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The cardiovascular system

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Abstract: this article gives information about modelling and simulating the growth processes that allow the cardiovascular system to adapt to the overall body development and tochanging physiological (and pathological) conditions. Within the cardiovascular system, we pay particular attention to the heart and the aorta.

Keywords: Cardiovascular system; adaptability; growth and remodelling.

A healthy aortic wall succeeds in keeping a homeostatic stress level in spite of long-standing alterations in pressure or flow by triggering growth and remodelling processes that change its stress-free shape and its structure. A normal heart grows in response to the gradually increasing haemodynamic loading exerted on myocardial fibres. Postnatal cardiac growth is a form of volume-overload hypertrophy, produced essentially by a progressive myocardial cell enlargement, with no cell proliferation involved. In our continuum model, growth is basically conceived of as the time evolution of the stress-free configuration of the tiny fragments into which the modelled tissue may be subdivided in imagination. It is governed by a novel balance law—the balance of accretive couples—independent of, but constitutively coupled with, the standard balance of forces.

The Cardiovascular System and its Adaptability

The primary function of the cardiovascular system is the transport of oxygen, carbon dioxide, nutrients, waste products within the body. Since the body undergoes major changes during its life cycle, the capability of the cardiovascular system to accommodate vastly increasing body demands is a vital requirement.

The system consists primarily of the heart, which serves as the pump, the blood, which serves as the conducting medium, and the vasculature, which serves as the conduit through which the blood flows.1 We focus on the heart and the aorta—the main artery of the body, supplying oxygenated blood to the circulatory system. In particular, we strive to model the growth process through which the aortic wall keeps a homeostatic stress level and the postnatal hypertrophy—either physiological or pathological—of the cardiac muscle. Both of these processes take place on a time scale—weeks to years—which is extremely long if compared with the heartbeat time scale. Nevertheless, understanding the basic machinery of the cardiac cycle is crucial to our endeavour, since physiological adaptation and pathological developments are both triggered by the values attained during a cardiac cycle by rather gross mechanical quantities, such as blood pressure and heart volume. This is demonstrated by an overwhelming physiological and medical evidence, even though the detailed feedback mechanisms are still largely unknown.

The following two subsections collect some basic information on the structural and functional properties of the heart and the aorta, respectively, as an essential preliminary to mathematical modelling. For a fairly recent overview on the biomechanics of cardiovascular development, the reader is referred to the survey paper by Taber.

Structure and function of the heart

The heart is a smart composite structure. Atria and ventricles have a different microstructural organization; also heart valves and ventricle walls possess a different microstructure; moreover, the heart walls are comprised of three different layers, with diverse structure and function. The inner layer—endocardium—and the outer layer—epicardium—are both very thin, each being about 100 μm thick; the middle layer, called myocardium, constitutes the bulk of the cardiac tissue and endows it with the ability to pump blood.

The myocardium is composed of cardiac myocites and fibroblasts, surrounded by an extracellular matrix. In adults, cardiac myocites are typically 10 –20 μm in diameter and 80 –125 μm in length; their cytoplasm contains mainly myofibrils (1–2 μm in diameter). Each myofibril consists of a string of contractile units, called sarcomeres, each of which is about 2 μm long. Each sarcomere consists of hundreds

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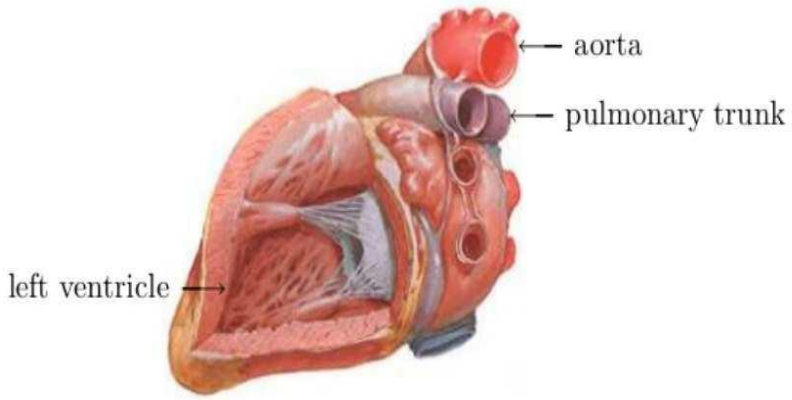
of filamentous protein aggregates (myofilaments). Thick myofilaments are composed of several hundred molecules of myosin; thin myofilaments are composed of two helically interwound polymers of actin.

Cardiac myocites are active components: they contract and relax at a high frequency producing the heartbeats; on a much longer time scale, they can grow bigger (hypertrophy). Muscle contraction is initiated by the release of calcium ions which are sequestered in the surrounding sarcoplasmic reticulum. A smooth ratcheting action (with a speed of about 15 µm/s) results from the shortening of the sarcomere, operated by the action of the actomyosin cross-bridges that release, move forward, and reattach. The release of calcium ions is triggered by an action potential that spreads from the cell membrane. The passage of the electrical signal from cell to cell is facilitated by the fact that myocites are highly interconnected. The biochemical mechanisms that drive the long-term hypertrophy process are more intriguing and far less understood. Some coarse-grained feedback mechanisms are hypothesized in Sec. 3.2, as a first step in our modelling effort.



The aorta as a prototype large vessel

Arteries are roughly classified as elastic or muscular. Elastic arteries are larger and are located close to the heart, like the aorta; muscular arteries are smaller and closer to the arterioles. The walls of both elastic and muscular arteries consist of three different layers: intima, media, and adventitia.

The intima, which is the innermost of the threes and consists of a single layer of endothelial cells, is extremely thin with respect to the arterial wall, at least in large arteries. Its contribution to the mechanical properties of the wall may only become significant in old age or under degenerative conditions: atherosclerosis, in fact, implies a thicker and stiffer intima. The middle layer—the media—is characterized by a complex mixture of smooth muscle cells, elastin and collagen fibrils. Vascular smooth muscles modify the distensibility of large arteries and regulate the lumen size in medium and small arteries. Consistent with their different roles, smooth muscles are organized differently in large (elastic) and in small (muscular) arteries. In elastic arteries, vascular smooth muscles are organized in musculoelastic fascicles—that is, alternate layers of smooth muscle and elastin (up to 70 layers in the human aorta). In muscular arteries, elastin is absent and smooth muscles form a single layer embedded in a matrix of connective tissue. The outermost layer—the adventitia—consists primarily of collagen fibres arranged in helical structures. It contributes significantly to the strength of the arterial wall.

The mechanical properties of an arterial wall are approximately homogeneous within each single layer, but not overall. In fact, the difference in stiffness exhibited by different layers plays an important role in the adaptive response of the arterial wall to perturbations from its homeostatic state.

Conclusion: In order for the aortic valve to open, it is necessary that the left-ventricle pressure exceeds the aortic pressure. Now, the value attained by the pressure inside the ventricle at the end of the isovolumic contraction is directly correlated with the stress generated within the ventricle wall by themyocardial contraction.32 A crude estimate of this correlation is readily provided by a simplistic application of Laplace's formula for a pressurized thin-walled spherical container, establishing that the ratio (surface tension)/(intramural pressure) equals the ratio (container diameter)/(wall thickness). This motivates the assumption—consistent with abundant empirical data—that the ventricle wall grows thicker as it grows

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wider. Therefore, cardiac hypertrophy has to multiply sarcomeres also transversally to myofibrils, packing more of them in parallel within each single myocite.

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