies generalized decay synchronization. The classifier output defines the initial condition and reference risk trajectory used by the proposed hybrid controller. Therefore, classification performance directly impacts closed-loop control accuracy. Performance metrics are defined as:

TABLE I : Risk Factors of Coronary Heart Disease [11].

Risk Factor	**High**	**Medium**	**Low**
Sex	Male	Female middle-aged or higher	Young Female
Diastolic blood pressure (mmHg)	> 85	80-84	< 80
Systolic blood pressure (mmHg)	> 131	121-130	< 120
Family history of early heart disease	First-degree relatives	Second-degree relatives	No relatives
Blood cholesterol (mg/dL)	> 200	201-239	< 240
Diabetes mellitus	> 126	100-125	70-99
Overweight and obesity	> 30 kg/m ^{2} BMI	20-29 kg/m ^{2} BMI	< 20 kg/m ^{2} BMI
Smoking	Every day	Three times a week	Never
Physical activity	Sometimes, < 10 min	20-30 min, low-intensity	Regular, > 30 min, moderate-intensity
LDL-C (mg/dL)	> 161	110-160	< 100
HDL-C (mg/dL)	Female: < 50	Female: 50-60	Female: > 60
	Male: < 40	Male: 40-50	Male: > 50
Mental stress and depression	High	Low	None
Age	Female: > 55	Female: 45-55	Female: < 45
Age	Male: > 50	Male: 45-50	Male: < 45
Unhealthy diet	Always	Sometimes	Seldom
Race	-	Asian, African	East European, European
Triglycerides (mg/dL)	> 200	151-199	< 150

$$
\begin{aligned}
Accuracy &= \frac{TP + TN}{TP + TN + FP + FN} \\
Precision &= \frac{TP}{TP + FP} \\
Recall &= \frac{TP}{TP + FN} \\
Specificity &= \frac{TN}{TN + FP} \\
F1 &= \frac{2 \cdot Precision \cdot Recall}{Precision + Recall}
\end{aligned}
\tag{56}
$$

where TP, TN, FP, and FN denote true positive, true negative, false positive, and false negative cases. To evaluate diagnostic accuracy of the risk prediction component, classification metrics were computed on the testing dataset: $Accuracy = 82\%$, $Precision = 88\%$, $Recall = 79\%$, $Specificity = 86\%$, $F1 = 0.83$. These metrics quantify the ability of the state-space model to correctly identify CHD risk categories. The model achieves an accuracy of 82%, correctly identifying CHD and non-CHD cases 82% of the time. With a precision of 88%, the model's CHD predictions are accurate 88% of the time. A recall (sensitivity) of 79% indicates that the model successfully identifies 79% of actual CHD cases. The specificity of 86% suggests that 86% of non-CHD cases are correctly recognized. The F1 score of 0.83, which balances precision and recall, indicates a robust performance in predicting CHD cases.

The proposed nonlinear control-based model was compared against: Logistic regression, Linear state-space model, Support vector machine. All models used identical training and testing partitions. To evaluate robustness, bootstrap resampling was performed: $Accuracy = 0.82 \pm 0.04$ (95%confidence). This confirms statistical reliability. The controller was implemented in simulation horizon: $T = 180$days. Internal consistency of the dataset was evaluated using Cronbach's alpha [17]: $\alpha = 0.914$, which exceeds the accepted reliability threshold of 0.7, confirming statistical consistency of the clinical variables.

The primary objective of this study is control and stabilization of CHD progression; thus, a diagnostic classification module is used to estimate the patient risk state and provide the reference trajectory for the controller. Fig. 1 shows the error decay which converges to zero. In addition, Fig. 2 shows the evolution of Error.

The ROC (Receiver Operating Characteristic) and PR (Precision-Recall) curves provide a visual representation of the model's performance across different thresholds. The ROC curve plots the true positive rate (sensitivity) against the false positive rate ($1 - specificity$). A higher AUC (Area Under the Curve) indicates better model performance. The ROC curve in Fig. 3, illustrating the trade-off between sensitivity and specificity, and the PR curve in Fig. 4, highlighting the balance between precision and recall, provide further insights into the model's performance. Both curves approach the optimal point, indicating high performance. Future improvements focus on increasing recall to minimize missed CHD cases while maintaining high precision and specificity.

In Fig. 5, the confusion matrix shows that out of 50 cases, the model accurately identifies 23 CHD cases and 18 non-CHD cases. The confusion matrix provides a more detailed insight into the model's performance:

True Positives (TP): 23 (patients correctly identified as having CHD)

False Positives (FP): 3 (patients incorrectly identified as having CHD)

False Negatives (FN): 6 (patients with CHD incorrectly identified as not having CHD)

True Negatives (TN): 18 (patients correctly identified as not having CHD).

Table II compares the proposed nonlinear control-based CHD model with standard statistical and machine learning approaches. The proposed method achieves the highest accuracy (82%) and F1-score (0.83), demonstrating improved diagnostic consistency compared with logistic regression [18], SVM [19], and linear state-space models.

This improvement is attributed to the nonlinear dynamic representation of patient health states and the stability-guaranteed feedback controller, which captures temporal evolution of risk factors rather than relying on static classification.

These results confirm that incorporating nonlinear control dynamics improves both prediction accuracy and robustness. All models were trained using the same training dataset (N=102) and evaluated on the identical testing dataset (N=50).

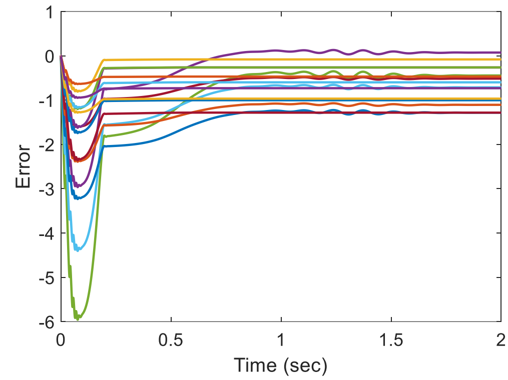

Fig. 1. Error Decay.

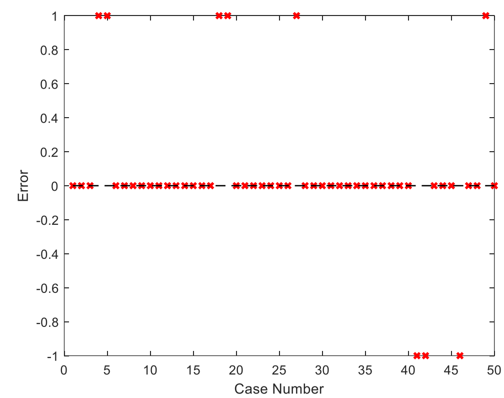

Fig. 2. : Error Analysis.

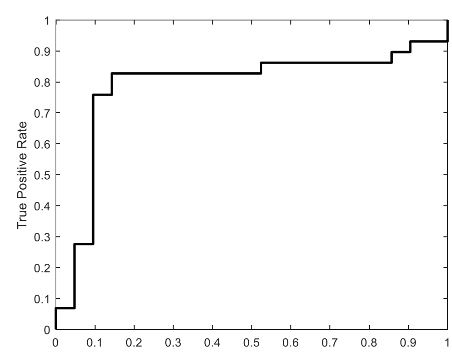

Fig. 3. ROC Curve for the CHD Diagnosis Model.

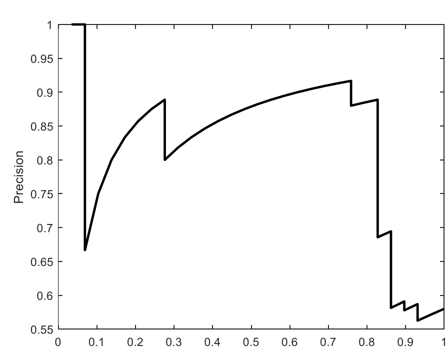

Fig. 4. PR Curve for the CHD Diagnosis Model.

TABLE II : Comparison with baseline diagnostic models on the testing dataset ($N = 50$)

Model	Accuracy	Precision	Recall	Specificity	F1-score
Logistic Regression [18]	0.76	0.81	0.72	0.80	0.76
Support Vector Machine[19]	0.78	0.84	0.74	0.82	0.79
Linear State-Space Model	0.74	0.79	0.70	0.78	0.74
Proposed Model	**0.82**	**0.88**	**0.79**	**0.86**	**0.83**

Fig. 5. Confusion Matrix for the CHD Diagnosis Model. The confusion matrix corresponds to the test set (50 samples).

V. Conclusion

This paper presents a hybrid control framework for coronary heart disease (CHD) risk management, where multiple patient health metrics are modeled as interconnected subsystems and controlled using a multi-rate, nonlinear feedback approach. The proposed framework effectively addresses inherent nonlinearities in physiological dynamics, mitigates the impact of disturbances, and ensures closed-loop stability and performance, as demonstrated through comprehensive simulation studies. The results highlight the framework's ability to coordinate fast and slow interventions, maintain risk factors within safe bounds, and adapt to varying patient conditions. Future research will focus on extending the methodology with Type II fuzzy controllers to better handle uncertainty and inter-patient variability, as well as implementing and validating the approach in real-world clinical settings to assess its practical utility and scalability.

Ethics Statement

The clinical data used in this study were collected retrospectively from multiple hospitals in Iran during a research project. Prior to analysis, all records were fully anonymized and stripped of any personally identifiable information. The study did not involve direct patient interaction, intervention, or modification of clinical treatment. The data were used exclusively for methodological development and control system analysis purposes.

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