Interactive Digital Twin of the Left Atrium Accelerated on NVIDIA GPUs

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Introduction

Heart disease and stroke are the leading causes of death worldwide [1,2]. Atrial arrhythmias are major contributors to stroke, heart failure, and, in some cases, acute myocardial infarction [3–5]. Catheter ablation is currently the most reliable and safest method available to physicians for treating patients with recurrent atrial arrhythmias [6–9]. However, much remains unknown about the underlying causes of atrial arrhythmias and the optimal placement of ablation lesions. As a result, ablation strategies remain as much an art as a science, with techniques refined over years of clinical practice. Gaining insight from procedures on live patients in the operating room poses obvious limitations. To address this, our group has developed a novel interactive computer model of the left atrium (LA). The LA was chosen because it is the most anatomically complex of the heart's four chambers and the primary site of most complex arrhythmias [10–13]. In this model, researchers can define muscle strand attributes that cause arrhythmias to emerge spontaneously. These arrhythmias can then be eliminated through simulated ablations. This approach enables researchers, outside of the operating room, to explore the initial conditions that lead to LA arrhythmias and to determine effective ablation strategies for eliminating them. The model's computationally intensive components were written in CUDA and accelerated on NVIDIA GPUs [14].

Cardiac Muscle Properties

Cardiac myocytes, or cardiac muscle cells, have vastly different refractory periods compared to skeletal muscle cells. The short refractory period of skeletal muscle, around 1–2 milliseconds, allows for rapid, repeated contractions. This enables skeletal muscles to produce strong, sustained contractions necessary for movement and posture. While this rapid firing is essential for skeletal muscle function, it would be fatal if cardiac muscle behaved the same way. The heart must contract to eject blood and then relax long enough to refill before the next beat. If the heart experienced sustained contraction, the rhythmic beating essential for life would stop. For this reason, cardiac muscle cells have a much longer refractory period, lasting approximately 250–300 milliseconds [15,16].

Note: For the remainder of this poster, we refer to a group of cardiac myocytes as "cardiac muscle" or simply "muscle."

Model Overview

The model consists of a set of nodes (mass points) sparsely connected by edges (muscles). When a node is activated by an electrical signal, it attempts to trigger every muscle it's connected to. Nodes can also be turned off (ablated), meaning they no longer propagate signals. Nodes carry the system's mass and move according to the forces applied to them by the muscles.

Cardiac muscles are modeled as massless springs with adjustable stiffness constants (K values). They apply force to connected nodes and also transmit electrical signals. Each muscle has an internal clock. When a muscle is in a rested state (i.e., "ready to fire"), its clock is at zero. When triggered by a signal, it starts its clock. If its clock is already running, it ignores any new signal. Depending on the elapsed clock time, the muscle applies a force to its two connected nodes. This force follows a bell-shaped curve to simulate the depolarization and repolarization phases of the cardiac refractory period. After a set duration, the muscle resets its clock, stops applying force, and waits for the next signal. When triggered, a rested muscle also sends a signal to the next node after a defined delay. If the muscle is already active, it ignores incoming signals. Both the contraction period and signal propagation speed can be adjusted at any time during the simulation.

Model Construction

Single Muscle Strand – Validating Propagation and Blockage

We began with a simple linear strand of cardiac muscle to demonstrate that an electrical wavefront, periodically triggered from a single node, could propagate along the strand and induce contractions, simulating a heartbeat. We tested whether wavefronts would appropriately halt at ablated nodes, validating the model's ability to represent conduction block.

Muscle Ring – Reentry Mechanism and Dual Pathways

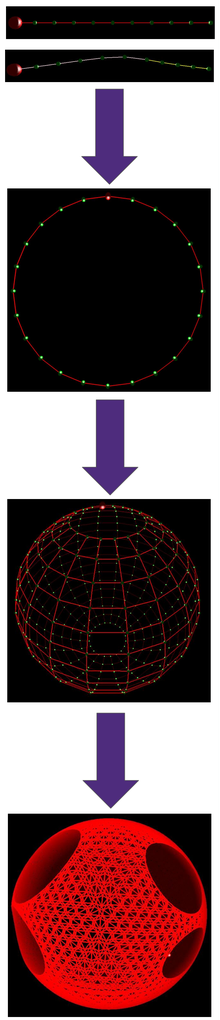
Next, we extended the strand into a closed ring by connecting its ends back to the pulse node. This configuration allowed us to test bidirectional propagation and observe wavefront interactions, specifically, whether they would cancel upon collision. By leveraging the model's ablation feature to selectively block and unblock nodes, we successfully reproduced a classic reentry tachycardia: a circulating wavefront overtook the beat from the pulse node by exploiting slow and fast conduction pathways, just as seen in clinical arrhythmia mechanisms.

Spherical Shell – Adding Directionality and Physiological Structure

We then expanded the model to a spherical shell, enabling us to observe multidirectional wave propagation, creating a more anatomically inspired framework. To approximate the right atrium, we removed the top and bottom nodes, representing the superior and inferior vena cava, and placed the pulse node near the edge of the top hole, mimicking the sinus node. A simulated blood pressure force was applied as an outward vector at each node. Although our final aim was to model the left atrium, this intermediate step established structural and functional validity. The extra spatial dimension allowed us to slow conduction in a small region of muscle and successfully generate a micro-reentry tachycardia, further validating the model's electrophysiological accuracy.

Left Atrium – Toward a Topologically Accurate Simulation

With these foundations in place, we developed an idealized yet topologically accurate model of the left atrium, incorporating key anatomical features such as the four pulmonary vein openings, the mitral valve, and Bachmann's bundle. To represent Bachmann's bundle, we created a tree-like structure of muscle fibers with increased conduction velocities, enabling rapid signal transmission across the left atrium in line with its physiological role. Additionally, we introduced a more intricate, biologically inspired muscle network, which improved both the stability of the model and the realism of electrical signal propagation.



Model Functionality

Our model includes real-time user interaction through callback functions, allowing dynamic adjustments during an active simulation. While all node and muscle properties are initially loaded from a user-defined setup file, users can modify them at any point using mouse and keyboard inputs or via an interactive GUI. This includes ablating nodes, adjusting the refractory period, changing signal propagation speeds, and introducing ectopic activity. Ectopic events (single triggers) and ectopic beats (recurring triggers with set frequencies) can be placed by clicking on nodes. Users can also change views, capture images and videos, and save configurations. This interactivity makes the model a powerful tool for both research and education, enabling users to create and analyze custom arrhythmias or test ablation strategies in real time.

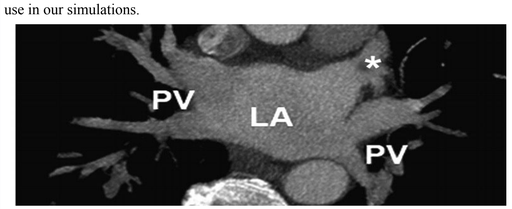
Results

Using this model, we successfully induced a left atrial flutter by slowing the conduction velocity of the muscle between two pulmonary vein openings and triggering an ectopic event at precisely the right location and time. Furthermore, we isolated the pulmonary veins and created a faulty ablation between the pulmonary vein groupings. With a properly timed ectopic event, this also induced a flutter that took control of the heartbeat. We were able to eliminate both of these flutters with simulated ablations, allowing the system to return to sinus rhythm.

2D Reentry | Micro Reentry | Atrial Flutter | Faulty PVI

Current Work

We are currently collaborating with Dr. Charles Puelz and Baylor College of Medicine on an NIH grant that will allow us to use computed tomography angiography (CTA) images from Texas Children's Hospital to construct an anatomically accurate, image-based, three-dimensional model of the left atrium for use in our simulations.



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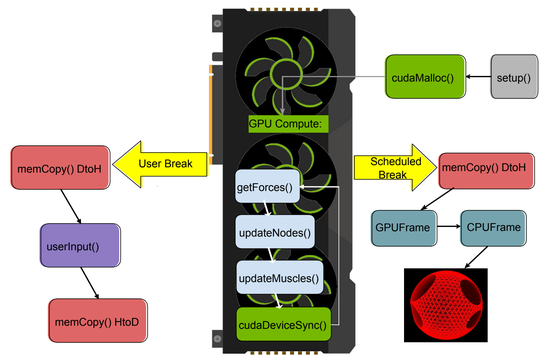
Methods

The code is written in C/C++ and CUDA. The simulations are accelerated on CUDA-enabled NVIDIA GPUs. Visualization and three-dimensional rendering are done in OpenGL and Blender.

- Blender-generated assets implemented with Python BPY API
- Generated nodes and muscles to lay over Blender assets
- Offloaded nodes and muscles to GPUs
- Periodic nodal dump for visualization
- Uses breakpoints to interact with the simulation



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References

[1] World Health Organization. The top 10 causes of death. World Health Organization;12/9/2020.https://www.who.int/news-room/fact-sheets/detail/the-top-10-causes-of-death.
[2] Virani SS, Alonso A, Aparicio HJ, Benjamin EJ, Bittencourt MS, Callaway CW, et al. Heart disease and stroke statistics-2021 update: a report from the American Heart Association. Circulation 2021 Feb 23;143(8):e254–743. https://doi.org/10.1161/CIR.0000000000000950. Epub 2021 Jan 27. PMID: 33501848.
[3] Brundel BJJM, Ai X, Hills MT, Kuipers MF, Lip GYH, de Groot NMS. Atrial fibrillation. Nat Rev Dis Primers. 2022 Apr 7;8(1):21. doi: 10.1038/s41572-022-00347-9. PMID: 35393446.

Full reference list