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Mathematical Modelling of the Cardiovascular System

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Abstract

We describe the problem of modelling the cardiovascular system and give current references.

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Chapter 1

Prologue

The human heart is a pump which converts metabolic energy to mechanical work and generates the pressure that drives the blood throughout the body's circulation system. It can do so for 70 years or more, with a rate varying from about 5 litres/min in an adult human male at rest to 35 litres/min in Olympic athletes. For an organ which is about the size of a fist this is a remarkable achievement.

In 1993 we began a longterm project to examine ways of modelling the heart, specifically its mechanical properties. This requires several steps:

- The relevant features of the heart must be formulated.
- Quantitative experimental data must be obtained.
- Appropriate mathematical models must be constructed.
- As far as possible, the models should be analyzed mathematically.
- Numerical methods must be used to obtain quantitative results.
- After comparing theory and experiment, the models must, if necessary, be modified.

There is an extensive literature on this subject. Nevertheless, we hoped to be able to make a meaningful contribution by taking advantage of the combined physiological, morphological and mathematical knowledge of the authors. Particular emphasis was to be placed on the fibre architecture of the myocardium and its influence on ventricular mechanics.

Since the mathematician amongst us (CWC) was a newcomer to the field, it was natural to begin by reading the literature and making notes. As the breadth of the subject became apparent, the notes evolved into the present text, which may therefore be particularly useful to applied mathematicians who wish to contribute to a fascinating area. While our principal interest lies in the mechanics of the heart, this cannot be considered in isolation and we include descriptions, sometimes extremely brief, of the often fascinating problems connected with other parts of the cardiovascular system.

The text is intended for use in a course on the mathematical modelling of the cardiovascular system, for mathematics students with little knowledge of physiology, physics, or chemistry. Hence some basic background information, with references to additional sources, is given in appendices. In the course of acquainting himself with the subject CWC found information such as the addresses of cardiovascular societies, sources of materials, etc. to be very useful, and this is also included. In this way we hope to have provided not just a textbook but also a sourcebook for anyone entering the field.

The modelling of the cardiovascular system is the subject of several recent books for engineers and physicists. Here we emphasize the mathematical aspects:

- By giving specific examples which can then be subjected to mathematical and numerical analysis.
- By drawing attention to open problems.
- By drawing attention to potentially useful mathematics.

The text differs from many books on mathematical modelling in that we assume an adequate knowledge of partial differential equations, such as could be provided by a two-semester course on the theory of partial differential equations and their numerical solution.

In the past, mathematicians have contributed relatively little to the subject and one may very well ask whether there is anything that they can do which could not be done better by the physicists, biomechanical engineers, etc. already working in the field. We would like to think so and hope that this book will help to bring this about. One source of encouragement is our experience that, given the opportunity and appropriate help, many mathematics students welcome the opportunity to model biological processes.

We are grateful to Butterworth and Heinemann Ltd for granting permission to reproduce several figures and short pieces of text from *An Introduction to Cardiovascular Physiology* by J.R. Levick [Lev91].

The present text would never have been conceived and written had it not been for the enthusiasm of the second author, Paul Peter Lunkenheimer, who spent many hours explaining and demonstrating the workings of the heart to his mathematical colleague.

Part I

Biomechanics

Chapter 2

The Human Machine

In this text we will primarily be concerned with modelling one aspect of the human body, the cardiovascular system, and in particular, the heart. The approach used mathematical modelling is, in principle, straightforward. We consider the individual components of the human body. For each component, for example the lungs, we seek to set up a set of mathematical equations, which embody the physical processes within the component. These equations, which are usually a combination of algebraic, ordinary and partial differential equations, provide a mathematical model which can be used for analysis and prediction.

In practice, mathematical modelling is often difficult. This is certainly the case in modelling the human body, where as one moves from the macroscopic to the microscopic and then to the molecular level, new structures are revealed.

This introductory part of the text serves two purposes:

- To discuss some of the general features of man considered as a machine
- To introduce the mathematical reader to some basic ideas and to give him or her a feel for the size of some relevant physiological quantities.

There are some well-written non-mathematical introductions to biomechanics and physiology

- **Life's Devices** by Steven Vogel [Vog88]
- **The Human Machine** by Alexander [Ale92a]

There are also several excellent texts on mathematical biology which include sections on modelling the human machine:

- Hoppensteadt and Peskin, *Mathematics in Medicine and the Life Sciences* [HP92]
- Murray, *Mathematical Biology* [Mur93]
- Mazumdar, *An Introduction to Mathematical Physiology and Biology* [Maz89]

And now, without more ado, let us turn to the task in hand.

Chapter 3

Energetics

We begin by recalling some basic facts about the human body considered as a mechanical device, the *human machine*. It may be noted here that there appears to be a fundamental difference between mathematical modellers and most physiologists. A modeller needs model problems, that is, problems whcih embody typical features. In the present context, the modeller would like to have *model man* or *model human machine*. There is of course no such thing, and physiologists seem to have little need to create it. The *normal* or *average* data given below have had to be extracted by us from several sources.

A normal young man weighs W = 70 kg [1]. Corresponding to a normal height H,

$$H = (70 + 100)cm = 1,7m.$$

His body surface area is about $1.8m^2$. Approximately 47% of the body's weight is made up of water.

A normal young man has an oxygen consumption of

$$\dot{V}_{O_2} = 250mlO_2/min,$$

breathing on average of $500ml$ of air twelve times per minute.
Rasch (cite???, p. 100) uses examples from weight lifting to illustrate the power and strength of athletes. He also quotes the following maxima for the output of a human being:

- $6hp = 6 * 0.746kW \doteq 4.5kW$ for a single movement lasting less than 1 second.
- 0.5 to $2hp \doteq 0.375$ to $1.5kW$ for exercise lasting between 1 and 5 minutes.
- 0.4 to $0.5hp \doteq 0.3$ to $0.375kW$ for steady-state work for 5 min to 150 min or more.
- $0.2hp \doteq 0.15kW$ for long-term work.

The book *The Fire of Life* by Kleiber [Kle75] is a well-known introduction to animal energetics, which contains various interesting details but is also somewhat incoherent.

[1] For more details and references see Appendix E where the average woman is also described.

Chapter 4

Mechanics

Part II

The Heart

Chapter 5

Introduction

The heart is the pump in the cardiovascular system. In this chapter the general features of this system are described. In subsequent chapters, more detailed information is provided.

We have drawn heavily on standard texts, including *Cardiovascular Physiology* by J. R. Levick [Lev91], *Cardiovascular Physiology* by R.M Berne and M.N. Levy [BL92], *Cardiovascular Physiology* by Milnor [Mil90], and *The Cardiovascular System - The Heart* edited by R. M. Berne [Ber79].

Many experimental results are only available for non-humans, and it is in any case often useful to compare results for different animals. When discussing non-human physiology, this will be explicitly stated.

5.1 The Circulatory System

The mammalian cardiovascular system consists of the heart and blood vessels. The basic function of this system is to transport blood. In this process oxygen and other products are conveyed to the tissues and waste products such as carbon dioxide and urea are removed.

The heart consists of two adjacent intermittant muscular pumps, the **right ventricle** and **left ventricle**. Each pump is filled from a reservoir, the **right atrium** or **left atrium** respectively. The designation *left* or *right* is from the perspective of the patient; the left ventricle is on the left side of the patient's body. Since the heart lies across the chest in a human, the right ventricle and atrium are towards the front (anterior) while the left ventricle and aorta are towards the rear (posterior). The right ventricle pumps blood through the lungs to the left atrium (the **pulmonary circulation**) while the left ventricle simultaneously pumps blood through the rest of the body and back to the right atrium (the **systemic circulation**). The blood is compelled to follow a circular pathway by one-way valves located in the heart and veins, as was first established by the London physician, William Harvey and published in 1628 [Har28], [Har93b] (see Figure 5.1).

- In the pulmonary circulatory system venous blood enters the right atrium from the two major veins, the **superior vena cava** and **inferior vena cava**, and the **coronary sinus**, then flows through a valve into the right ventricle. This ventricle, which is composed mainly of cardiac muscle, receives the blood while it is in a state of relaxation called **diastole**. Contraction, or **systole**, then forces part of the blood out through the pulmonary artery and into the lungs at a low pressure. Gases exchange by diffusion in the lung air sacs raising the blood oxygen content from approximately 150 ml/l (venous blood) to 195 ml/l[1] The oxygenated blood returns through the pulmonary veins to the left atrium and left ventricle.

- In the systemic circulatory system the left ventricle contracts virtually simultaneously with the right and ejects approximately the same volume of blood but at a much higher pressure. The blood flows through the aorta and the branching arterial system into fine thin-walled tubes called **capillaries**. Here the dissolved gases and nutrients diffuse between the capillary blood and the tissue cells. The circulation of the blood is completed by the **venous system** which conducts blood back to the venae cavae.

[1]See Appendix C for a discussion of the relevant physical chemistry. The statement that the oxygen content of venous blood is 150 ml/l means that 1 litre of venous blood contains the equivalent of 150 ml of Oxygen (O_2) at STPD. (Standard Temperature and Pressure, that is, 0°C and atmospheric pressure.)

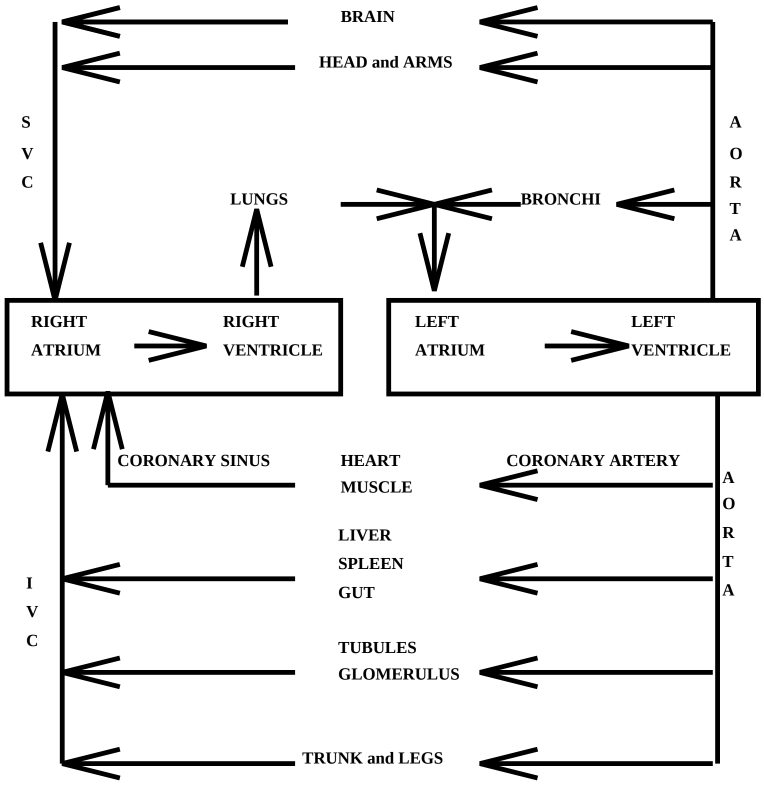

Figure 5.1: The (simplified) circulatory system

The flow of blood in the cardiovascular system is shown in simplified form in Figure 5.1. There are four 'circuits': the pulmonary 'circuit', where blood flows from the right ventricle through the lungs to the left atrium; the 'upper' or 'superior' circuit where blood flows through the aorta to the head and arms, returning via the superior vena cava (SVC); the 'lower' or 'inferior' circuit where blood flows through the aorta to the lower body, returning via the inferior vena cava (IVC); and the 'coronary' circuit where blood flows through the coronary artery to the heart muscle, returning via the coronary sinus. Together, the superior, inferior and coronary circuits make up the systemic circuits. The return of blood from the bronchii is anomalous, since it flows into the left atrium. Under normal conditions the bronchial blood flow constitutes at most 1 % of the venous return to the heart. In certain pathological states the bronchial circulation may become substantial. See Berne and Levy [BL92] page 245.

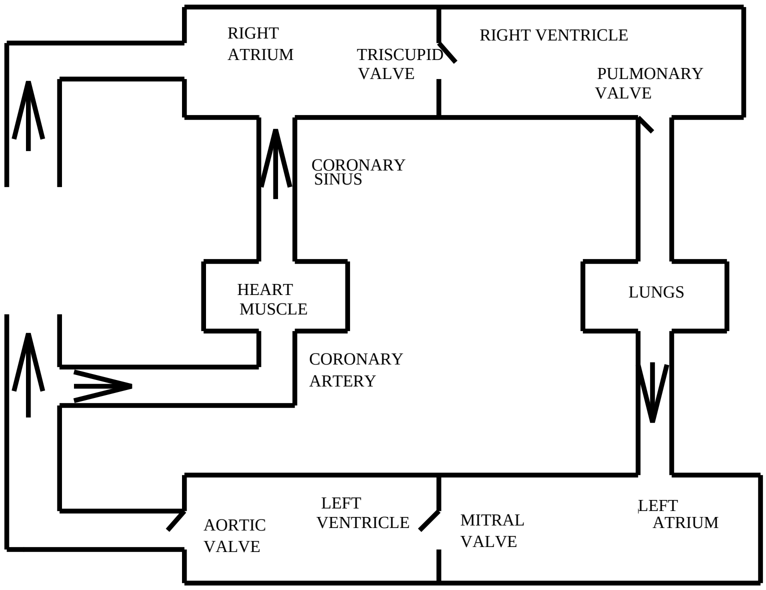

Figure 5.2: The plumbing of the normal heart

The flow of blood through the normal heart can be represented functionally as in Figure 5.2.

Each ventricle has an inlet valve (the mitral and tricuspid valves, respectively) and an outlet valve (the aortic and pulmonary valves, respectively). These valves open and close at appropriate times during the cardiac cycle, as described in Section 6.2.

In Figure 5.2 the supply of blood to the heart muscles through the **coronary artery** and **coronary sinus** is shown separately, because this blood supply is influenced by the pressures in the heart wall, and is of importance for the modelling of the heart.

5.2 The Heart

In an adult male weighing 60 kg the heart weighs about 350 grams and roughly occupies a cube with sides of length 10 cm. The location of the heart is shown in Figure 5.3.

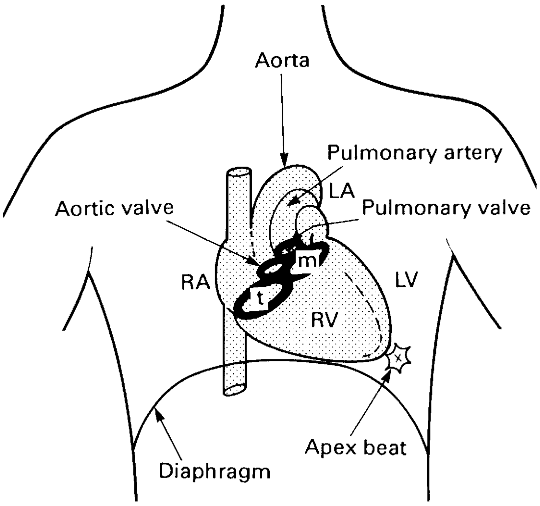

Figure 5.3: The location of the heart
The heart lies obliquely across the chest. The fibrotendinous ring (black) forms the base of the heart. It contains the tricuspid (t), mitral (m), aortic and pulmonary valves grouped in an oblique plane beneath the sternum. The apex of the heart is formed by the left ventricle (LV), and the anterior surface is formed by the right ventricle (RV) and right atrium (RA). The inferior surface of the heart and the pericardium (not shown) rest on the central tendon of the diaphragm. (From J.R. Levick, *An Introduction to Cardiovascular Physiology*, by permission.)

As is shown in Figure 5.4 (a) the heart consists of two adjacent pumps, the **left ventricle** and (**right ventricle**). Blood flows into each ventricle from an antechamber, the **left atrium** (**right atrium**), through a valve the **mitral valve** (**tricuspid valve**). Blood leaves the left (right) ventricle through another valve, the **aortic valve** (**pulmonary valve**). The edges of each 'inflow' valve are tethered by 'strings', the **chordae tendineae**, to two muscles, the **papillary muscles**, which bulge from the ventricle's walls.

The wall dividing the left ventricle and atrium from the right ventricle and atrium is called the **septum**. The four heart valves lie approximately in a plane, the intersection of which with the heart is called the **fibrotendinous ring** (see Figure 5.3).

The right ventricle only pumps blood to the lungs and back to the left atrium while the left ventricle must pump blood through the whole body. As to be expected, the outer wall of the right ventricle is much thinner (about 0,5 cm) than the outer wall of the left ventricle (about 1,5 cm). This is illustrated in Figure 5.4 (b), which shows a view of the lower part of the two ventricles.

5.2.1 Visualization

Since the heart is three-dimensional it is quite difficult to visualize; for a non-medical reader it may help to use a plastic model, such as can be obtained from medical bookshops or, even better, the prepared heart of an animal. Recently, videos showing the motion of the heart have become available[2].

[2]See Appendix K for the names and addresses of some suppliers.

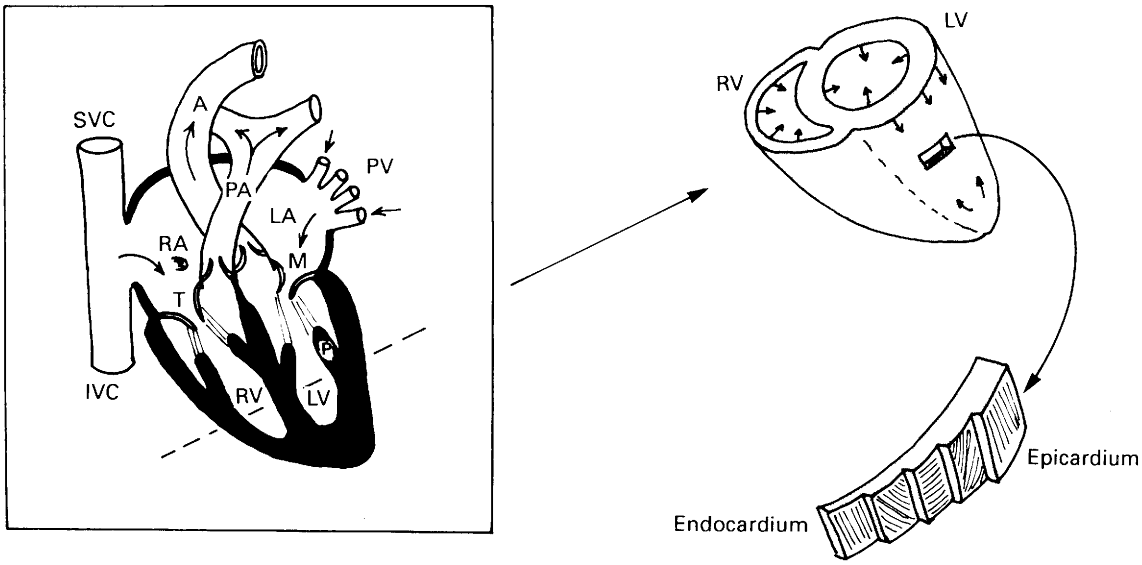

Figure 5.4: Cross-sections of the heart
(a) Schematic diagram of an oblique section. (b) Section across the ventricles to illustrate mode of emptying. (c) Arrangement of muscle fibres in the ventricle wall. RA, LA, right and left atrium. The opening just below the label RA is the coronary sinus. RV, LV, right and left ventricle; T, M, tricuspid and mitral valves; P, papillary muscle with chordae tendineae; A, aorta; PA, PV, pulmonary artery and veins; SVC, IVC, superior and inferior venae cavae (From J.R. Levick, *An Introduction to Cardiovascular Physiology*, by permission.)

Several books provide extensive drawings and photographs:

- Anderson and Becker, **The Heart. Structure in Health and Disease** [AB92].
- M. J. Davies, **Colour Atlas of Cardiovascular Pathology** [Dav86]
- Thomas, Gebert, and Hombach **Grundlagen der klinischen Medizin. Anatomie, Physiologie, Pathologie, Mikrobiologie, Klinik: Band 1: Herz und Gefaesse** [TGH89]
- Vesalius **The Illustrations from the Works of Andreas Vesalius of Brussels** [Ves50b]

For the text of Anderson and Becker an accompanying set of slides is available.

5.3 Disease and Pathology

An important justification for modelling the cardiovascular system is that it may suggest ways to improve the treatment of illness. Examples of this approach are given in *McDonald's Blood Flow in the Arteries* pages 350 - 356 [NO90].

An immense variety of pathological situations is observed in practice. Every part of the heart can be affected, either through congenital defects or disease.

See:

- Anderson and Becker, **The Heart. Structure in Health and Disease** [AB92].
- Acierno, *The History of Cardiology* [Aci94]
- Braunwald, *Heart Disease* [Bra92]
- Davies, *Colour Atlas of Cardiovascular Pathology* [Dav86]
- Julian and Cowan, *Cardiology* [JC92]
- Lilly, *Pathophysiology of Heart Disease* [Lil93]
- Thomas, Gebert, and Hombach **Grundlagen der klinischen Medizin. Anatomie, Physiologie, Pathologie, Mikrobiologie, Klinik: Band 1: Herz und Gefaesse** [TGH89]
- White, *Heart Disease* [Whi51]

Pathological situations present a challenge to mathematical modelling. A model should be able to correctly reproduce the effects of any pathology. Such a model would be a valuable diagnostic tool. No current models have such capabilities. We believe that this represents a tremendous opportunity.
In this connection we observe that the following discuss changes in heart function caused by disease:

- Grant *Architectonics of the heart* [Gra53]

5.4 Comparative Physiology

A great deal can be learnt about the human cardiovascular system by considering the cardiovascular systems of other animals:

- Some measurements are more easily made on certain animals.
- For the development and testing of a good theory it is of importance to be able to vary parameters, e.g. the size of the heart.

The prime purpose of a cardiovascular system is to transport oxygen within the body. Two physical processes are involved:

1. Diffusion, which is only effective over short distances, because the average time t required for a particle to diffuse a distance x is given by

$$t = x^2/2D$$

   where D is a constant, the diffusion coefficient. For example, for a solution of glucose in water at 37°, $D = 0.9\ 10^{-5}\ cm^2/s$, so that the time required to diffuse 1 cm is

$$t = \frac{10^5}{2 \times 0.9} s = 55556 s = 15.4\,\mathrm{h}$$

   that is 15 hours and 24 minutes.

   Even over short distances, nature compensates for the slowness of diffusion by providing large surfaces:

   - The lungs have a surface of about $50 - 100m^2$ in a normal adult (Guyton [Guy91] page 427)
   - The blood capillaries offer a surface of $500 - 700m^2$ (Guyton [Guy91] page 170.)See Cameron [Cam89] and Weibel [Wei84].

2. Convection, which is effected through the circulatory system and is effective over long distances.

Each animal has its own system of oxygen distribution, and nature has provided a marvellous range of alternatives, some of which are listed below:

- No circulatory system.
- Open circulatory systems in which blood is pumped by the heart through a system of blood vessels, but then moves freely between the tissues of the body.
- Closed circulatory systems in which the blood is pumped by the heart through a system of blood vessels which it never leaves.

In circulatory systems many possible arrangements of the heart(s) occur:

- One pump (e.g. the frog)

- One bidirectional pump (e.g. in *tunicates*), see Schmidt-Nielsen 1990, [SN90], page 95.
- Two pumps in one heart (mammals and birds)
- Three separate pumps (e.g. the octopus - see Steven Vogel [Vog92], page 36)

The great range of cardiovascular mechanisms is clearly reviewed in Chapter 11 of Scheer [Sch60], while in Chapter 15 of Prosser [Pro91] more details are given, including tables of blood volumes and blood pressures in various animals.

Other references include:

- *Comparative Animal Physiology* by Prosser [Pro91].
- *Comparative Physiology* by Bradley T. Scheer [Sch48]
- *Animal Physiology* by Knut Schmidt-Nielsen [SN90]
- *Vital Circuits* by Steven Vogel [Vog92]
- *Biologie in Zahlen* by Flindt [Fli95]

5.5 History of Cardiology

The history of cardiology is a fascinating topic. For the interested reader we can wholeheartedly recommend the translation of Harvey's *The Circulation of the Blood* [Har93b], which is beautifully clear, even for today's reader. Foster's *Lectures on the History of Physiology* [MF24] is interesting but alas long out of print. The recent book, *The History of Cardiology* by Acierno [Aci94] is well-written and describes not only historical contributions but also quite recent developments. Other currently or until recently available references are:

- Giovanni Alphonso Borelli, *On the Movement of Animals*, 1989, [Bor89]
- Clendening, *Source Book of Medical History* [Cle60]
- Harris, *The Heart and Vascular System in Ancient Greece* [Har73]
- Leibowitz *The History of Coronary Heart Disease* [Lei70]
- Andreas Vesalius, *Epitome*, [Ves50b]
- Andreas Vesalius, *The Illustrations from the Works of Andreas Vesalius of Brussels*, [Ves50a]
- Willius and Keys*Classics of Cardiology (previously called Cardiac Classics*, [WK83]

Further references are given by

- Acierno [Aci94].

It is of interest to look at old textbooks such as:

- Braun, *Diagnose und Therapie der Herzkrankheiten* [Bra13]

- Evans, *Starling's Principles of Human Physiology* [Eva36]
- Grollman, *The Cardiac Output of Man in Health and Disease* [Gro32]
- Tigerstedt, *Die Physiologie des Kreislaufes* [Tig23]

Chapter 6

The Cardiac Cycle

6.1 Introduction

The basic facts about the cardiac cycle are described. Some standard texts are:

- Braunwald, Ross, jr., and Sonnenblick,*Mechanisms of Contraction of the Normal and Failing Heart* [BRS76]
- Baan, Arntzenius and Yellin, *Cardiac Dynamics*, [BAY80]
- Burton, *Physiology and Biophysics of the Circulation. An Introductory Text* [Bur72]
- Berne, *Handbook of Physiology. Section 2. The Cardiovascular System. Vol. I. The Heart* [Ber79]
- Frank, *Zur Dynamik des Herzmuskels*, [Fra95]
- Katz, *Physiology of the Heart* [Kat92]
- Noble, *The Cardiac Cycle*, [Nob79]
- Opie, *The Heart. Physiology and Metabolism*, [Opi91]
- Rushmer, *Cardiovascular Dynamics* [Rus68] This textbook for medical students was a standard reference.
- ter Keurs and Tyberg, *Mechanics of the Circulation* [tKT87]

6.2 The Cardiac Cycle

The cardiac cycle corresponding to one full beat of the human heart lasts about 0.9 seconds (for a resting person). The cycle comprises four parts during which the inlet valves (mitral and tricuspid) and outlet valves (aortic and pulmonary) open and close as appropriate.

The four parts of the cardiac cycle described above are traditionally divided into two phases - **systole** and **diastole**. During the systolic phase (isovolumetric contraction and ejection) the heart is actively pumping blood and the heart muscles are tensed. During the diastolic phase (isovolumetric relaxation and ventricular filling) the heart muscles are relatively passive. However, this terminology is somewhat confusing since, see e.g. McCulloch and Omens [MO91] p. 89, the heart muscle is not fully relaxed even during the diastolic phase.

Typical measurements of pressure and flow on the left side of the human heart (adult, resting, supine) are shown in Figure 6.1. The following description of events is based on the book of Levick ([Lev91], pages 15 - 19); we have added some comments in footnotes.

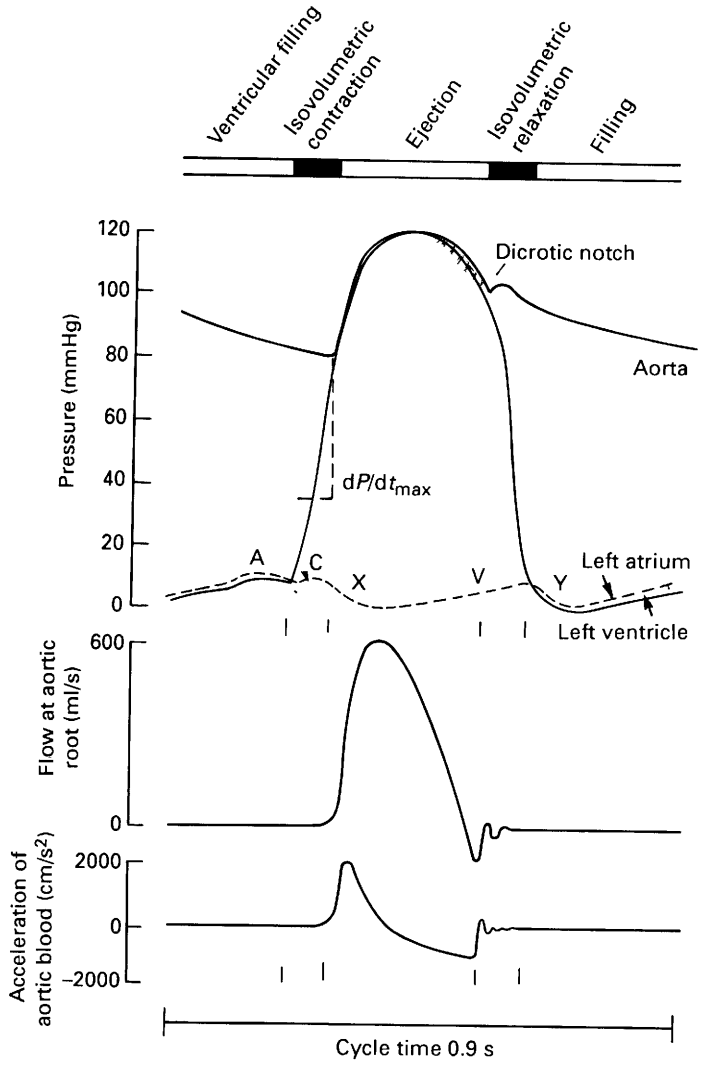

Figure 6.1: The cardiac cycle

Diagram of pressure and outflow on the left side of the human heart, based on data from intracardiac catheters and velocity measurements at the aortic root. $(dP/dt)_{max}$ is the maximum rate of rise of ventricular pressure, a measure of myocardial contractility. The stippled region highlights the pressure gradient which decelerates outflow during the late ejection phase. Note the slight reversal of aortic flow at aortic valve closure. The waveforms in the right heart are of similar shape but the pressures are lower. From J.R. Levick, *An Introduction to Cardiovascular Physiology*, by permission.)

6.2.1 Ventricular filling

Duration: 0.5 s
Inlet valves (tricuspid and mitral): open
Outlet valves (pulmonary and aortic): closed

Initially the atria are in diastole and blood flows passively from the great veins through the open atrioventricular valves[1] into the ventricles. There is an initial phase of rapid filling, lasting about 0.15 s, which has a curious feature; even though ventricular volume is increasing, ventricular pressure is falling. The reason is that the ventricle wall is recoiling elastically from the deformation of systole, and is in effect sucking blood into the chamber. As the ventricle reaches its natural volume, filling slows down and further filling requires distension of the ventricle by the pressure of the venous blood; ventricular pressure now begins to rise. In the final third of the filling phase, the atria contact and force some additional blood into the ventricle. (PPL fragen) In resting subjects, this atrial boost is quite small. During exercise, however, when the time available for passive ventricular filling is curtailed, the atrial boost becomes important. The volume of blood in a ventricle at the end of the filling phase is called the **end-diastolic volume**, or **EDV**, and is typically around 120 ml in an adult human. The corresponding **end-diastolic pressure**, or **EDP**, is a few mmHg.

6.2.2 Isovolumetric contraction

Duration: 0.05 s
Inlet valves (tricuspid and mitral): closed
Outlet valves (pulmonary and aortic): closed

Ventricular systole lasts 0.35 s and is divided into a brief isovolumetric phase and a longer ejection phase. As soon as ventricular pressure rises fractionally above atrial pressure, the atrioventricular valves are forced shut by the reversed pressure gradient. Backflow during closure is minimal because the cusps are already approximated by vortices behind them in the late filling phase [2]. The ventricle is now a closed chamber, and the growing wall tension causes a steep rise in the pressure of the trapped blood.

[1] The **atrioventricular valves** are the tricuspid and mitral valves, which link the atria and ventricles.

[2] Although the flow around the tricuspid and mitral valves has been extensively studied, we are not aware of a precise justification for this assertion. Discussed in Section 9.4.2.1

6.2.3 Ejection

Duration: 0.3 s
Inlet valves (tricuspid and mitral): closed
Outlet valves (pulmonary and aortic): open

When ventricular pressure exceeds arterial pressure, the outflow valves are forced open and ejection begins. Three quarters of the stroke volume are ejected in the first half of the ejection phase (0.15 s) and at first blood is ejected faster than it can escape out of the arterial tree. As a result, much of it has to be accommodated by distension of the large elastic arteries, and this drives arterial pressure up to its maximum or 'systolic' level. Vortices behind the cusps of the open aortic valve prevent the cusps from blocking the adjacent openings of the coronary arteries.[3]

As systole weakens and the rate of ejection slows down, the rate at which blood flows away through the arterial system begins to exceed the ejection rate and pressure begins to fall. Active ventricular contraction actually ceases about two-thirds of the way through the ejection phase, but a slow outflow continues for a while owing to the momentum of the blood. As the ventricle begins to relax, ventricular pressure falls below arterial pressure but the outward momentum of the blood prevents immediate valve closure. Finally a brief backflow (comprising less than 5% of stroke volume) closes the outflow valve. Valve closure creates a brief pressure rise in the arterial pressure called the **dicrotic wave**.

The ventricle does not empty completely; the average **ejection fraction** in man is 0.67, corresponding to a **stroke volume** of 70-80 ml in adults. The residual **end-systolic volume** of about 50 ml acts as a reserve which can be utilized to increase stroke volume in exercise.

[3] As of December 1993 we do not know of a reference for this statement.

6.2.4 Isovolumetric Relaxation

Duration: 0.08 s
Inlet valves (tricuspid and mitral): closed
Outlet valves (pulmonary and aortic): closed

With closure of the aortic and pulmonary valves, each ventricle once again becomes a closed chamber. Ventricular pressure falls very rapidly owing to the mechanical recoil of collagen fibres within the myocardium, which were tensed and deformed by the contracting myocytes. When ventricular pressure has fallen just below atrial pressure, the atrioventricular valves open and blood floods in from the atria, which have been refilling during ventricular systole.

6.3 Global Motion of the Heart

The heart is not stationary but moves relative to the rest of the body during each cardiac cycle. The rotation of the left ventricular apex has been measured by Buchalter, Weiss et al. [BWR +90]

6.4 The Pericardium

When the chest is opened, the motion of the heart is largely hidden from view because of the **pericardium**, a fibrous fluid-filled sac in the thoracic cavity which encloses and restrains the heart. The pericardium reduces friction between the heart and surrounding organs and provides a barrier against infection.

The pericardium consists of two layers: a strong outer layer of tough fibrous tissue, the **fibrous pericardium** and an inner double-layer sac, the **serous pericardium**. The fibrous pericardium is firmly attached within the body. The serous pericardium is normally filled with fluid.

The influence of the pericardium on the motion of the heart has been discussed by several authors (see McCulloch and Omens [MO91], p. 94). The consensus of opinion seems to be that the pericardium only plays a small role for normal hearts under normal conditions. Indeed, humans with no pericardium (as a result of a congenital defect) do not usually show any unusual symptoms. (see [Bra92] p. 1506.) However, Braunwald [Bra92], p. 1465, lists three roles for the pericardium which deserve consideration by modellers:

- The distribution of hydrostatic forces on the heart
- The prevention of acute cardiac dilation
- Diastolic coupling of the two ventricles

In the case of illness or injury the pericardium probably plays an important role in restraining the heart see Braunwald [Bra92]. Over a period of time, the pericardium can adapt to changes in the size of the heart.

In health, the pericardium may be unobtrusive but there are several illnesses associated with the pericardium including (see Chapter 45 of Braunwald [Bra92]):

- **pericarditis acute** caused by inflammation of the pericardium
- **pericardial effusion** caused by an increase in the amount of fluid in the pericardial space [4] According to Braunwald [Bra92], p. 1474, the pericardial space in humans normally contains between 15 and 50 ml of fluid. If additional fluid increases slowly, the pericardium can stretch, and can accommodate up to 2 l of fluid without apparent discomfort. If the fluid increases rapidly, or the pericardium is not sufficiently flexible, then a marked rise of intrapericardial pressure will occur. This can lead to **cardiac tamponade** which is characterized (Braunwald [Bra92], p. 1473) by:
  - Elevation of intracardiac pressures
  - Progressive limitation of ventricular diastolic filling
  - Reduction of stroke volume and cardiac output
- **pericarditis constrictive** which occurs when a fibrotic, thickened and adherent pericardium restricts diastolic filling of the heart.

Rowell [Row93] p. 180 and pages 195 - 199 discusses the role of the pericardium. It has been demonstrated experimentally (Rowell [Row93] Figure 5.10 p. 182) that **pericardectomy** (the surgical removal of the pericardium) leads to a substantial (10%) increase in stroke volume for exercising dogs. Rowell [Row93] p. 199 comments on anecdotes that racing dogs have had the pericardium cut to improve their performance. Blomqvist and Salting [BS83] have suggested that physical conditioning may alter apparent ventricular compliance by modifying right and left ventricular pericardial interactions (see Rowell [Row93] p. 196) Tyberg, Misbach, Parmley and Glantz [TMPG80] discuss the role of the pericardium. They suggest (see p. 167 with confirming experimenta data) that "the pericardium has a dominant role in the mechanism of acute shifts in the left ventricular diastolic pressure-volume relationship". The authors suggest that the observations can be explained by a simple **hydraulic model**. As long as the volume of the heart is less than the unstressed volume of the pericardium, the diastolic pressure in the left ventricle is the same whether the pericardium is open or closed. However, when the volume of the heart exceeds the unstressed volume of the pericardium, the ventricular diastolic pressure equals the transmural pressure necessary to distend the ventricle plus the pericardial pressure. This implies that changes in the left ventricular diastolic pressure whenever the unstressed volume of the pericardium has been exceeded. The authors point out that such shifts in the pressure-volume curve are important in explaining the response of patients with cardiac failure to **vasodilators**.

6.5 Heart Sounds and Murmurs

During the cardiac cycle, a variety of sounds, clicks, murmurs, etc., is produced and provides valuable diagnostic information. The generation of some of these noises has also been analyzed mathematically, as will be discussed later (see Peskin [Pes75] p. 115, Pedley [Ped80], page 55 and page 308.

[4] The terms *pericardial space* and *pericardial sac* are used interchangeably.

Some common **heart sounds** are listed below. For more details and descriptions of other sounds, see Lilly [Lil93], Chapter 2; Luisada [Lui73a], [Lui73b]; Luisada and Portaluppi [LP82]; Guyton [Guy91], Chapter 23; Novey, Pencak and Stang, [NPS88]

1. **Heart Sounds** There are four recognized heart sounds of which two are easily heard:

   S_1 The first heart sound is caused by the closure of the tricuspid and mitral valves.

   S_2 The second heart sound is caused by the closure of the aortic and pulmonary valves. The mechanism of heart sounds (as observed in experiments) is considered by Luisada and Portaluppi [LP82], p. 53.

2. **Heart Murmurs**
   Heart murmurs are abnormal sounds caused by turbulence, which is normally absent. Typically this turbulence is caused by two types of valve defects (see Levick [Lev91] page 21):

   - **Stenosis** is a narrowing of a valve so that a high-pressure gradient is needed to force blood through a stenosed valve.
   - **Incompetence** is failure of a valve to close tightly, thus allowing a **regurgitation** of blood.

3. Korotkoff Sounds
   Korotkoff sounds are heard when blood pressure is measured (in the usual way) by placing a cuff containing an inflatable bag around the upper arm. If the cuff is first inflated, and then the pressure is gradually reduced, there comes a point when the blood begins to flow through the brachial artery again ; at this point, the Korotkoff sounds can be heard in a stethoscope placed against the artery.

6.6 Concluding Remarks

In this chapter we have described the basic observations of the mammalian heart. Some, such as the Korotkoff sounds or the maximum blood pressure, can be made by elementary non-invasive means and form part of the diagnostic techniqes of every doctor. Others, such as the measurement of the blood pressure at the aortic root, require the use of sophisticated invasive techniques. All these observations have been made and recorded innumerable times.

It is the job of the modeller to predict and explain these observations.. To do so, requires much more detailed knowledge. In the remainder of this Part we attempt to summarize what is known. It will become apparent that there are often surprising gaps in this knowledge.

Chapter 7

The Structure of the Myocardium

7.1 Introduction

The myocardium is a complex fibrous continuum, which contains not only the muscle cells or **myocytes** but also an extensive network of connective tissue. We refer to Levick [Lev91] Chapter 3 for a description of individual myocytes and their function and to Lunkenheimer, Lunkenheimer and Torrent Guasp [LLG85] Chapter 3 for photographs of specimens.

7.2 Connective Tissue

The myocardium is a complex fibrous continuum which contains not only the muscle cells but also an extensive network of connective tissue. It is reasonable to suppose that this tissue can contribute to the expansion of the ventricles during diastole.

This idea has a very long history (see Lunkenheimer, Lunkenheimer and Torrent Guasp [LLG85], pages 62-66), but much more detailed information has now become available through microscopic analysis.

Hunter and Smaill [HS89] review recent findings and consider the formulation of constitutive laws. A. Horowitz, Y. Lanir, F.C.P. Yin, M. Perl, I. Sheinman and R. K. Strumpf, [HLY +88] consider constitutive laws for the passive myocardium.
The individual myocytes have a definite direction and together form muscle fibres which are clearly visible after preparatory treatment, such as boiling. The determination of the direction of the fibres has been the subject of investigation and speculation for many centuries. Influential contributions were made by Lower (1669), Pettigrew (1860, 1864), MacCullum (1900), Mall (1991), Flett (1928) [Fle28] and Thomas [Tho57]. Flett [Fle28] includes "as complete a bibliography a possible" of the literature as of 1928.

The earlier workers recorded their findings in often very attractive drawings. To our knowledge, the first detailed quantitative measurements are due to Hort [Hor57] and Hort

[Hor60]. Although the individual myocytes are joined together, the determination of the direction field of the fibres is not easy. The literature has therefore tended to split:

I. On the one hand there are many classical descriptions of the complicated twisted form taken by the fibres. As described by Borelli in 1680 [Bor89] p. 249 :

> The fibres of the heart are not straight and parallel to each other but curved and coiled. They are amazingly intricate. Their texture is not similar to that of a wicker-basket, as Vesalius believed, but they are arranged in a more sophisticated way. Immediately underneath the pericardium, from the basis of the heart and from the circular tendinous orifices of the vena cava and pulmonary auricle, and from the origins of the aorta and pulmonary arteries, a layer of fleshy fibres spreads. These fibres are parallel to each other and they are orientated from the basis straight to the apex of the heart where they are diversely curved and intertwined, reflecting towards the cavities of the ventricles. This layer is followed by other layers of fibres, oblique and descending in spirals. They are more and more inclined and orientated also towards the apex of the heart. Before reaching the apex, they intersect and cover each other and other ordinary fibres. Hence they are deviated to the inside and partly reflected in oblique and transverse spirals like scythes, towards the base of the heart. Part of them seem to compose internal columns to which the strings of the tricuspid and mitral valves are attached and others are intertwined transversally and form the cavity of the right ventricle.

Grant [Gra65] briefly and succintly summarizes earlier work on the muscular architecture of the left ventricle. He describes the work of Lower and Mall and points out that Lower drew attention to the twisting of the fibres - Grant (p. 304) compares this (with reservations) to a twisted rope. Grant also discusses the possible role of embryonic development in the structure of the ventricle (see Grant [Gra65] page 305).

Greenbaum, Ho, Gibson, Becker and Anderson [GHG +81] briefly summarized previous work on the fibre architecture and report the outcome of a study using dissection and histology on 25 normal postmortem human hearts. They conclude (p. 248): "Models based on uniform myocardial fibre structure cannot explain wall movement in normal subjects, and are likely to have significant limitations if used to investigate left ventricular function in disease."

Lunkenheimer, Lunkenheimer and Torrent Guasp [LLG85] and [LW93] have extensive discussions of the fibre architecture. The latter reference, in particular, contains a detailed evaluation of current knowledge.

As participants of this conference are well aware, Torrent Guasp [Gua73] has extended this work and postulated a model, based on these observations, of twisting fibres.

II. On the other hand, many models assume that:

1. The fibre directions vary smoothly through the heart wall.

2. The fibres run parallel to the inner and outer surfaces.

3. The innermost (endocardial) and outermost (epicardial) fibres run longitudinally from the base to the apex, the central fibres run circumferentially, and the other fibres run obliquely.

These assumptions are usually justified by reference to the papers of Streeter and Basset [SB66] who measured fibre orientation in pig heart and Streeter, Spotnitz, Patel, Ross and Sonnenblick [SSP +69] who measured fibre orientation in canine hearts. (See also the Ph.D. thesis of Streeter [Str69] and the article by Streeter [Str79].) However, this is an oversimplification of the results of Streeter who was well aware of the earlier work. Streeter did measure transmural fibre directions , but asserted that the associated angle α_3 (which he called the **imbrocation angle**) was smaller than about 5°.
The measurements of Streeter were based on a small number of samples at nonrandom sites. The actual situation is much more complex, as can be seen from the pictures of treated hearts in Lunkenheimer, Lunkenheimer and Torrent Guasp [LLG85] pages 38-53. Several authors have commented on the shortcomings of the results of Streeter. For example, Hunter and Smaill [HS89], page 106 comment:

> However, despite the large number of studies there is still a dearth of quantitative information on ventricular muscle fibre orientation. No studies appear to have measured the muscle fibre orientation at more than 8 sites on a single heart.

As reported by Lunkenheimer (personal communication) the fibres frequently dip into the surface with an angle of as much as 45°, contradicting assumptions 1 and 2 above.

As McLean and Prothero [MP91b] p. 392 state:

> We have no a priori reason to assume that the global (large-scale) organization of the myofibers in the heart can be inferred with confidence from a study restricted to the local organization at one or a few sites in the developing or adult heart wall.

At present several studies are under way which should provide much more detailed information about the fibre orientation. McLean, Ross and Prothero [MRP89] have mapped fibre directions in the mouse heart, and report that they apparently differ significantly from those in the human heart.[1] Nielsen [Nie87] and Nielsen, Le Grice, Smaill and Hunter [NLSH91] have made detailed measurements of fibre orientation in the canine heart. (See also McLean, Ross and Prothero [MRP89])

7.3 Previous Mathematical Models

Until recently only a few models took account of the fibre direction. A few authers have considered fluid-fibre models, in which the myocardium is treated as a fluid with an embedded fibre matrix. Such models thus ignore the elasticity of the myocardium, but, because of their simplicity can sometimes be solved analytically. See:

- Caflisch, 1987, [Caf87]
- Fung, 1984, [Fun84] p. 70

[1] Unfortunately this work has been discontinued

- Skalak, 1982, [Ska82]
- Chadwick, 1981 und 1982, [Cha81], [Cha82]

It seems clear, that the results should be incorporated into models. See:

- Bovendeerd, Huyghe, Arts, van Campen and Reneman, *Influence of endocardial-epicardial crossover of muscle fibers on left ventricular wall mechanics* [BHA +94]
- Bovendeerd, Arts, Huyghe, van Campen and Reneman *Dendence of local left ventricular wall mechanics on myocardial fiber orientation: A model study* [BAH +92]

Yang, Taber and Clark [YTC94] page 215 briefly mention observations on fibre orientation in embryonic chick hearts which they incorporate into their model.
Grant [Gra65] briefly and succintly summarizes earlier work on the muscular architecture of the left ventricle. He describes the work of Lower [Low69] and Mall [Mal11] and points out that already Lower had drawn attention to the twisting of the fibres - Grant compares this (with reservations) to a twisted rope (page 304). Grant also discusses the possible role of embryonic development (see Grant [Gra65]). Finally Grant (page 306) also observes that on its outer surface (epicardium) the left ventricle has two vortices which merge below the surface. The right ventricle has its own vortex.

See also:

- Puff [Puf60]
- Flett [Fle28]
- Mall [Mal11]
- MacCullum [Mac00]
- Thomas [Tho57]
- Streeter and Ramon [SR83]
- Streeter, Daniel, Powers, Ross and Torrent Guasp [SPRG78]

7.4 The Pericardium, Endocardium and Epicardium

For observations on the **pericardium**, **endocardium** and **epicardium** see:

- Faller [Fal44]

Chapter 8

Experimental Observations

It is obviously difficult to obtain observations of the heart. The layman may well feel that direct measurement must be nigh to possible. However, the heart is in some ways remarkably robust as one author (CWC) first realized when holding a beating pig heart with one hand. There is a substantial and rapidly growing body of knowledge which serves both as a challenge and a guide for modelling. There seems to be a definite need to design experiments so as to confirm confound theoretical and numerical predictions.

8.1 The Ventricular Function Curve

The measurements in Figure 6.1 refer to a normal working heart. However, the heart continues to function outside the body. In 1895 Otto Frank [Fra95] studied the frog[1] ventricle and measured the relation between pressure and volume (see Knowlton and Starling [KS12], Markwalder and Starling [MS14], Patterson and Starling [PS14], Patterson, Piper and Starling [PPS14], as well as Starling's *Principles of Human Physiolog* Evans, [Eva36] and Levick [Lev91] page 68). This work was extended by Starling and his collaborators around 1912. Figure 8.1 (from Levick p. 69 [Lev91]) shows the experimental setup. The Starling resistor shown in Figure 8.1 a device to maintain constant arterial pressure, is discussed in detail by Fung ([Fun84], page 188),
The results of this classical experiment are shown in Figure 8.2 where the **stroke volume** is plotted against the filling pressure (for an isolated dog heart).

A graph whose ordinate is stroke volume or any other measure of contractile energy, and whose abscissa is filling pressure or any other index of resting fibre length is called a **ventricular function curve**, or **Starling curve**.

The upward part of the curve is the basis of the celebrated **Frank-Starling-Law** or **Starling's Law of the Heart**, which asserts, that **the greater the stretch of the ventricle in diastole the greater the stroke volume**. This law explains a basic control mechanism of the heart.

[1]The frog heart has two atria but only one ventricle

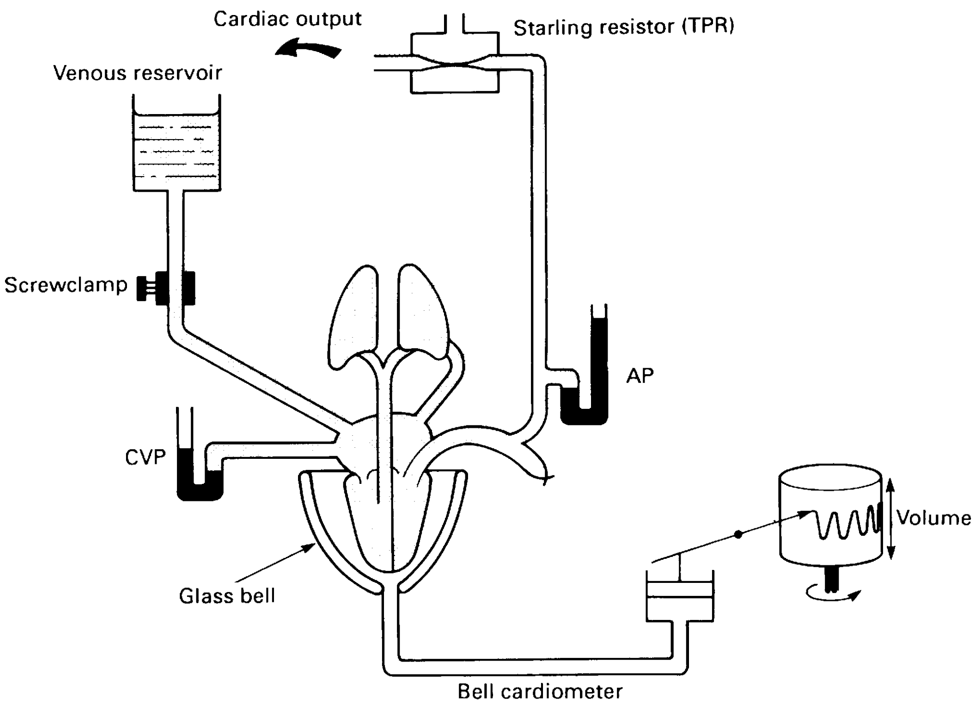

Figure 8.1: Simplified diagram of isolated dog heart-lung preparation of Starling. The height of the venous reservoir and the screwclamp regulated central venous pressure (CVP). CVP and arterial pressure (AP) were measured by manometers and AP was held constant by a variable resistance equivalent to the total peripheral resistance (TPR). Ventricular volume was measured by Henderson's bell cardiometer (an inverted glass bell) which is attached to the atrioventricular groove by a rubber diaphragm. Beat-by-beat volume changes were recorded on a rotating smoked drum. Mean cardiac output was measured directly by collecting blood in a measuring cylinder beyond the resistor. (After Knowlton and Starling [KS12]) (From J.R. Levick *An Introduction to Cardiovascular Physiology*, by permission.)

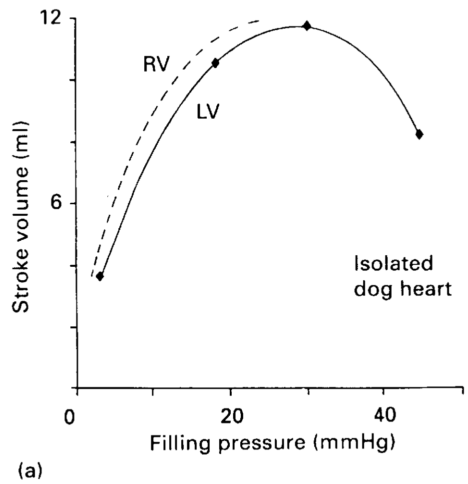

Figure 8.2: The ventricular function curve.
Effect of filling pressure on the stroke volume of an isolated dog heart pumping against a constant arterial pressure. The solid line shows Starling's data for left ventricular stroke volume (LV) and left atrial pressure. The dashed line shows how the right ventricle has a similar shaped curve but a slightly lower filling pressure. (From J. R. Levick *An Introduction to Cardiovascular Physiology*, by permission.) ([Lev91], p. 74)

The results of experiments such as Starling's are of considerable importance when modelling ventricular mechanics, because they provide data about the heart as a mechanical pump when isolated from the atria and the rest of the circulatory system. In particular, the effects due to the elasticity of the aorta are eliminated.

Some of Starling's smoked-drum recordings are reproduced by Levick [Lev91] p. 70. It is interesting to note that when the filling pressure is changed suddenly the heart adjusts to the new conditions both quickly and smoothly. In Figure 8.2, the stroke volume peaks at a pressure of about 30 mmHg and then falls. This is an abnormal region (sometimes called the **descending limb**, and there may be experimental errors due to leakage through the atrioventricular valves (see Levick [Lev91] p. 7), but it would still be of interest to reproduce this behaviour with a model, since it is a challenging test for a model. Katz [Kat65] p. 871, suggests that in some chronically ill patients the heart may operate upon this 'descending limb'.

For discussions of Starling's Law see

- *The Physiological Basis of Starling's Law of the Heart* by the Ciba Foundation Staff [Sym74]
- *Starling's Law of the Heart Revisited* by Keurs and Noble [tKN88]

8.2 In Vivo Stress Measurements

In the past, various devices have been implanted in the heart wall and used to measure the stress, but it is difficult to allow for the inevitable effects of the intrusion. Lunkenheimer, Lunkenheimer and Torrent Guasp [LLG85] pages 106-129, summarize a variety of such methods. The stress vector has six components, and it is difficult to measure all of these. See also

- Huisman, 1977, [Hui77]
- Huisman, Sipkema, Westerhof and Elzinga, 1979 [HSWE79]
- Huisman, Sipkema, Westerhof and Elzinga, 1980 [HSWE80] (quoted by Hunter and Smaill [HS89] p. 129.)

(Muss ergaenzt werden - PPL fragen)

Left ventricular wall motion has been observed by Greenbaum and Gibson [GG81], who give further references.

Right ventricular wall motion in dogs has been measured by Chuong, Sacks, Templeton, Schwiep and Johnson [CST +91]

8.3 In Vivo Strain Measurements

A variety of methods has also been used to measure strains in the heart wall. Less intrusion is necessary and recent developments in cardiac imaging promise that accurate measurements of movements of the surfaces will soon be available. See

- Lunkenheimer, Lunkenheimer and Torrent Guasp [LLG85] p. 130-136
- Hunter and Smaill [HS89] pages 130-138
- Waldman [Wal91],
- McCulloch [McC86]

(Muss ergaenzt werden, PPL fragen)

8.4 Coronary Perfusion

The suggestion that changes of pressure and flow in the coronary vessels alter the passive properties of the left ventricle has been studied by many workers. Some references are given by McCulloch in his Ph.D. thesis [McC86], p. 86.

The term **coronary perfusion** refers to the introduction by normal or artificial means of additional blood in the coronary arteries[2]. This is related to the **garden hose effect**.

8.5 Intramyocardial Pressure

The extravascular forces in the myocardium are commonly referred to as the **intramyocardial pressure**.

See

- Ph.D.thesis of Rabbany [Rab91].

[2]CWC check this definition.

8.6 Other Experimental Observations

In Chapter 3 of *Circulatory Dynamics* by Wiggers [Wig52] a variety of *in vivo* experiments are described.

8.6.1 Pericardial Effusion

Wiggers [Wig52] pages 63-65 discusses the effect of changes in **pericardial effusion**, that is, ... (PPL fragen)

He quotes several papers including:

- Kuno [Kun17]
- Fineberg [Fin36]
- Wiggers [Wig34]

Wiggers [Wig52] Fig. 23, p. 64, shows graphically the effect of increasing the pericardial pressure: the minimum ventricular pressure increases, but the maximum ventricular pressure decreases. For instance, if the pericardial pressure is increased to 135 mmHg, the minimum ventricular pressure rises to 30 mmHg while the maximum ventricular pressure falls to 80 mmHg. (PPL fragen)

8.6.2 Hypervolemia

8.6.3 Oligemia

8.6.4 Arterial Hypertension

8.6.5 Aortic Coarctation

8.6.6 Aortic Stenosis

8.6.7 Pulmonary Stenosis

8.6.8 Idoventricular Rhythms

8.6.9 Coronary Occlusion - Myocardial Ischemia

8.6.10 Cardiac Valves

8.6.11 Aortic Regurgitation

8.6.12 Mitral Insufficiency

8.6.13 Mitral Stenosis

Part III

Mathematical Modelling of the Heart

Chapter 9

Introduction

The modelling of the heart has a long history. Already in the seventeenth century Borelli [Bor89] sketched the arrangement of muscle fibres on the surface of the heart and considered how these could contract.

At the end of the nineteenth century, Woods [Woo92] applied the results of Laplace to relate the pressure in a ventricle to the stress in the heart wall.
The Law of Laplace, which is derived in section 13.1 is often quoted in medical texts. One conclusion from this law which finds medical application is that if a diseased heart increases in size, its efficiency drops since R_1 and R_2 increase. For a recent discussion of the Law of Laplace see Moriarty [Mor80]. Any realistic model must of necessity be far more complicated. Fortunately, modern computer developments make it possible to present the results of such models in a readily accessible manner.

The longterm goal of mathematical modelling of the cardiovascular system is to predict features such as:

- The blood pressure and other characteristics of the blood flow.
- The stresses in, and motion of, the components of the heart.

The general approach is to set up a system of equations which describes the underlying physical and biological processes. This system of equations will include partial differential equations for stresses in the heart wall and blood velocities, but may also include ordinary differential equations and algebraic equations. This system of equations must then be considered both mathematically, to try to derive general features, and numerically, to obtain quantitative results.

There are several excellent general references. The three-volume work of Fung [Fun84], [Fun90], [Fun93], one volume of which has recently appeared as a second edition, is a broad and leisurely introduction to biomechanics. The lecture notes of Peskin [Pes75] are often quoted, and emphasize the fluid mechanical and mathematical aspects of modelling the heart, aspects which are often somewhat neglected in the literature. There has recently been a surge of activity in the modelling of the heart, in part owing to the rapid improvements in computing facilities. The proceedings *Theory of Heart* edited by Glass, Hunter and McCulloch [GHM91] is an excellent introduction to models currently being developed and used. There is also an extensive review article by Hunter and Smaill [HS89], which discusses many aspects of modelling the heart using continuum mechanics. Yin [Yin85] compares numerical results for different geometries and constitutive laws.

Antaki [Ant91] has a lengthy introductory section of 108 pages in which models of the heart are critically discussed and questions such as fibre direction (p. 36, p. 65 and p. 91) and compressibility (p. 93) are addressed with extensive references.
Other references include:

- *Part II, Ventricular and Myocardial Mechanics*, in *Cardiovascular Dynamics and Models* by Brun et al. [BCL88].
- *Cardiovascular Biomechanics* by Chandran [Cha92]
- Chapter 7, *Circulatory System Dynamics* by Noordergraaf [Noo78].
- *The Mechanics of the Circulation* by Caro, Pedley, Schroter and Seed [CPSS78]. This is an excellent introduction to crdiovascular mechanics, which tries, "wherever possible, to supply numerical data" but also "avoids mathematical formulations and presents mechanics in readily comprehensible terms."
- *The Physics of Heart and Circulation* by Strackee and Westerhof [SW93]
- *Engineering Design of the Cardiovascular System of Mammals* by Dawson [Daw91]
- Noble, *The Cardiac Cycle*, [Nob79]
- Dinnar, *Cardiovascular Fluid Dynamics*, [Din81]
- Fung, *A First Course in Continuum Mechanics*, [Fun94]
- Sagawa, Maughan, Suga and Sunagawa, *Cardiac Contraction and the Pressure-Volume Relationship* [SMSS88]
- Sideman and Beyar *Analysis and Simulation of the Cardiac System-Ischemia* [SB89],
- Ghista, Van Vollenhoven, Yang, Reul and Bleifeld, *Cardiovascular Engineering. Part I: Modelling* [GVY +83]
- Hoppensteadt and Peskin, *Mathematics in Medicine and the Life Sciences* [HP92]

The first step in the modelling process is to decide which variables, parameters and processes significantly influence the problem. This decision is partly heuristic and is strongly influenced by the need to reduce the complexity to manageable proportions. In modelling the mechanics of the heart, two typical assumptions are:

1. To assume a simplified geometry,
2. To assume that heat flow in the heart walls does not influence the mechanics of the heart.

Once the ‘significant variables’ have been chosen, it is necessary to formulate the **field equations** governing these variables. There are two types of equations. Firstly, there are the equations which represent general physical laws such as conservation of momentum.

Secondly, there are the **constitutive laws** which embody physical properties of the medium. In some cases these can be simple and uncontroversial, as when one can assume that heat flow is linearly proportional to the temperature gradient. In other cases, the formulation of suitable constitutive laws is difficult. This is the case in ventricular mechanics, where the relationship between stress and strain in the heart walls is extremely complex. One frequent cause of difficulty is that models nearly always use only one geometric scale; the constitutive laws must reflect processes at smaller scales. The dilemma facing the modellist is illustrated by the Law of Laplace 13.10. This law is a useful simplification, but is based on a great number of assumptions.

The first models of the heart's wall were developed about thirty years ago and used linear elasticity and/or simplified geometry. While these assumptions were justified at the time, the myocardium is certainly nonlinear, and the deformations are so large that the assumptions of linear elasticity are not tenable. With current computing facilities it is feasible to handle nonlinear material properties.

9.1 The Field Equations

We assume that the myocardium is incompressible and satisfies the nonlinear elasticity equations:

$$\begin{aligned} -\mathbf{div}\ \sigma^{\mathbf{d}} + \nabla\ p &= 0 \quad \text{for } \mathbf{x} \text{ in } \varphi(\Omega) && (9.1)\\ det\ \mathbf{F} &= 1 && (9.2)\\ \mathbf{F} &= \nabla\varphi && (9.3) \end{aligned}$$

Here, $\mathbf{F}$ denotes the *deformation gradient tensor*, $\sigma^{\mathbf{d}}$ the *deviatoric Cauchy stress tensor*, and p the *pressure*. These equations are formulated in the deformed body $\varphi(\Omega)$. When solved, they must first be transformed back onto the undeformed body Ω.

9.2 The Constitutive Equations

There is a considerable literature on the form of the constitutive equations. Following Bovendeerd [Bov90] we assume the following constitutive equations:

$$\mathbf{S} = \mathbf{S}_p(\mathbf{E}) + \mathbf{S}_a(l_s, l_c, t) \tag{9.4}$$

where $\mathbf{S_p}$ represents the passive stress and $\mathbf{S_a}$ represents the active stress created by the activation of the muscle. The terms l_c and l_s represent the lengths of the *contractile element* and *sarcomere*, respectively.

$\mathbf{S}$ is the second Piola-Kirchhoff stress tensor which is related to the Cauchy stress tensor $\sigma^{\mathbf{d}}$ via:

$$\mathbf{S} = (\det \mathbf{F})\ \mathbf{F^{-1}}\ \sigma^{\mathbf{d}}\ \mathbf{F^{T}} \tag{9.5}$$

It is assumed that the material is hyperelastic, that is, that a function W exists, the **stored energy function**, such that

$$\mathbf{S_p} = \frac{\partial W}{\partial \mathbf{E}} \tag{9.6}$$

where

$$W(\mathbf{E}) = C\left[\exp(a_1 I_E^2 + a_2 II_E + a_3 E_{33}^2 + a_4(E_{31}^2 + E_{32}^2)) - 1\right] \tag{9.7}$$

and

$$\begin{aligned} I_E &= E_{11} + E_{22} + E_{33} \\ II_E &= {E_{12}}^2 + E_{22}^2 + E_{31}^2 - E_{11}E_{22} - E_{22}E_{33} - E_{33}E_{11} \end{aligned} \tag{9.8}$$

The active stress $\mathbf{S}_a$ is defined as

$$T_a = E_a\, T_{max}(l_s - l_c) \tag{9.9}$$

$$T_{max} = T_1\, A(l_c)\, B_r(t_s)\, B_d(t_s, l_s) \tag{9.10}$$

where the functions T_1, $A(l_c)$, $B_r(t_s)$, and $B_d(t_s, l_s)$ are explicitly defined and l_c satisfies the ordinary differential equation:

$$\frac{d\, l_c}{d\, t} = -\left(\frac{T_a - T_{max}}{T_a + T_0}\right) v_0 \qquad T_a \leq T_{max} \tag{9.11}$$

$$\frac{d\, l_c}{d\, t} = -\left(\frac{T_a - T_{max}}{T_a + T_0}\right) v_0 \exp\left(a_T\, \left(\frac{T_a}{T_0}\right)\right) \qquad T_a > T_{max} \tag{9.12}$$

It is a feature of nonlinear elasticity, that standard pure-traction, traction-displacement and pure-displacement problems may have more than one solution, and this is indeed desirable. (see Ciarlet *Mathematical Elasticity. Vol. I Three-Dimensional Elasticity* [Cia88] page 244). An example (see Gurtin [Gur81] p. 47) shows that if an incompressible isotropic homogeneous cube is loaded uniformly and the constitutive equation is of the form

$$\mathbf{T} = -\pi\mathbf{I} + \beta\mathbf{F}\,\mathbf{F}^T$$

where π is a real number and β is a given constant, then there may be up to 7 different solutions of the tension problem.

As shown by Ball (see Ciarlet [Cia88] p. 371) the displacement-traction problem has a solution if, among other conditions, the stored-energy function W is **polyconvex**, that is,

$$W(\mathbf{E}) = \hat{W}(\mathbf{x}, \mathbf{F}, \mathbf{CofF}, \det\mathbf{F})$$

where $\hat{W}$ is convex.
In the accompanying talk we will try - perhaps provocatively - to stimulate discussion on several questions:

- Which model is appropriate?
- Which numerical methods are best?
- What should be the goal of modelling?

9.3 The Goal of Modelling

We conclude with a number of questions.

- Which, if any, of the following should be included in a model?
  - Fibre direction
  - Porous flow
  - Residual stress
  - Restoring forces
  - Arteries, veins
  - Inertia
  - Epicardium, Endocardium, Pericardium
- Is it true to say that current models can predict **global** relationships such as the pressure-volume curve, reasonably well?
- Is the next goal the prediction of **local** variables, particularly in diseased hearts? If so, how should one approach this task?
- Should some standard problems be developed for use in comparing software?

9.4 Diastolic Expansion

As described in the section 6.2, the ventricles are closed for a short time (about 0.08 s) during which the pressure falls (isovolumetric relaxation). Then the tricuspid and mitral valves open, and the ventricles fill with blood from the atria (about 0.5s).

The mechanisms underlying the motion of the ventricles during diastole have been the subject of conjecture for many centuries. Lunkenheimer, Lunkenheimer and Torrent Guasp [LLG85], pages 61 - 66 summarize the development of various explanations from Aristotle to the present day. Gilbert and Glantz [GG89] give a recent review, as do McCulloch and Omens [MO91], p. 92. Fung [Fun84], p. 14, quotes the Chinese book *Internal Classic* written about 300 BC, but the ideas seem very imprecise.

The variation of blood pressure with time during diastole is shown in Figure 6.1. In particular, it can be seen that initially the blood pressure continues to fall. From this, and from direct measurements, (Meesmann [Mee58] and Robinson, Factor and Sonnenblick [RFS86]) it is usually concluded that 'diastolic suction' takes place, that is, that the heart walls expand and suck blood in. While this is quite likely, it does not seem to be essential; one could imagine a system in which the pressure decrease arose because the ventricles expanded and, the pressure dropped in accordance with the Law of Laplace (see Equation 13.10 on page 67).

To explain the observations two basic mechanisms suggest themselves:

9.4.1 Ventricular Blood Pressure

In 1628 Harvey asserted that the blood pressure is the sole driving force during diastole and that the ventricle muscles play no role. While this is not completely true, the blood pressure is certainly an important factor. Towards the end of the ventricular filling phase the atria contract, thereby forcing more blood into the ventricles; this is called the 'atrial boost' and is of importance during exercise when the time for passive ventricular filling is reduced (see Levick [Lev91], page 16).

The role of the filling blood pressure seems to us to have been somewhat neglected in the literature on modelling. So far as we are aware, only Peskin and his coworkers have modelled the flow of blood in the atria and ventricles. (see [PM93b]). These results are significant, but they do not, so far as we can see, provide values for the blood pressure which can be compared with the observations in Figure 6.1.

9.4.2 Myocardial stress

Although the heart is 'relaxed' during diastole, the heart walls are not stress free. For example in (PPL fragen), Dr. James Johnson (see Philip [Phi23] p. 397) found that the freshly excised heart of a turtle continues to beat and (propels itself forward?) when placed in water - where the effect of filling pressure is absent. Three sources of stress have been considered.

9.4.2.1 Elastic Recoil

It has been known (but at times forgotten) (PPL fragen) since the work of Vieussenius in 1706 (see [Vie06]) that the relaxed heart recoils. It is possible to observe this - an empty ventricle expands after being compressed manually. (see Lunkenheimer, Lunkenheimer and Torrent Guasp [LLG85] p. 63.) In the past, various mechanical models have been proposed to explain the elastic recoil. For example, in Figure 9.1 a mechanical model for elastic recoil due to Hort [Hor57][1] shown. Hort suggests that the intramyocardial tissue between the myocytes is twisted during systole and then recoils during diastole.
Recently, scanning electron microscopes have been used to reveal the detailed structure of the connective tissue in the myocardium. However, this has not yet led to an acceptable constitutive law (see Hunter and Smaill [HS89], p. 119).

Hort [Hor57] considers the question of reorientation of the fibres during systole and diastole. Thus (Hort [Hor57] p. 722): "Aus der Uebereinstimmung des Faserverlaufes in kontrahierenden und dilatierenden Herzen geht hervor, dass es in der Herzmuskulatur eine Mechanismus geben muss, der es den einzelnen Muskelschichten ermöglicht, sich verschiedenen Kammerfuellungen durch Aenderungen der Schichtdicke und Flaechengroesse anzupassen."

Hort ([Hor57] p. 727) also discusses the idea that energy is stored during systole for release during diastole, and quotes: Feneis; Fenn; Benninghoff; Rushmer, Chrystal and Wagner.

[1] See Lunkenheimer. There is a mistake here since neither Hort [Hor57] nor Hort [Hor60] has this figure.

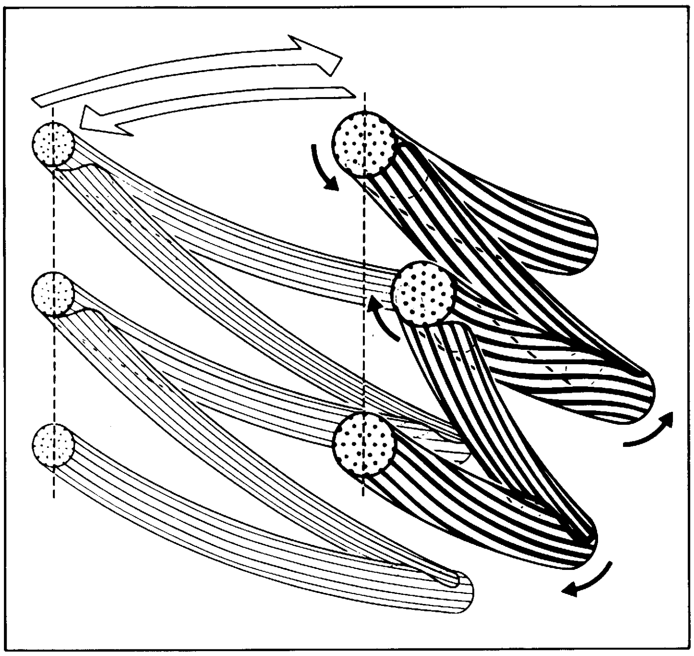

Figure 9.1: Hort's Model for elastic recoil

9.4.2.2 Residual Stress

Fung [Fun84], p. 54, vigorously argues the case that at the end of systole the heart has a residual stress distribution (stress in the absence of external forces). This idea has been developed by Omens [Ome88] and McCulloch and Omens [MO91], p. 102. Fung points out that residual stress is well-known and used in engineering. Both Fung [Fun84], p. 59, and McCulloch and Omens [MO91], p. 106, show examples of living tissue which relaxes when cut, thus proving the existence of residual stress.

The difference between 'residial stress' and 'elastic recoil' appears to be as follows. Residual stress can arise in statically indeterminate elastic bodies, even when the constitutive equations are linear. 'Elastic recoil' presupposes that while the myocyte fibres are contracting during systole, other components are being compressed; in other words, elastic recoil presupposes a nonlinear mechanism in which different elastic components act during systole and diastole. (CWC: check)

Residual stress may play a role in several ways:

1. If the constitutive laws are nonlinear, then the relationship between incremental stress and incremental strain depends upon the total stress and is thus affected by any residual stress.

2. If residual stress is included then the total stress distribution is found to be smaller, as would be expected in an organ which has developed both through growth and through evolution. The Ph.D.thesis of Omens (see [Ome88]) contains a very clear description of residual stress and its measurement.

3. Residual stress may affect the coronary blood flow and hence influence the onset of **ischaemia** (reduced blood supply) (see [Ome88] page 131).

4. The relationship between residual stress and **cardiac hypertrophy** (enlargement of the myocytes due to an increased workload) is investigated by Omens 1988 in Chaper V [Ome88].

Yang, Taber and Clark [YTC94] allow for residual stress in their model of the embryonic chick heart.

9.5 Ventricular Geometry

As is clear from Figure 5.4 the heart is a complicated three-dimensional object. In 1892 Woods [Woo92] modelled the left ventricle as a thin-walled sphere. Later, thin-walled ellipsoidal geometry was used by several authors. Since the left ventricular wall is of comparable thickness to the ventricular radius, thick walled models were introduced. (For references see Hunter and Smaill [HS89], p. 138).

Only comparatively recently have fully three-dimensional measurements begun to become available. The fully three-dimensional ventricular geometry of the dog heart was measured mechanically by Nielsen [Nie87] and Nielsen, Le Grice, Smaill and Hunter [NLSH91]. The present authors intend to provide similar information for the human heart in the near future. (PPL fragen)

So far as we are aware (October 1993), no workers have as yet used models with full three-dimensional geometry for the human heart.
A large number of possible factors can be considered:

- Geometry
  - Cylindrical
  - Spherical
    Woods [Woo92]
  - Ellipsoidal
    Wong and Rautaharju [WR68]
  - Prolate Spheroidal
    Mirsky [Mir69]
- Shell Theory
  - Thin
    Woods, [Woo92]
  - Thick
    Mirsky, [Mir69]
    Wong and Rautaharju, [WR68]
- Constitutive Stress-Strain-Equations
  - Linear Elasticity

The symposium *The Physiological Basis of Starling's Law of the Heart* [Sym74], page 193-290 contains interesting articles and discussions on general topics, such as the effect of geometry.

Chapter 10

Muscle Mechanics

10.1 Introduction

A **muscle** is any one of the contractile fibrous band or bundles that produce movement in animal bodies. A muscle is therefore biological tissue which can be either relaxed or active; in the latter state, force can be generated. For our purposes we are interested in **muscle mechanics**, the study of the motion of, and forces generated by muscles.

The two basic types of muscle, **striated muscle** and **smooth muscle**, differ in their organization. Striated muscle cells are organized very systematically, and, when viewed under a microscope, have a definite banded structure. Smooth muscle cells are organized much less regularly.

Striated muscles occur as:

- **skeletal muscles** which make up approximately 40 per cent of the human body, by weight. As the name suggests, the skeletal muscles are attached to the skeletal structure of the body, and are responsible for the movement of arms, legs etc.
- **cardiovascular muscles** which make up about 0.5 per cent (CWC check) of the human body by weight and are the source of the forces in the heart. Although similar in structure to skeletal muscles, they also differ in important ways.

Approximately 9 per cent of the human body is smooth muscle, which is found in the inner organs, the eye, the blood vessels, etc. There are two major types (Guyton [Guy91] p. 87):

- **Multi-unit smooth muscle** which is composed of discrete smooth muscle fibres, each of which can contract independently of the others.
- **Single-unit smooth muscle** which consists of hundreds or millions of muscle fibres that contract together as a single unit. This type of smooth muscle is also known as **synctial smooth muscle** or **visceral smooth muscle**

The above classification of muscles is very broad: within each type of muscle there are many special varieties, each one adapted to its particular role.
For our purposes, we wish to characterize the motion of, and the forces generated by, muscles, so that these can be incorporated into mathematical models. Two basic approaches can be followed:

- **The phenomological or engineering approach**, in which the elastic properties of muscles are measured in the laboratory and used to formulate constitutive laws. Under this heading we include the the classical experiments on muscles of Blix (1895) [Bli95], Hill [Hil38].
- **The molecular approach.** With the development of high-powered electron microscopes and sophisticated chemical methods it has become possible to elucidate the structure and mechanisms of individual muscle cells, to the extent that one can, for example, say that a particular type of cell has about 187 myosin heads per μm which form crosslinks with the adjacent actin molecules.

Of the two appproaches mentioned above, the molecular approach is clearly the more fundamental. However, for the forseeable future, this approach cannot, by itself, provide all the information needed by the modeller:

1. The molecular approach has concentrated on the contractile units, the **sarcomeres**. But one must also take account of the elastic properties of other components of the muscle.
2. Even when the mechanism of the sarcomere is fully understood, this is not the end of the story, because the activation is caused by calcium ions, whose density must be modelled.

On the other hand, the phenomological approach is also not without difficulties:

1. The preparation and testing of specimens is difficult.
2. The stress-strain response is highly nonlinear. It is not clear how the strain energy function should be chosen. In at least one case (Smaill and Hunter [SH91] p. 21) the best-fit material parameters have physically unacceptable values.

The modeller should therefore combine the information from both the phenomological and the molecular approach. The situation is well-summarized by the following quotation from Smaill and Hunter 1991 [SH91] pages 25-26:

> We have already stated that any realistic constitutive formulation for passive myocardium must incorporate microstructural information. It is worth reiterating that an appropriate constitutive law should be compact, with unique material parameters. We have demonstrated that the last of these requirements cannot easily be met by phenomenological constitutive formulations that use highly nonlinear functions. However, it is unlikely that the first requirement could be satisfied by a constitutive law based completely on cardiac mircostructure. Thus, a constitutive formulation that incorporates the most advantageous features of both phenomenologically and microstructure-based approaches appears the best option at present.

In the reminder of this chapter we first summarize these two approaches and then conclude with some typical models of cardiac muscle.
Some general references include:

- R. McNeill Alexander, *Elastic Mechanisms in Animal Movement* [Ale88].
- A. V. Hill *First and Last Experiments in Muscle Mechanics*, [Hil70]
- Z. He, *Mathematical models in muscle by periodic stimuli* [He94]
- Thomas A. McMahon *Muscles, Reflexes, and Locomotion* [McM84]
- Robert M. Miura, *Lectures on Mathematics in the Life Science, Vol. 16, Some Mathematical Questions in Biology: Muscle Physiology* [Miu86].
- ter Keurs and Noble, *Starling's Law of the Heart Revisited* [tKN88]
- Hoppensteadt and Peskin, *Mathematics in Medicine and the Life Sciences* [HP92]
- Daniel J. Schneck, *Mechanics of Muscle* [Sch92] (an engineering approach)
- Yuan-cheng Fung, *Biomechanics: Mechanical Properties of Living Tissues* [Fun93], Chapter 9 (Skeletal Muscle), Chapter 10 (Heart Muscle) and Chapter 11 (Smooth Muscle).

The book of McMahon *Muscles, Reflexes, and Locomotion* [McM84] is an excellent introduction.

10.2 The Phenomological Approach

Muscle, in particular cardiac muscle, is a biological material which can be either relaxed or passive (during diastole) or active (during systole). It is usual to consider these states separately.
Early in this century the term **new elastic body** was coined to convey the idea that excitation of muscle instantly changes its elastic state from a compliant level into a stiff level. The excited muscle thus becomes a new elastic body.
Hill (1911, 1913) and other physiologists likened contracted skeletal muscle to a cocked spring, speculating that the amount of energy released by contraction depends only on the preloaded length; this was disproved by the discovery of the **Fenn effect** by Fenn [Fen23] (see Sagawa, Maughan, Suga and Sunagawa, [SMSS88] p. 186.)

10.3 The Fenn effect

The **Fenn effect** was first observed by O. Fenn in 1923: in a skeletal muscle isotonically contracting at a constant preload, the total heat generation plus external work significantly increased and then decreased as the extent of shortening was increased by decreasing the afterload force from isometric force to nearly zero. (Sagawa, Maughan, Suga and Sunagawa [SMSS88] p. 187). See Rall [Ral82].

10.4 Feng effect

Concerning the **Feng effect** see:

- Ciba Foundation *The Physiological Basis of Starling's Law of the Heart* [Sym74] p. 238
- Feng *The effect of length on the resting metabolism of muscle*, [Fen32]

10.5 Classic Results

For the purposes of biomechanics it is necessary to determine the force generated by a muscle. It turns out that in cardiac muscle, these is a complicated relationship between the force developed, the length of the muscle, the velocity of shortening, and the time (see e.g. Brutsaert [Bru74]).

To a growing extent, this relationship can be explained at the molecular level, as briefly discussed in Section 10.6. Here we describe some classical results.
)
Some brief remarks on early work on muscle mechanics, with especial reference to cardiac muscle are given by Guz [Guz74] and Jewell [Jew74]. It was shown by Schwann in 1835 that muscles contract more forcibly when stretched. A famous paper by Blix in 1895 [Bli95] studied the relation between length and tension in muscle. Early experimental work on the heart is due to Roy (1879), Martin (1881), Howell and Donaldson (1884), and Roy and Adani (1892)[1]). The most widely quoted papers are those of Otto Frank [Fra95] on the frog heart and Patterson, Piper and Starling [PPS14]; the latter paper presenting work performed at University College, (London) and the University of Berlin. It is interesting that these workers begin their papers by quoting work on isolated muscles and using this as a guideline for their own work on the heart.

A typical experimental setup for a single muscle is shown in Fig. 10.1. The muscle is first fixed at one end and then stretched using a small weight w, the **pre-load**. The muscle is then stimulated electrically. As it shortens, it picks up a second weight W, the **afterload**. Some basic modes of testing are:

- **isotonic**. Here, the muscle is allowed to shorten (the bearing afterload W). The force is thus known. The length of the muscle and speed of shortening are measured. (See (ii) and (iii) in Fig. 10.1).
- **isometric loading**. Here, the muscle length is kept fixed. The tension is measured. (See (i) in Fig. 10.1)

As can be seen from Fig. 10.1 an increase in the initial length of the muscle leads to an increase of tension, velocity, and shortening. Several comments are due here:

- A very plausible explanation of the ...
- In Fig. 10.1 the dependency on time is not shown.
- In Fig. 10.1 the length-tension curve only goes up to the $mm = 4$ (ordinate?)
- Models?

[1]We have not consulted these early papers

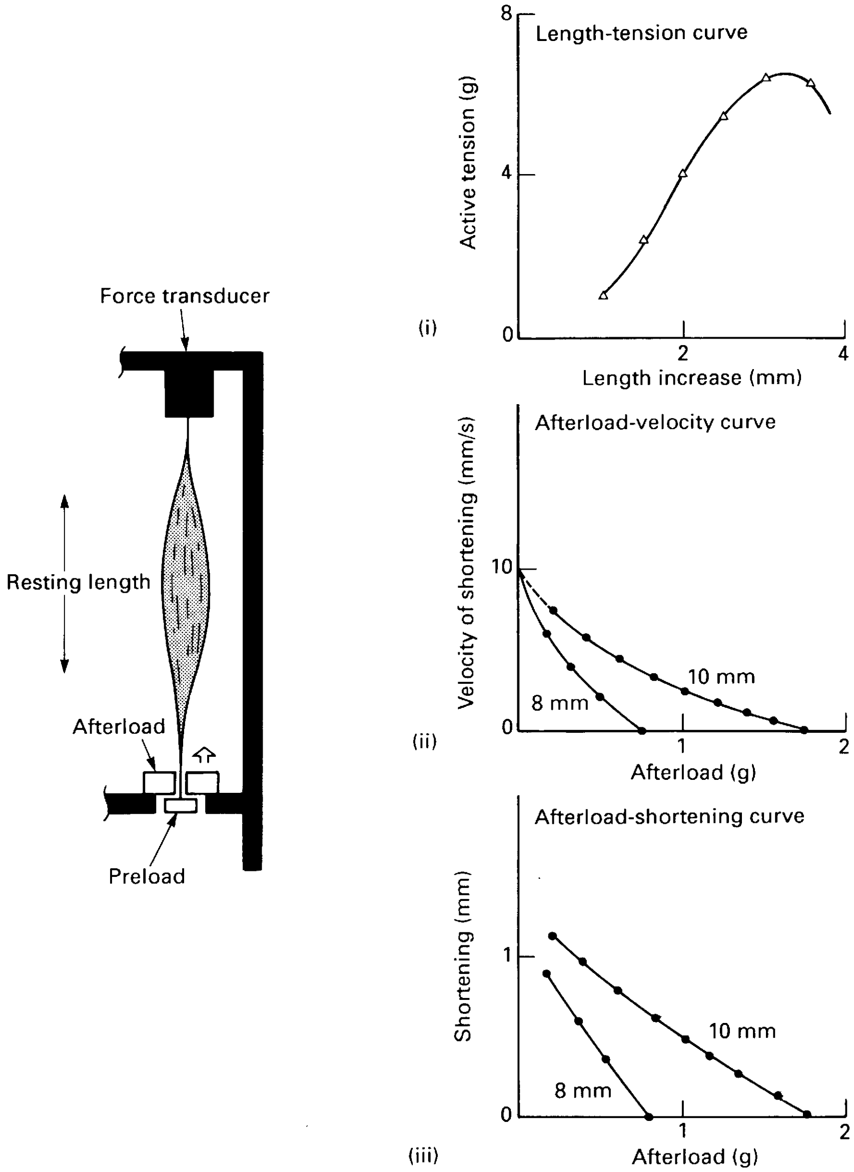

Figure 10.1: Contractile behaviour of isolated myocardial muscle.
Left: Simplified diagram of arrangement used to study the contraction of isolated cat papillary muscle. The arrangement shown allows isotonic contraction to occur, the weight labelled "afterload" being picked up as soon as shortening begins. The weight labelled "preload" sets the resting length. If the preload is clamped in place, contraction becomes isometric. Right: Three fundamental relations: (i) shows isometric contraction at increasing lengths, (ii) and (iii) show isotonic contractions beginning from two different resting lengths (8 mm and 10 mm). Contractile force, velocity and shortening are all increased by stretching the relaxed muscle. (See Sonnenblick [Son62]). (From J.R. Levick, *An Introduction to Cardiovascular Physiology*, by permission.)

10.6 Molecular Muscle Mechanics

In 1954 Hugh Huxley and J. Hanson [HH54] and Huxley and Niedergerke [HN54] independently proposed a model on the molecular level for...

10.7 Ventricular Interaction

So far as we are aware (February 1995) no workers have as yet modelled **ventricular interaction**. The influence of the right ventricle is discussed by McCulloch and Omens [MO91], p. 93 - 94, and Antaki [Ant91], p. 25., who give further references. Since the two ventricles share muscle fibres and adjoin one another, one must expect a significant iteraction (see [SG86]and [SGL89]). Influence of epicardium etc. - see Hunter and Smaill [SH91] p. 19 and Fung [Fun93] p, 459. and Humphrey and Yin [HY88].

10.7.1 Passive Myocardium

Passive myocardium is an inert biological material which can, *in principle*, be tested in the laboratory, so that, *in principle*, constitutive laws can be experimentally determined. There are, however, many difficulties some of which are mentioned below.

10.7.2 Experimental Methods and Results

General references on the testing of biological materials will be found in Appendix H.2. We are not concerned here with hte specific techniques, but do wish to draw attention to some of the difficulties (see also Smaill and Hunter [SH91] p. 14-25):

1. Soft biological material can be damaged on excision. It is also necessary to a buffer solution and to keep the specimen in control the temperature. (See also Fung [Fun93] p. 432)

2. The myocardium is three-dimensional, but so far to our knowledge as of April 1995, only one-dimensional (uni-axial) and two-dimensional (bi-axial) tests have been carried out;

3. Almost all tests have been carried out on papillary muscle and trabeculae carnae[2] But the myocardium is far from being homogenous as observed, for example, by Feneis [Fen44] and shown in recent electron micrographs (see e.g. Figures 1.1 A to 1.1 D in Smaill and Hunter [SH91]

The elastic properties of the passive myocardium have been measured by several workers, including:. Pinto and Fung [PF73]; terKeurs, Rijnsburger, van Heuningen, and Nagelsmit [tKRvHN80]; Yin, Strumpf, Chew and Zeger [YSCZ87]. In Figure 10.2 we reproduce the representative results of Smaill and Hunter [SH91].
Figure 10.2 illustrates several points:

1. The stress-strain curves are monotone and convex.

[2] We remind the reader that the papillary muscles bulge from the endocardium and are attached to the chordae tendineae (see Figure 5.4 Cross-sections). The **trabeculae carnae** also bulge from the inner wall, but are not as conspicuous as the papillary muscles.

Figure 10.2: Stress-extension relations for left ventricular midwall specimen during 1st, 9th and 10th cycles of equibiaxial loading. Cycle period 30 sec. and specimen thickness 1.83 mm. The solid lines indicate the order of loading in the cross-fiber direction. for clarity these are omitted in the fiber direction.

2. There is a point beyond which the material does not stretch.

3. For the same strains the stress in the fibre direction is far higher than that in the cross-fibre direction.

4. The (crude) estimates of Young's modulus E are quite low. For the fibre direction, an extension ratio of 1.2 corresponds to a stress of 5 kPa. That is,

$$E \approx \frac{\text{stress}}{\text{strain}} = \frac{5kPa}{(0.2/1.0)} = 25kPa$$

For comparison[3]:

Material	Young's modulus E
Steel	
stainless steel	201 GPa
Tendon	
wet tendon	1.5 GPa

Thus,wet tendon is about

$$\frac{1.5 \quad 10^9}{25 \quad 10^3} \approx 6.7 \quad 10^4$$

times stiffer than the passive myocardium. The great discrepancy between tendons and muscles is simply "explained": a tendon is a relatively simple material which must only transmit tension. In the context of the heart, one sees that relatively thin chordae tendineae[4] suffice to transmit the forces developed by the papillary muscles.

10.7.2.1 Constitutive Equations

Given experimental data such as that in Figure 10.2 one must construct corresponding constitutive equations which express the stress as a function of the strain (and possibly other properties).

10.7.2.2 Strain-Energy Functions

Although the stress is known to be dependent on time (relaxation), past history (a la Bowditch), and frequency of stimulation, (see section ?), the majority of workers have

[3]See: Kaye and Laby, *Physical and Chemical Constants* [KL86] and Bennet, Ker, Dimery and Alexander *Regional deformation and contractile function in canine right ventricular free wall* [BKDA86]. Dry tendon has substantically different properties (see Vincent *Structural Biomaterials* [Vin90]).

[4]We are not aware of measurements of the elastic properties of the chordae tendineae, and are assuming that they are mimilar to those of other tendons.

chosen to formulate the constitutive equations using a strain-energy function. That is,

$$\mathbf{S} = \frac{\partial W(\mathbf{E})}{\partial \mathbf{E}}$$

where S is a stress tensor, E is a strain tensor and W is a strain-energy function[5]

10.7.2.3 Bovendeerd

Bovendeerd [Bov90] page 39 suggested the following strain-energy function:

$$\mathbf{S} = \frac{\partial W(\mathbf{E})}{\partial E}$$

where $\mathbf{S}$ is the second Piola-Kirchhoff stress tensor, based on the deviatoric Cauchy stress tensor, and $\mathbf{E}$ is the Green-Lagrange strain tensor.
The myocardial tissue is assumed to be transversely isotropic with respect to the fibre direction. With respect to an orthonormal coordinate system with direction $\mathbf{e}_3$ parallel to the fibre direction, and cross-fibre directions $\mathbf{e}_1$ and $\mathbf{e}_2$, the strain tensor $\mathbf{E}$ is written as the strain matrix $\underline{E}$ with components E_{ij}. The strain-energy function $W\underline{E}$ for a material that is transversely isotropic with respect to e_3, can be expressed as a single valued function of the following set of scalars (Malvern [Mal69], Green and Adkins [GA70], page ?):

$$\begin{array}{lcl}
I_E & = & E_{11} + E_{22} + E_{33} \\
II_E & = & {E_{12}}^2 + E_{22}^2 + E_{31}^2 - E_{11}E_{22} - E_{22}E_{33} - E_{33}E_{11} \\
III_E & = & \det\left(\underline{E}\right) \\
I'_E & = & E_{33} \\
II'_E & = & E_{31}^2 E_{32}^2
\end{array}$$

The functional form of $W(\underline{E})$ is chosen so that (1) W is zero in the unstrained state, (2) stress is an exponential function of strain and (3) no stresses are predicted in the undeformed situation. The following strain-energy function is consistent with these conditions:

$$W(\underline{E}) = C\left[\exp(a_1 I_E^2 + a_2 II_E + a_3 E_{33}^2 + a_4(E_{31}^2 + E_{32}^2)) - 1\right]$$

Because of the additional condition of incompressibility of the cardiac tissue the scalar III_E is left out of this strain-energy function. The values of the material parameters are estimated from fitting this function to experimentally obtained data. Firstly, according to the experimental findings of Yin, Strumpf, Chew and Zeger [YSCZ87], the ratio of second Piola-Kirchhoff fibre to cross-fibre stress is required to equal 2 under conditions of equal fibre and cross-fibre stretch. This condition is satisfied by setting:

$$a_1 = 2a_2 = a_3$$

Secondly, it is required that the uniaxial stress-strain data obtained by ter Keurs, Rijnsberger, van Heuningen and Nagelsmit [tKRvHN80] are described reasonably well. Thirdly, a_4 is set at zero, because of the lack of experimental data on the mechanical behaviour of cardiac tissue under conditions of shear stress. The resulting parameter values are listed in table 10.7.2.3.

[5]See Appendix G.4

parameter	C	a_1	a_2	a_3	a_4
value	0.7	5.0	10.0	5.0	0.0
unit	kPa	-	-	-	-

10.7.2.4 Smaill and Hunter

Smaill and Hunter [SH91], p. 20, adapt a suggestion of Fung and use the following strain-energy function for passive myocardium:

$$W = C_1(\mathrm{e}^{Q_1} - 1) + C_2(\mathrm{e}^{Q_2 - 1})$$

where

$$Q_1 = C_2 E_{11}^2 + C_3(E_{12}^2 + E_{13}^2$$

and

$$Q_2 = C_5(E_{22} + E_{33})^2 + C_6(E_{22}E_{33} - E_{23}^2$$

It is hereby assumed that the material is transversely isotropic: direction 1 is aligned with the fibre axis. The stress is given by

$$\sigma_{ij} = \frac{1}{2}\left(\frac{\partial W}{\partial E_{ij}} + \frac{\partial W}{\partial E_{ji}}\right) - p\delta_{ij}$$

where p is the hydrostatic pressure. The best-fit parameters for four left ventricular mid-wall specimens are given in Table 10.1 [6]

	C_1	C_2	C_3	C_4	C_5	C_6
	0.0532	24.13	0.00	0.0078	2.404	-5.06
	0.0175	48.02	0.00	0.99×10^{-3}	0.041	-12.98
	0.0607	159.3	0.00	0.0976	6.492	-43.95
	0.0803	23.53	0.00	0.4587	1.429	-1.09
mean	0.0529	178.73	0.00	0.1413	2.5916	-15.77
σ_{n-1}	0.0262	64.17	0.00	0.2162	2.7750	19.43

Table 10.1: Best-fit material parameters for left ventricular midwall specimens.

10.8 Inverse Methods for Parameter Estimation

Given the difficulty of direct measurement, some authors have constructed models and then estimated the parameters so as to fit the observations:

- Guccione, McCulloch and Waldman [GMW91]
- Sideman

[6] where we have corrected apparent errors in the mean and standard deviation for C_2.

Chapter 11

Cardiac Mechanics

11.1 Introduction

The myocardium is a complex structure containing (see Robinson and Kinne [RK90], Smaill and Hunter [HS89], Horowitz [Hor91], and Lunkenheimer et al. [LRC +94b].

- **myocytes** The **myocyte** is the heart's muscle cell. In shape it resembles an ellipsoidal cylinder with a diameter of 10 - 20 μm and length of 50 - 100 μm. The cell is branched and is attached to adjacent cells in an end-to-end fashion. Each myocyte is connected with several others to form a three-dimensional network of cells. The interface between adjacent cells is called an **intercalated disk**.

- **coronary blood vessels** Within the heart wall is a dense network of coronary blood vessels. One or more capillaries run along the boundary of each myocyte, parallel to the long axis. The coronary arteries and veins act much like rivets within the wall.

- **collagen matrix** Individual myocytes and bundles of myocytes are connected by a **collagen matrix**, which consists of a dense network of struts of collagen, type I and type III, predominate.

- **connective tissue** Additional connective tissue consists of collagen, elastin, glycoaminoglycans and glycoproteins.

- The interstitial space or **interstitium** between cells is filled by **interstitial fluid**. Since the pores are so small, the fluid moves mainly through diffusion.

The monograph edited by Robinson and Kinne [RK90] *Cardiac Myocyte-Connective Tissue Interactions in Health and Disease. Issues Biomed.* provides a very readable overview of the structure of the myocardium. Topics considered include:

1. The biochemistry of the myocardium

2. The structure of the connective tissue

3. The elastic properties of myocytes

In particular, Covell [Cov90] discusses the functional dynamics of the myocardial tissue. Robinson [Rob90] summarizes results obtained using scanning electron microscopy, the light microscopy, high voltage transmission electron microscopy, antibody localization and in situ hybridization. For our purposes, the following observations are of particular interest.

1. Robinson (see page 70) reports on significant differences on collagen content in rats (see Table 11.1).

	Young adults	Aged rats
Left ventricle	5.5 %	12.4 %
Right ventricle	7 %	19.5 %

Table 11.1: Collagen content

2. Robinson [Rob90] p. 72 summarizes the occurrence of four self-similar patterns **fractal analysis** of structures: weave, branced fibre, bilt, and heliv. These patterns occur at several levels.
3. Robinson [Rob90] p. 74 speculates on the transfer of information between myocyte and fibroblast.
4. Robinson [Rob90] p. 73 raises the question of the relative mechanical strengths of different networks.
5. Robinson [Rob90] p. 70 refers to major differences in cases of stunned myocardium or infarct.

For modelling purposes, it is necessary to set up constitutive laws which model the myocardium on a larger scale, but it is of course important to take account of the microscopie structure. We, therefore, briefly describe the components of the heart's wall in somewhat greater detail before moving on to describe possible constitutive laws.

11.2 The Components of the Heart's Wall

11.3 Experimental Stress-Strain Measurements

11.4 Constitutive Laws

There is an extensive literature on the mechanical properties of isolated cardiac muscle. The first experiments were apparently performed by Sonnenblick [Son62] who measured the behaviour of isolated myocardial cat muscle[1]. His results are shown in Figure 10.1. These results show that stretching the muscle enhances its subsequent contractile energy, in agreement with the Frank-Starling-Law. Levick [Lev91] p. 66 discusses the physiological mechanisms underlying this behaviour.

[1]This seems very late. PPL fragen

11.4.1 Hydraulic Effects

Living tissue is bathed in liquid (PPL fragen). In addition, the heart is criss-crossed by a network of blood vessels. As a result, one may regard the myocardium as a structure of fibres and blood vessels surrounded by fluid. The pressure in coronary blood vessels is called the **perfusion pressure** or **coronary perfusion pressure** (PPL fragen) and provides a mechanism to explain the expansion of the ventricles. There is an analogy with a hosepipe which straightens as it is filled with water. For this reason hydraulic effects are sometimes called the **garden hose effect**. See Lunkenheimer, Lunkenheimer and Torrent Guasp [LLG85], p. 64, for further references. See McCulloch and Omens [MO91], pages 94 - 95 and pages 101 - 103, and Hunter and Smaill [HS89], pages 137 - 138, for an evaluation of the influence of coronary perfusion.

McCulloch [McC86] reports on measurement on the effect of coronary perfusion on left ventricle mechanics. The pressure in fluid outside the myocardial fibres and blood vessels is called the **intramyocardial pressure**. See Rabbany [Rab91].

Several models take account of the fluid in the myocardium. A theory for an elastic structure filled with a fluid - think of a sponge - was first formulated by Biot [Bio41]. It was later extended to the case of finite deformations by Biot [Bio72], and was further generalized to a theory of mixtures by Bowen ([Bow76], [Bow80]). These ideas have been used (CWC: check) by Bogen (see [Bog87a] and [Bog87b]) to model the swelling of tissue. Huyghe, van Campen, Arts and Heethar [HvCAH91] use a porous medium finite element model to model the diastolic left ventricle.

Bogen [Bog87a], [Bog87b] considers strain energy descriptions of biological swelling. In the second article, multiple fluid compartment models are considered, together with their application to cardiac muscle. The models are visualized as constisting of networks of springs or elastic tubes. For the case of a single fluid with two elastic networks, a and b, Bogen [Bog87b] equ. (5) suggests the strain energy function

$$\begin{aligned}
\Phi_s &= \tfrac{\mu_a}{k_a}\lambda_s^{k_a-3}\lambda_s^{k_a-3}(\Lambda_1^{k_a}+\Lambda_2^{k_a}+\Lambda_3^{k_a}) \\
&+ \tfrac{2\mu_a}{K_a}\lambda_a^{-k/2-3}\lambda_s^{-k/2-3}(\Lambda_1^{-k_a/2}+\Lambda_2^{-k_a/2}+\Lambda_3^{-k_a/2}) \\
\Phi_s &= \tfrac{\mu_b}{k_b}\lambda_s^{k_b-3}\lambda_s^{k_b-3}(\Lambda_1^{k_b}+\Lambda_2^{k_b}+\Lambda_3^{k_b}) \\
&+ \tfrac{2\mu_b}{K_b}\lambda_b^{-k/2-3}\lambda_s^{-k/2-3}(\Lambda_1^{-k_b/2}+\Lambda_2^{-k_b/2}+\Lambda_3^{-k_b/2})
\end{aligned}$$

Bogen draws some general conclusions, but does not give specific applications to the heart. He also considers the puzzling observation by Pogatsa, Koltai and Grosz [PKG82] that the ventricular stiffness increase when myocardial water content is either increased above or reduced below the normal water content. (PPL fragen)
In the first article Bogen considers various possible laws and shows that some are impossible ([Bog87a] p. 254).

11.5 Myocytes

Concerning **myocytes** see:

- Rabbany, Funai, and Noordergraaf [RFN94]

Chapter 12

Modelling the Fibre Architecture of the Myocardium

In Part I the observations of the fibrous structure of the myocardium have been described. In this chapter, we attempt to use geometrical and topological methods to describe this structure. There seem to be several possibly useful tools:

12.1 Foliations

In various works, e.g. Krehl [Kre91], Pettigrew [Pet64], Streeter [Str79] Figure 53, Torrent Guasp [Gua72], the heart and in particular the left ventricle, is depicted as consisting of nested shells.

Mathematically, this corresponds to a **two-dimensional foliation**[1] of the heart or left ventricle, the individual shells being called **leaves**.

12.2 Topology

The analysis of three-dimensional structures is treated by Winfree [Win87] pages 225, Chapter 8 and 9 and pages 256-260. References given include:

- Bouligand [Bou72]
- Gordon and Winfree [GW87]
- Thursten and Weeks [TW84]
- Delbruck [Del62]
- Fuller [Ful71]
- Fuller [Ful78]

[1] See Appendix Foliations

- Winfree [Win85]
- Winfree and Strogatz [WS84]

12.3 Geodesics

Concerning **geodesics** see:

- Thompson *On Growth and Form* [Tho61] p. 745.

Chapter 13

Models with Spherical Geometry

13.1 The Law of Laplace

We begin the Law of Laplace, which was first stated by Woods [Woo92], can be derived with the help of Figure 13.1 (see Fung [Fun84] page 23).

Figure 13.1: The Law of Laplace

Consider a thick-walled hemisperical shell with inner radius r, thickness t, and outer radius $r + t$. Let the pressure acting on the outside be p_0 (pressure from pericardium). Consider the equilibrium of forces in the vertical direction. One obtains

$$< \sigma_\theta > . \, (\pi(r+t)^2 - \pi r^2) = p_i \pi r^2 - p_0 \pi (r+t)^2 \tag{13.1}$$

where $< \sigma_\theta >$ is the average circumferential wall stress.

Hence,

$$< \sigma_\theta >= \frac{p_i r^2 - p_0(r+t)^2}{r^2-(r+t)^2}. \tag{13.2}$$

When $p_0 = 0$ and t is small, we obtain in the limit as $t \to 0$, the equation

$$\sigma_\theta = \frac{p_i r}{2t} \tag{13.3}$$

for the stress σ_θ, which can also be written in the form

$$p_i = \frac{2t\sigma_\theta}{r} \tag{13.4}$$

For applications see Woods [Woo92], Moriartiy [Mor80] and Grossman [Gro80]
The equation 13.4 is the simplest form of a relation known as the **Law of Laplace**. It shows that the pressure created by a thin shell is proportional to the thickness t, proportional to the circumferential stress σ_θ, and inversely proportional to the radius r.

Laplace's Law is often used to compute the stress in the left ventricular wall; in fact, the curvature of the left ventricular is often negative! (see Greenbaum and Gibson [GG81])

If we wish to obtain expressions for the pressure p_i the circumferential stress σ_θ, and the thickness t as functions of r, it is necessary to derive additional equations.

Let the original shell have radius R and thickness T. Since the volume of the heart wall remains nearly constant, we have

$$\frac{4\pi}{3}[(R+T)^3 - R^3] = \frac{4\pi}{3}[(r+t)^3 - r^3],$$

so that

$$(r+t)^3 - r^3 = (R+T)^3 - R^3. \tag{13.5}$$

For small t we obtain

$$r^2 t = R^2 T$$

Next we need a relationship between the wall stretch λ_θ and the wall stress σ_θ. We use the relationship proposed by Valanis and Landel [VL67],

$$\sigma_\theta = C\lambda^\theta + H$$

where C and H are constants, and fitting this to the data for rat papillary muscle, it is found (Moriarty [Mor80] page 325) [1] that

$$\sigma_\theta = C(\lambda^\alpha - \lambda^{-2\alpha}) \tag{13.6}$$

with

$$\alpha = 18$$

[1] The value for C seems to differ greatly from values given by Fung *Mechanical Properties* pages 271 and 437.

and

$$
\begin{aligned}
C &= 2.37 g/cm^2 && (13.7)\\
&= 2.37 \times 98.06 \quad Pa && (13.8)\\
&= 232 \quad Pa && (13.9)
\end{aligned}
$$

The equations 13.4, 13.5, and 13.6 comprise a system of three equations, from which σ_θ, t and p_i can be determined as functions of the radius r or, equivalently, as functions of the left ventricular volume $v = \frac{4}{3}\pi r^3$.

13.2 Spherical Models

We summarize the predictions for the pressure-volume ($P-V$) and pressure-stress ($P-S$) relations predicted by several mathematical models in which the left ventricle (LV) is modelled as a spherically symmetric sphere. We draw heavily on the article of Moriarty [Mor80]. Chapter 8 of the book of Mazumdar [Maz89] contains a short description of some models, apparently also based on the paper of Moriarty.

13.3 Analytical Work

There is clearly relatively little scope for analytical work. Nevertheless, such work is often valuable, since it provides guidelines and viewpoints in a way that numerical results cannot do.

The sort of results which one might hope to establish analytically would be of the form:

- To show that for a certain class of models particular a e.g. variable, the circumferential wall stress, increases monotonically from epicardium to endocardium.
- To show that for a certain class of models a particular variable, e. g. the wall stress, is maximized for axially symmetric geometry.

In greater generality, the Law of Laplace can be stated as follows:
Let P denote the normal force exerted by the heart wall on the blood in the ventricle. Let S be the tangential stress in the wall, w the thickness of the wall, and R_1 and R_2 the principal curvatures. Then the **Law of Laplace** [2] states that

$$P = Sw \cdot \left(\frac{1}{R_1} + \frac{1}{R_2}\right) \tag{13.10}$$

Woods [Woo92] verified this formula experimentally. Authors using the formula to compute the stress in the heart's wall include:

- Sandler and Dodge [SD63] (for patients with heart disease, using both R_1 and R_2).

[2] Sometimes called the Laplace-Young equation since it was discovered independently by the Marquis de Laplace and Thomas Young.

13.4 Comparison of Models

References in which various models are compared include:

- Huisman, Sipkema, Westerhof and Elzinga, 1980, [HSWE80]

Sandler and Dodge [SD63] use Laplace's Law to estimate wall stress. They quote references to show that the difference between thin-walled and thick-walled models is of the order of 15% (Sandler and Dodge [SD63] p. 104)

Chapter 14

Models with Cylindrical Geometry

- Theo Arts, *A Mathematical Model of the Dynamics of the Left Ventricle and the Coronary Circulation* [Art78] model the left ventricle as a cylinder composed of 8 concentric cylindrical shells.
- Arts, Reneman and Veenstra *A model of the mechanics of the left ventricle* [ARV79]
- Toezeren, *Static analysis of the left ventricle* [Toe83]
- Feit, *Diastolic pressure-volume relations and distribution of pressure and fiber extension across the wall of a model left ventricle* [Fei79]
- Chadwick, *The myocardium as a fluid-fiber continuum: passive equilibrium configurations* [Cha81], and Chadwick, *Mechanics of the left ventricle* [Cha82]
- Caflisch, *Strukturgerechte Analyse der Myokardfaserspannungen*, [Caf87]
- Pelle, Ohayon, Oddou and Brun, *Theoretical models in mechanics of the left ventricle*, [POOB84]
- Skalak, *Approximate formulas for myocardial fiber stresses* [Ska82]
- Costa, Hunter, Wayne, Waldman, Guccione and McCulloch, *A three-dimensional finite element method for large elastic deformations of ventricular myocardium. Part II: Prolate spheroidal coordinates* [CHW +94]
- Guccione, McCulloch and Waldman [GMW91] *Passive material properties of intact ventricular myocardium determined from a cylindrical mode* construct a cylindrical model which they use to estimate material properties.

In their second paper, Arts, Veenstra and Reneman [AVR82] compare their theoretical results with experimental data for four dogs and obtain good agreement (CWC: check).

Chapter 15

Models with Spheroidal Geometry

- Beyar and Sidemean, *Spatial energy balance within a structural model of the left ventricle*, [BS86]

- Bovendeerd, Huyghe, Arts, van Campen and Reneman, *Influence of endocardial-epicardial crossover of muscle fibers on left ventricular wall mechanics*, [BHA +94] also incorporate transversal fibres.

- Bovendeerd, Arts, Huyghe, van Campen and Reneman, *Dependence of local left ventricular wall mechanics on myocardial fiber orientation: A model study*, [BAH +92]

- Beyar and Sideman, *A computer study of the left ventricular performance based on the fiber structure, sarcomere dynamics and transmural electrical propagation velocity incorporate fibres and electrical propagation*, [BS84a]

- Bovendeerd, Ph.D. thesis, *The Mechanics of the Normal and Ischemic Left Ventricle During the Cardiac Cycle: A Numerical and Experimental Analysis*, [Bov90]

- Costa, Hunter, Rogers, Guccione, Waldman and McCulloch, *A three-dimensional finite element method for large elastic deformations of ventricular myocardium. Part I: Cylindrical and Spherical Polar Coordinates*, [CHR +94] and Costa, Hunter, Wayne, Waldman, Guccione and McCulloch *A three-dimensional finite element method for large elastic deformations of ventricular myocardium. Part II: Prolate Spheroidal Coordinates.*, [CHW +94]

Chapter 16

Models with Additional Features

16.1 The Pericardium

Only a few workers have modelled the **pericardium**:

- Misra and Singh [MS85] incorporate the pericardial pressure in their model and conclude [MS85], page 65, that "the effect of the pericardial pressure which is assumed to be one-tenth of the left ventricular pressure is quite prominent."
- Humphrey and Yin [HY88] discuss the effect of the pericardium. Fung [Fun93] p. 459 also comments on the role of the pericardium.

16.2 Fibres

16.2.1 Hydrostatic Skeletons and Muscular Hydrostats

A **hydrostatic skeleton** (see Kier [Kie92]) is a fluid mechanism that provides a means by which contractile elements may be antagonized. Hydrostatic skeletons occur in a remarkable variety of organisms with examples from not only invertebrates but also vertebrates. A hydrostatic skeleton is typically considered to include a liquid-filled cavity surrounded by a muscular wall reinforced with connective tissue fibres.

A **muscular hydrostat** is a hydrostatic skeleton that consists of a tightly packed three-dimensional array of muscle fibres. Examples include the tentacles of the octopus, the tongues of many animals, and the trunk of an elephant.

There are obvious similarities between muscular hydrostats and the fibrous structure of the heart wall. Thus, Lunkenheimer and Whimster [LW93], section 4.2 use the structure of the uvular muscle and the tongue to illustrate possible mechanisms during diastolic unfolding.

There are some interesting mathematical problems in this area. For example, Kier [Kie92] page 207, considers helical muscles wrapped around a circular cylinder and claims that there is a torsional force with no longitudinal force only if the angle of the muscles is exactly 54°44!

See also:

- Johnson and Kier [JK93]
- Alexander in Chapter 5 *Fibre-wound animals* of *Elastic Mechanisms in Animal Movement* [Ale88]

See also Alexander [Ale87], Clark and Cowey [CC58] and Harris and Crofton [HC57]. For a brief useful account of various biological structures, see Chapters 9 and 10 of *Life's Devices*, by Vogel [Vog88].

16.2.2 Fluid-Fibre Models

Several authers have considered **fluid-fibre models**, in which the myocardium is treated as a fluid with an embedded fibre matrix. Such models thus ignore the elasticity of the myocardium, but, because of their simpicitiy can sometimes be solved analytically. See:

- Caflisch, 1987, [Caf87]
- Fung, 1984, [Fun84] p. 70
- Skalak, 1982, [Ska82]
- Chadwick, 1981 und 1982, [Cha81], [Cha82]

For example, Chadwick [Cha82] considers the fibre direction $\boldsymbol{\tau}$ as a given function of $\mathbf{x}$. The Cauchy stress tensor $\boldsymbol{\sigma}$takes the form

$$\sigma_{ij} = -p\ \delta_{ij} + T\ \tau_i\ \tau_j \tag{16.1}$$

where T is the magnitude of the fibre tension. The equation of equilibrium is:

$$\mathbf{div}\ \boldsymbol{\sigma} = 0 \tag{16.2}$$

If $\mathbf{u}(\mathbf{x})$ denotes the displacement,

$$\varphi(\mathbf{x}) = \mathbf{x} + \mathbf{u}(\mathbf{x}),$$

then, since the material is assumed to be incompressible,

$$\text{div } \mathbf{u} = 0. \tag{16.3}$$

Finally, the equations are completed by an equation which expresses the relationship between the fibre tension T, the time t, and the fibre deformation ϵ,

$$\begin{aligned} \epsilon &:= [(\boldsymbol{\tau} \cdot \nabla)\mathbf{u}] \cdot \boldsymbol{\tau} \\ T &= [(1 - \beta(t))E + \beta(t)E^*]\epsilon + \beta(t)T_0 \end{aligned} \tag{16.4}$$

where the constants E and E^* represent the passive and active elasticity moduli, T_0 is a constant, and $\beta(t)$ is a given function of time reflecting the transition between active and passive states.

16.3 Systolic Contraction

As described in the section 6.2, the ventricles are closed for a short time (about 0.05 s) during which the pressure rises and the ventricles deform (isovolumetric contraction). This is followed by a period lasting about 0.3 s during which the blood is ejected.

The contraction of the heart muscles is iniated by electrical impulses the propagation of which is discussed in Section 21.1. The literature on systolic contraction (involving 'active' muscles) seems to be much smaller than the literature on diastolic expansion (involving 'passive' muscles).

Relatively early calculations were performed by Ghista and Hamid [GH77] and Chadwick [Cha82] while Waldman [Wal83] discussed the coupling of the heart to the circulatory system.

Beyar and Sideman [BS84b] consider systolic contraction under the following hypotheses:

1. The LV (left ventricle) is a thick-walled prolate spheroid with time - independent shape, i.e. constant axis ratio.
2. The LV is incompressible.
3. The fibre angle acros the myocardium varies linearly from $-60°$ to $60°$ in the unstressed reference configuration, and does not change during systole.
4. The sarcomere length distribution is uniform at the reference configuration.
5. The electrical activation propagates radially from endocardium to epicardium.
6. The mechanical activation function is half a sinusoidal wave.
7. The stress-strain relationship for the muscle fibre is linear.
8. The active stress-sarcomere length relationship is linear.
9. The passive stress-sarcomere length relationship is exponential.
10. Transfibre stresses are not included.
11. The afterload (aortic pressure) is modelled.

Beyar and Sideman [BS84b] give a flow chart for their Fortran program, which calculated the various variables through out systole. Although their work contains a great many approximations, these are clearly stated. The parameters used in the model are given and compared with reported values. The sensitivity with respect to various parameters is also studied.

16.3.1 Fibres

The following include fibres in their model:

- Bovendeerd, [Bov90]

- Yang, Taber and Clark, [YTC94] (modelling the chick heart)
- Beyar and Sideman, [BS84b]

Authors using more complex models and allowing for fibres parallel to the surface include:

- Costa, Hunter, Rogers, Guccione, Waldman and McCulloch, *A three-dimensional finite element method for large elastic deformations of ventricular myocardium. Part I: Cylindrical and spherical polar coordinates*, [CHR +94]
- Costa, Hunter, Wayne, Waldman, Guccione and McCulloch, *A three-dimensional finite element method for large elastic deformations of ventricular myocardium. Part II: Prolate spheroidal coordinates*, [CHW +94]

Recently, in his Ph.D. thesis, Bovendeerd [Bov90] used a model which allowed transmural fibres. This work has been extended. See:

- Bovendeerd, Huyghe, Arts, van Campen and Reneman, *Influence of endocardial-epicardial crossover of muscle fibers on left ventricular wall mechanics* [BHA +94]
- Bovendeerd, Arts, Huyghe, van Campen and Reneman *Dependence of local left ventricular wall mechanics on myocardial fiber orientation: A model study* [BAH +92]

Yang, Taber and Clark [YTC94] page 215 briefly mention observations on fibre orientation in embryonic chick hearts which they incorporate into their model (as torsion).

16.4 Treatment of the Band Structure

The incorporation of a band-like structure into a model of the heart requires the following:

- A more detailed description of the orientation of the fibres than is currently available. In particular, the band-like structure implies the existence of a twisted structure which is more complicated than the usual 'Streeter structure'. This in turn will require the use of much greater number of finite elements.
- The band-like structure explicitly involves the right ventricle, which should therefore be included in the model.

16.5 Poroelastic Models

Several models take account of the fluid in the myocardium and use **poroelastic models**. A theory for an elastic structure filled with a fluid - think of a sponge - was first formulated by Biot [Bio41]. It was later extended to the case of finite deformations by Biot [Bio72], and was further generalized to a theory of mixtures by Bowen ([Bow76], [Bow80]). These ideas have been used (CWC: check) by Bogen (see [Bog87a] and [Bog87b]) to model the swelling of tissue. Huyghe, van Campen, Arts and Heethar [HvCAH91] use a porous medium finite element model to model the diastolic left ventricle. Simon and Gaballa [SG88a] use a poroelastic finite element model to model the repsonse of large arteries.

See also:

- Kenyon [Ken76]

For the mathematical theory of porous flow see Douglas and Hornung [DH93]. See also:

- Sorek and Sideman [SS86b]
- Sorek and Sideman [SS86a]

The thesis of Huyghe [Huy86] is extensive. The myocardium is modelled using a combination of a deformation model and a perfusion model. (CWC must look further into this). The full cardiac cycle is also modelled.
In his Ph.D. thesis, Huyghe [Huy86] models the myocardium using a porous medium finite deformation theory of blood perfused soft tissue. He then derives two special models:

- A deformation model of the beating left ventricle.
- A perfusion model of the left ventricular coronary circulation.

Numerical results are given and a complete cycle is simulated.

Yang, Taber and Clark [YTC94] model the **embryonic heart** of a chick (stage 21) using a poroelastic model. This is a reasonable model for the chick heart at this stage of development.

16.6 Other Factors

In this paper we are primarily interested in the role played by the fibre direction and have chosen to neglect a variety of other factors:

- the **pericardium** which encloses the heart and restrains the motion of the heart, particularly during certain abnormal conditions. Braunwald [Bra92] comments that whereas the pericardial space normally contains at most 50ml of fluid, this can increase to 2 litres in severe cases of pericardial effusion.
- So far, no models have directly considered the role of the **endocardium** and **epicardium** which may possibly be significant. One reference on this is: Humphrey and Yin, *Biaxial mechanical behaviour of excised epicardium*, [HY88]
- The heart wall contains a large amount of fluid in the form of **interstitial fluid and blood**. The flow of this fluid has been modelled using the equations of Biot by Huyghe and co-authors
  - The Ph.D. thesis of Huyghe, [Huy86]
  - Huyghe, Arts, van Campen and Reneman, *Porous medium finite element model of the beating left ventricle*, [HAvCR92],
  - van Campen, Huyghe, Bovendeerd and Arts, *Biomechanics of the heart muscle*, [vCHBA94].

  However,it should be noted that in this work the effects of porosity and elasticity are uncoupled.
- In the literature, it is customary to neglect the effects of **inertia**, the paper of Moskowitz [Mos80] being quoted in justification. However, the paper of Moskowitz compares solutions with and without the effects of inertia in a very simple case. In the opinion of the authors, this needs further investigation.

- **Residual stress**. See e.g. McCulloch and Omens [MO91].
- As observed by Hort, Feneis and other authors, the myocardium is not homogeneous and there are gaps which can be interpreted as 'sliding planes'. If we accept this, how can it be modelled?

Chapter 17

Numerical Models

In preceding chapters we have considered models classified according to their geometry. Here, we briefly summarize the development of numerical models. See:

- In his Ph.D. thesis McCulloch *Deformation and Stress in the Passive Heart*, [McC86] developed special finite elements.
- Yin, [Yin85]
- Costa, Hunter, Rogers, Guccione, Waldman and McCulloch, [CHR +94]
- Costa, Hunter, Wayne, Waldman, Guccione and McCulloch, [CHW +94]

Interesting recent work includes:
Authors using more complex models and allowing for fibres parallel to the surface include:

- Costa, Hunter, Rogers, Guccione, Waldman and McCulloch, *A three-dimensional finite element method for large elastic deformations of ventricular myocardium. Part I: Cylindrical and spherical polar coordinates*, [CHR +94]
- Costa, Hunter, Wayne, Waldman, Guccione and McCulloch, *A three-dimensional finite element method for large elastic deformations of ventricular myocardium. Part II: Prolate spheroidal coordinates*, [CHW +94]

Recently, in his Ph.D. thesis, Bovendeerd [Bov90] used a model which allowed transmural fibres. This work has been extended. See:

- Bovendeerd, Huyghe, Arts, van Campen and Reneman, *Influence of endocardial-epicardial crossover of muscle fibers on left ventricular wall mechanics* [BHA +94]
- Bovendeerd, Arts, Huyghe, van Campen and Reneman *Dependence of local left ventricular wall mechanics on myocardial fiber orientation: A model study* [BAH +92]

Yang, Taber and Clark [YTC94] page 215 briefly mention observations on fibre orientation in embryonic chick hearts which they incorporate into their model (as torsion).

17.1 Numerical Methods

It is proposed to initially solve the equations using finite elements, in particular using the 20-node brick element for the displacements and the 8-node brick element for the pressure. Preliminary calculations were made using the NAG Finite Element Package, but future work will probably use Diffpack.

Chapter 18

Properties of Blood

See:

- Chapter 6, Biological Fluid Dynamics, of *An Introduction to Mathematical Physiology and Biology* by Mazumdar, [Maz89]

Chapter 19

Fluid Flow in the heart

19.1 Introduction

See:

- Sugawara, Kajiya, Kitabatake and Matsuo *Blood Flow in the Heart and Large Blood Vessels*, [SKKM89]

19.2 Numerical Methods

19.2.1 The Immersed-Boundary Method

In a series of papers, Peskin and collaborators used the **immersed-boundary method** to compute two-dimensional flow in the heart. Applications included: flow patterns around heart valves ([Pes72], [Pes77]), blood flow in the heart, and the design of cardiac valves. An improved version of the method, which improves the conservation of mass, was recently described by Peskin and Printz [PP93], who give further references. Peskin and Printz [PP93] illustrate the immersed-boundary method by considering a two-dimensional model of the heart, which includes a left atrium, a left ventricle and a mitral valve. (The aortic valve is modelled as a sink). The entire left heart is modelled as though it were floating in a fluid. The boundary points are modelled using a mechanical analogue consisting of elastic, plastic and contractile components (see McQueen, Peskin and Yellin, [MPY82]). This model is clearly somewhat out-of-date. However, it also has interesting features. It is used to model the entire cardiac cycle and is run until a periodic solution is obtained. There is an interesting discussion of these results and comparisons with experiments in Sugawara, Kajiya, Kitabatake and Matsuo, [SKKM89], p. 17.
Yoganathan, Lemmon, Kim, Walker, Levine and Vesier [YLK +94] have used the immersed-boundary method to model three-dimensional early systolic motion. Fluid velocities in the outflow tract matched NMR data to within 10%. The aortic valve was not simulated physiologically. The connection of the papillary muscles to the mitral valve by the chordae tendinea was simulated.

See the related work on **immersed interface problems** of Adams *A multigrid algorithm for immersed interface problems* [Ada93].
The authors comment (page 309) on deficiencies of the immersed-boundary method. In effect, the cardiac tissue is modelled as an infinitely thin layer (page 308). "Unfortunately, one of the consequences of a thin-walled left ventricle is its tendency to become spherical at a physiologic left ventricular pressure. The other consequence is that a thin-walled structure must produce much larger stresses than a thick-walled structure to support the same pressure." To overcome these deficiencies, it was articficially assumed that "all structural points representing muscle tissue were tethered to the ventricular centerline."
See also the Ph.D. thesis of Vesier [Ves91] and Vesier and Yoganathan [VY91].
Adams [Ada93] presents a multigrid algorithm for immersed interface problems. See also LeVeque and Li [LL94].

19.2.2 Euler-Lagrangian Formulation

Recently, Peskin and McQueen [PM93b] have introduced a mixed Eulerian-Lagrangian formulation of the interaction of a viscous incompressible fluid with an elastic and possibly active [1] material. The elastic part of the system is treated using the Lagrangian equations, while the fluid part is treated using the Eulerian equations. The use of Lagrangian equations for the elastic part is based on ideas of Ebin and Saxton [ES86] [ES87].

Peskin and McQueen [PM93b] use this approach to model the three-dimensional flow of blood in the heart. The model heart contains the left and right ventricles; the left and right atria; the mitral, tricuspid, aortic and pulmonary valves; and segments of the aorta, main pulmonary artery, superior and inferior vena cavae, and the four pulmonary veins. The authors specify a stress-strain law for the muscle fibres and assume that the elastic energy density depends only on the strain in the fibre direction. To complete the model, the paths of the fibres are specified.

Several computational results are shown graphically. Coloured plates are shown in an article by Peskin and McQueen [PM93a]. Tu and Peskin [TP93] and Mayo and Peskin [MP93] discuss the stability merits of implicit versus explicit methods for computing the elastic force density from the configuration of the elastic material.

The results of Peskin and McQueen [PM93b] are impressive. Unfortunately, the geometry is only shown graphically, so that the reader cannot reproduce the results.

Taylor, Okino and Yamaguchi [TOY94] describe the use of a commercial software package SCRYU (using a finite volume integration of the three-dimensional Navier-Stokes equations based on the SIMPLE algorithm with a body-fitted (BFC) coordinate system). The time variation of the left ventricle wall was assumed as given.

A spherical model was used, but fully three-dimensional computations are planned.

[1] That is, the material can exert force, as for example when muscle fibres contract.

19.3 Vortex and whirl flows

The flow of blood into the left ventricle during diastole is closely connected with the motion of the mitral valve. Blood entering the ventricle through the mitral valve is directed towards the apex and may be likened to a **jet**. It is deflected by the apex and takes on the form of a **vortex**, see Fig 19.1. This vortex-like motion plays an important role in the closure of the mitral valve (see Section 20.2). In the case of a dilated left ventricle, a **whirl flow** can be observed, see Figure 19.2.

(Figure Blood flow from Strackee and Westerhof *The Physics of Heart and Circulation*, Figure 17.1, p. 339, [SW93]

Figure 19.1: Blood flow (a)

Figure 19.2: Blood flow (b)

See the Bot's Ph.D. thesis, *Mathematical Models of Diastolic Blood Flow Patterns in the Human Left Ventricle*, [Bot89], and Verburg, Bot, Strackee and Delemaire [VBSD93].
See also:

- Bellhouse, [Bel72]
- Bellhouse and Bellhouse, [BB72]

Chapter 20

Modelling the Heart Valves

20.1 Introduction

The heart has four valves, the tricuspid, mitral, pulmonary and aortic valves. The tricuspid and mitral valves are called **atrioventricular valves** because they lie between the atria and the ventricles. The pulmonary and aortic valves are called **arterioventricular valves** because they lie between the arteries and the ventricles or, alternatively, the **semilunar valves** [1] because their leaflets have the shape of a half moon. The operation of the heart valves has aroused considerable interest since the valves are flaps of connective tissue and are largely or wholly passive.

The operation of the heart valves has nearly always been considered separately in the literature. The problem was considered by Leonardo da Vinci (see Peskin [Pes72] p. 9). Henderson and Johnson [HJ12], probably gave the first correct explanation. Fung [Fun84], pages 39-46, describes the operation of the different valves.

Peskin has made several contributions to the subject. He first considered the topic in his Ph.D.thesis ([Pes72]). There is a further discussion in his lecture notes ([Pes75], p. 26). Numerical results were later published ([Pes77]).
For a general discussion of the operation of the valves see:

- Section 1.3 of *Fluid Mechanics of Large Blood Vessels* by Pedley [Ped80].
- Section 2.5 of *Biodynamics: Circulation* by Fung [Fun84]
- Swanson and Clark [SC74].

20.2 The Mitral Valve

See

- Bellhouse and Bellhouse, [BB72]

[1] In German "Taschenklappen", i.e. "Pocket valves".

- Bellhouse, [Bel72]
- The Ph.D.thesis of Lee [Lee77].

For measurements see:

- Sugawara, Kagijiya, Kitabatake and Matsuo, [SKKM89]

20.3 The Aortic Valve

The operation of the mitral valve was considered by Lee and Talbot [LT79]. Some of their results are reproduced by Fung [Fun84] page 45. See also:

- Section 1.3.1 of *Fluid Mechanics of Large Blood Vessels* by Pedley [Ped80]
- *The Aortic Valve* by Thubrikar [Thu90]
- Section 1.3.2 of *Fluid Mechanics of Large Blood Vessels* by Pedley [Ped80]
- Section 2.5 of *Biodynamics: Circulation* by Fung [Fun84]

The aortic valve consists of three leaflets and three sinuses. The leaflets are the mobile parts of the valve. The sinuses are cavities behind the leaflets on the aortic (downstream) side of the valve. Each leaflet is pocket-shaped. When the ventricle is filling, and the pressure in the aorta is greater than in the ventricle, the pockets billow out, and together close the valve. When the pressure in the ventricle increases and exceeds that in the aorta, the leaflets are forced open.

There is a plausible explanation of why three leaflets are used. Consider a circular opening of radius r closed by n equal leaflets. The free edges of the leaflets have the length $2r$, while the length of the circumferential part of the leaflet is $2\pi r/n$. If $n = 1$ or 2 then the free edge is too short to fold back. If $n \geq 4$, the free edge is excessively long and will crease. If $n = 3$ the free edge can fold back easily without creasing. It may be noted in this connection that the aortic valve does not normally open completely: the area when open is approximately 50% ??? of the total area.

20.3.1 Architecture of the Aortic Valve Leaflets

Peskin and McQueen [PM93b] consider the structure of the aortic valve.It is assumed that the function of the closed valve is to support a uniform pressure load p_0. It is further assumed that this load is transfered to the aortic wall by a one-parameter family of fibres under tension.
Let (u, v) be coordinates on the surface of the leaflet, chosen in such a way, that the curves $v =$ constant are the fibres, with $v = 0$ being the free edge of the leaflet. The coordinate u measures arc length along the fibres, with $u = 0$ along the midline of the leaflet. The leaflet is given by

$$\mathbf{x} = \mathbf{X}(u, v).$$

Let $T(u, v)$ be the fibre tension, chosen in such a way that $T(u, v)dv$ is the force transmitted by the group of fibres in the interval $(v, v + dv)$. Let p_0 be the uniform pressure applied to the leaflet.

For the equilibrium of a patch of surface $[u_1, u_2] \times [v_1, v_2]$ we obtain

$$\int_{v_1}^{v_2} \int_{u_1}^{u_2} \left[\frac{\partial}{\partial_u} \left(T \frac{\partial \mathbf{X}}{\partial_u} \right) + p_0 \left(\frac{\partial \mathbf{X}}{\partial_u} \times \frac{\partial \mathbf{X}}{\partial_v} \right) \right] \quad dudv = 0$$

It is possible to derive certain consequences from equation 20.1:

- The torsion $T(u, v)$ is independent of u, so that the tension is constant on each fibre v.

  To see this, form the scalar product of $\frac{\partial \mathbf{X}}{\partial_u}$ with equation:

$$\frac{\partial T}{\partial_u} \frac{\partial \mathbf{X}}{\partial_u} \cdot \frac{\partial \mathbf{X}}{\partial_u} + T \frac{\partial \mathbf{X}}{\partial_u} \cdot \frac{\partial^2 \mathbf{X}}{\partial_{u^2}} + p_0 \frac{\partial \mathbf{X}}{\partial_u} \cdot \left(\frac{\partial \mathbf{X}}{\partial_u} \times \frac{\partial \mathbf{X}}{\partial_v} \right) = 0 \tag{20.1}$$

  Since

$$\frac{\partial \mathbf{X}}{\partial_u} \cdot \frac{\partial \mathbf{X}}{\partial_u} = 1$$

  we have, by differentiation,

$$\frac{\partial \mathbf{X}}{\partial_u} \cdot \frac{\partial^2 \mathbf{X}}{\partial_n^2} = 0.$$

  Also, for general vectors $\mathbf{a}$ and $\mathbf{b}$,

$$\mathbf{a}.(\mathbf{a} \times \mathbf{b}) = 0.$$

  We conclude that

$$\frac{\partial T}{\partial_n} = 0$$

- The fibres are geodesics on the valve leaflet surface.

This yields the equation of equilibrium

$$\frac{\partial}{\partial u} \left(T \frac{\partial \mathbf{X}}{\partial u} \right) + p_0 \left(\frac{\partial \mathbf{X}}{\partial u} \times \frac{\partial \mathbf{X}}{\partial v} \right) = 0 \tag{20.2}$$

Equation 20.2 can be integrated (numerically). Because of a singularity at the centre of the aortic valve, the (computed) fibre architecture has a fractal nature.

The resulting (computed) architecture agrees well with the true architecture. (PPL fragen) Some questions on these results of Peskin and McQueen (as of July 1 1994):

- To what extent do the assumptions lead to the consequences that
  - The fibre length is constant.

- The fibres are geodesics.

- Suppose we have a cylinder with circular fibres supporting an internal pressure p_0. Are there transformations (differential geometric) which transform this surface into a leaflet, preserving the properties?

- What do the numerical results really mean?

- Do we really just have a problem with inextensible cords?

- For what classes of surfaces does a family of geodesics have constant curvature?

20.4 Artificial Valves

Concerning **aritificial valves** see:

- Chapter 7 und Chapter 10 in *Cardiovascular Biomechanics* by Chandran, [Cha92]

- *The Aortic Valve* by Thubrikar [Thu90].

- *Blood Flow in Artificial Organs and Cardiovascular Prostheses* by Barbenel, Fisher, Gaylor et al. [BFG +89]

- *Unsteady flow through prosthetic heart valves: An integral approach coupled with a vortex method.* In: "Boundary Elements X - Vol. 2: Heat Transfer, Fluid Flow and Electrical Applications" by Cassot, Morvan and Tonietto [CMT88]

20.5 Valve Vibration

Concerning **valve vibration** see:

- Chapter 7, Analysis and Applications of Heart Valve Vibrations, of *An Introduction to Mathematical Physiology ad Biology* by Mazumdar, [Maz89]

Chapter 21

Modelling other Ventricular Processes

The mechanical events in the heart are accompanied by electrical, chemical, and thermal processes. In general it is assumed that the coupling is not very strong, so that the mechanical events can be treated separately, but it is necessary to be aware of the other processes. It may well be that in diseased hearts the effects of coupling are significant. (PPL fragen)

21.1 Electrical and Electrophysiological Activity

The contraction of the heart is activated by electrical impulses. These spread rapidly along the endocardium (inner surface) via the Purkinje fibre network and more slowly towards the epicardium (outer surface) via muscle cell conduction. The propagation is complete after about 100 ms. Hunter and Smaill [HS89] pages 150 - 158 describe experimental results and compare these to numerical models.

So far as we are aware (November 1993), the electrical activation has not been modelled in conjunction with ventricular mechanics. It seems reasonable to assume that the two aspects can be considered separately. It would , however, perhaps be of interest to determine whether it is of significance that most of the endocardium is activated first, in other words, that a 'pressure pulse' moves outward. This might for instance affect the blood flow in the myocardium. One surprising fact is that a small part of the left ventricular endocardium is activated relatively late (see Fung [Fun84] p. 31). The plot of isochronal contours on the epicardium given by Hunter and Smaill [HS89], Figure 27, page 152 also shows interesting features. (PPL fragen)
The energy for the contraction of heart muscle is provided by chemical reactions. Potential differences across cell membranes are linked to the flow of ionic currents. In turn, a rise in calcium ion concentration with in a cell activates the contractile proteins in the cell. The electrophysiological activity of the heart is considered in several of the later chapters in the collection *Theory of Heart* [HSHY91], to which we refer.

The underlying mathematical equations are related to the well-known **Hodgkin-Huxley equations** for conduction and exitation in nerves. Much of the emphasis in the modelling is on phenomena such as **fibrillation**, which are distinct from ventricular mechanics. The equations are also the subject of active mathematical research: See:

- Peskin [Pes75], p. 241
- Peskin [Pes76]
- The introductory text of Grinrod [Gri91]
- Fife [Fif79]

It has recently been suggested by Lab and Holden [LH91] that mechanoelectric feedback may occur.
See:

- Rogers and McCulloch, [RM94]
- Frese, [Fre94]

In the book *When Time Breaks Down. The Three-Dimensional Dynamics of Electrochemical Waves and Cardiac Arrhythmias* by Winfree [Win87] there are interesting figures, and ideas, with reference to **cardiac arrhythmias** or **arrhythmias**.
See also:

- Glass and Mackey *From Clocks to Chaos: The Rhythms of Life*, [GM88]
- Cohen and Hirsch *Mathematical developments in Hodgkin-Huxley theory and its approximations*, [CH76]
- Murray *Mathematical Biology*, [Mur93], p. 344
- Winfree *The Geometry of Biological Time*, [Win90]
- Kaplan, Ph. D. Thesis [Kap89]
- Winfree *Puzzles about excitable media and sudden death* [Win94]
- Scott *Oscillations, waves, and chaos in chemical kinetics* [Sco94]

21.2 Oxygen Diffusion

The heart needs a steady supply of oxygen without which misfunction occurs, the extreme example being a 'heart attack'. The demands placed on the coronary circulation system are high, and the uptake of oxygen from the blood in the myocardium is about three times higher than in the rest of the body ([Lev91], Figure 1.5 and Section 12.1). The flow of blood is influenced by the pressure in the heart walls. During isovolumetric contraction the coronary blood flow ceases and even reverses (see Levick [Lev91], p. 206). At the time of writing (November 1993) we are not aware of modelling in this area.

21.3 Heat Flow

Hunter and Smaill [HS89], pages 146 - 150, describe the modelling of heat flow in the heart.
According to Ghista [Ghi79] pages 135-136 the heart produces heat at the rate of about 46 kcal/day, and so produces about 12 % of the body's heat.

This figure seems doubtful, since a human uses about 2000 kcal/day. (see Guyton [Guy91] p. 794, Schmidt and Thews [ST93] p. 731 and p. 653, Schmidt-Nielsen [SN84] p. 57.) (CWC January 1995).
One night expect the temperature of the heart to influence the heart's output, since the following are certainly temperature dependent:

- The blood's viscosity and hence the flow of blood in the myocardium
- The elasticity parameters of the myocardium. For instance, collagen becomes pliant when heated (CWC - give reference). According to Guyton [Guy91], page 109.

Chapter 22

Comparative Physiology of the Heart

22.1 Introduction

It is of interest to compare the performance of hearts in different species:

- Out of scientific curiosity;
- Since experiments on animals are used to obtain information about humans;
- Since a good model should be able to predict the performance of the hearts of different species.

Nature offers a great range of circulatory systems and associated pumps. Two distinct groups of animals - the mammals and the birds - have developed closed circulatory systems with two separate pumps in one unit - the heart. It is reasonable to assume that this arrangement has advantages and to try to find them. It is for instance not immediately obvious why the two pumps should be in the same unit. For example, Woods [Woo92] page 369 suggested that it would follow from the **Law of Laplace** that it would be mechanically more efficient to have two separate pumps.

22.2 Sources of Data

The basis of any comparison of the hearts of different species must be the systematic accumulation of data.

At the time of writing (April 1994) we are aware of the following sources of information:

- *Biology Data Book, volume III* by Altman and Dittmer [AD74].
- *Comparative Physiology of the Heart* by Clark [Cla27].
- *Merck Veterinary Journal* by Fraser [Fra91]

- *The Ecological Implications of Body Size* by Peters [Pet83].
- *Comparative Animal Physiology* by Prosser [Pro91].
- *Animal Physiology* by Scheer [Sch60].
- *Scaling: Why is Animal Size so Important?* by Schmidt-Nielsen [SN84]

Schmidt-Nielsen [SN84], page 126 lists further references.

22.3 Allometric Equations

Despite the great range of diversity in nature there are also considerable similarities. For example, for the *mammalian* heart, the following equation relates the mass of the heart M_h (in kilograms) to the body mass M_b (in kilograms) ([SN84] page 126):

$$M_h = 0.058 M_b^{0,98}. \tag{22.1}$$

Equation 22.1 is valid over the full range of mammals from shrews with a body weight of 3 grams to elephants with a body weight of 4000 kg. Equation 22.1 states that, in general, the mass of the mammalian heart is approximately 0.6 % of the body mass.

Equation 22.1 is an example of an equation of the form

$$y = ax^b \tag{22.2}$$

or, equivalently

$$\log y = \log a + b \log x \tag{22.3}$$

with $y = M_h$ and $x = M_b$.

Equations of the form 22.2, frequently with x equal to the body weight M_b, are called **allometric equations** in biology. In the book *The Ecological Implications of Body Size*, by R.H. Peters [Pet83], a large number of allometric equations are given.

For the study of the cardiovascular system, the following allometric equations are of special interest:

If P_{met} denotes the metabolic rate for animals at rest (in kcal/day) then the well-known **mouse-to-elephant curve** is expressed by

$$P_{met} = 73.3 M_b^{0,74}. \tag{22.4}$$

This was given by Max Kleiber in 1932 but similar equations had long been known (see [Cla27]). Kleiber subsequently suggested that the equation

$$P_{met} = 70 M_b^{0.75} \tag{22.5}$$

would be equally reasonable.

The heartbeat frequency f_h (beats per minute) is given by (K. Schmidt-Nielsen [SN84] page 127)

$$f_h = 241 M_b^{-0.25} \tag{22.6}$$

The cardiac output $\dot{Q}_h$ (ml per minute) is given by (Schmidt-Nielsen, [SN84] page 127)

$$\dot{Q}_h = 187 M_b^{0.81} \tag{22.7}$$

Finally, the blood volume V_b (ml) is given by (Schmidt-Nielsen [SN84] page 117)

$$V_b = 65.6 M_b^{1.02}, \tag{22.8}$$

so that the blood volume accounts for between 6% and 7% of the body mass. In the book *Engineering Design of the Cardiovascular System of Mammals*, Dawson [Daw91] systematically applies scaling laws.

The foregoing allometric equations are intrinsically interesting, but must be treated with some caution. Clark [Cla27] quotes many such formulas but often has different values for the constants b. Clark also gives several graphs which illustrate the not inconsiderable scatter of the data.

22.4 Differences Between Mammalian Hearts

While there are far-reaching similarities between all mammalian hearts, there are also differences:

- Allometric equations represent trends, and there are substantial deviations for some animals. For example, the masked shrew, which has a body weight of 3 grams, has a heart which is 3 times larger than is predicted by equation 22.1.
- There are some qualitative differences which may be masked by allometric equations:
  - The structure of the myocardium of the dog heart may be different from that of man (Lunkenheimer, personal communication).
  - There may be qualitative differences in the fibre geometry between the mouse heart and the human heart. (McLean, Rross and Prothero [MRP89] as quoted by Horowitz [Hor91] page 54).
- Pedley [Ped80] page 41 comments on a significant difference between the coronary circulation in the horse and the dog.
- It has been suggested (see Schmidt-Nielsen [SN84] page 129) that the large size of the shrew heart may arise because there is a physical upper limit to the frequency of heart beats. As it is, the shrew heart can beat over 1200 times a minute, that is, one beat every 50 milliseconds!
- Schmidt-Nielson [SN90] page 115 comments on a difference between birds and mammals. For reasons which are not clear, some birds have blood pressures above 250 mmHg.
- Rowell [Row93] p. 5 emphasizes the fact that for upright humans, 70% of the blood volume is below the heart level, whereas for many animals, 70% of the blood volume is at or above heart level.
- During the Alicante conference, differences between human and pig hearts were reported??

22.5 Concluding Remarks

In summary, it seems to one of us (CWC) at the time of writing (April 1994) that - with the possible exception of very small hearts - there are no significant qualitative differences between mammalian hearts, which could be used to test different mathematical models of the heart.

What conclusions can be drawn which are relevant to the modelling of the cardiovascular system?

Chapter 23

Modelling the Global Motion of the Heart

As mentioned in section 6.3 the heart as a whole moves quite dramatically during a single heart beat. This motion has apparently only been considered by a few authors.

Figure 5.4 (b) illustrates the motion of the heart walls during systole: the apex of the heart moves towards the base, the outer walls contract, the septum becomes more convex with respect to the left ventricle. (PPL fragen) At the same time, the apex moves forward and taps against the chest (see Figure 5.3).
Until quite recently, it was not possible to accurately measure the motion of the heart, since one had to rely on visual observation in much the same way as William Harvey, who made some of his observations on dying animals, whose heartbeat was reduced. (Harvey [Har28], chapter 2.)
Robinson, Factor and Sonnenblick [RFS86] p. 69 comment on its implications and refer to the early technique of **ballistic cardiography** in which a subject lay on a wheeled table while the motions of the table in response to the motions of the heart were recorded. Related references are [Son80] and [YSF80]. The lighthearted book *Vital Circuits* by Steven Vogel also contains a brief reference to this subject [Vog92] page 149. So far as we are aware (April 1994), quantitative estimates of the effect of the heart's motion on its performance have not been made.
See also:

- Pedley [Ped80] pages 62-71 considers momentum.
- Books on ballistic cardiography (see Starr and Noordergraaf; Noordergraaf [Noo78]).
- Acierno [Aci94] pages 514-518.
- Rushmer [Rus68] pages 103-105.
- Buchalter, Weiss et al. [BWR +90] measure the rotation of the left ventricular apex using MRI tagging.

Beyer and Sideman [BS84b] appendix B consider the effect of twisting on fibre extension.

23.1 The Heart as a Mechanical Pump

Many authors have considered the **heart as a mechanical pump**:

- Burton [Bur57]
- Burch, Ray and Cronvich [BRC52]
- Beyar and Sideman *Spatial energy balance within a structural model of the left ventricle* [BS86]
- Peters and Visscher *The energy metabolism of the heart failure and the influence of drugs upon it* [PV36]
- Robinson, Factor, Sonneblick [RFS86]
- Wong *Some proposals in cardiac muscle mechanics and energetics* [Won73]
- Moskowitz [Mos81] estimates inertial effects.

The determinants of **myocardial oxygen consumption** $M\dot{V}_{O_2}$ have been considered by many authors:

- Kahler, Braunwald, Kelminson, Kedes, Chidsey and Segal *Effect of alterations of coronary blood flow on the oxygen consumption of the nonworking heart* [KBK +63]

The work W done by the heart is usually estimated as the sum of the mechnical work (**external work** or **volume-pressure-work**) and the kinetic energy:

$$W = \Delta\, P \,\cdot\, \Delta\, V + \frac{1}{2} m v^2$$

where ΔP is the pressure difference (*mean ejection pressure* minus *mena filling pressure*), ΔV is the mass of blood ejected and v is the mean ejection velocity of the blood. (see e.g. Levick [Lev91] p. 87)

For comparison with ordinary mechanical pumps see:

- Rogers and Mayhew *"Engineering Thermodynamics: Work and Heat Transfer* [RM92]
- Ryder and Bennett

23.2 Cardiac Energetics

Concerning **cardiac energetics** see:

- Gibbs *Cardiac energetics* [Gib87]
- Kleiber *The Fire of Life* [Kle75]
- Ryder and Bennett

Chapter 24

Growth

The human heart, like many other parts of the body, grows while maturing but also in response to changes in loading conditions. It is a challenge to determine the mechanisms which control this growth. The study of the **embryonic heart** is relevant here. (**growth** and **remodelling**).
Wolff's Law see:

- Wolff,*Das Gesetz der Transformation der Knochen* [?].

See:

- Thompson [Tho61]
- Lin and Taber [LT94]
- Rodriguez, Hoger, and McCulloch [RHM94]
- Yang, Taber, and Clark [YTC94]
- Currey *The Mechanical Adaptation of Bones* [Cur84]

Lin and Taber [LT94] model the growth of embryonic chick ventricles. They use the passive strain-energy function

$$\begin{aligned} W &= \tfrac{a}{b}\mathrm{e}^{bQ} \\ Q &= I_1 - 3 + \tfrac{1-2\nu}{\nu}[I_3^{-\dfrac{\nu}{1-2\nu}} - 1] \end{aligned}$$

where

$$\begin{aligned} I_1 &= \lambda_r^{*\,2} + \lambda_\theta^{*\,2} + \lambda_z^{*\,2} \\ I_3 &= \lambda_r^{*\,2}\lambda_\theta^{*\,2}\lambda_z^{*\,2} \end{aligned}$$

are strain invariants, introduced by Yang, Taber and Clark [YTC94].
The study of the growth, adaptation, remodelling and repair of living tissue raises interesting questions, some of which have mathematical counterparts.

In the present context one may ask whether knowledge about the stress distribution in an overloaded heart can be used to explain the development of hypertrophy (see Fung [Fun90] page 512). See:

- In Chapter 8 of *The Mechanical Adaptations of Bones* Currey [Cur84] critically discusses possible mechanisms.
- The book *On Growth and From* by D'Arcy Thompson [Tho61] is a classic in this field.
- Huxley [Hux93]
- Wolff [?]

Chapter 25

Miscellaneous Topics

25.1 Heart Sounds

Concerning **heart sounds** see:

- *The Guide to Heart Sounds: Normal and Abnormal* [NPS88]

Concerning **Korotkoff sounds** see:

- Pedley [Ped80] pages 55 and 308

25.2 The Interval-Force Relationship

Concerning the **interval-force relationship** or **force-frequency relationship** or **force-interval relationship** or **interval-strength relationship** associated with the name of **Bowditch**. See:

- Noble and Seed, *The Interval-Force Relationship of the Heart: Bowditch Revisited* [NS92b],
- Wiggers, *Circulatory Dynamics* [Wig52],
- Ciba Foundation *The Physiological Basis of Starling's Law of the Heart* [Sym74]
- Bowditch, 1871, *Ueber die Eigenthuemlichkeiten der Reizbarkeit, welche die Muskelfasern des Herzens zeigen* [Bow71] and Bowditch, 1992, *On the pecularities of excitability which the fibres of cardiac muscle show. (Translation of paper of 1871)* [Bow92]

25.3 Assistance and Replacement

See

- Chapter 10 *Circulatory System Dynamics* by Noodergraaf [Noo78]
- Chapter 7 and Chapter 10 in *Cardiovascular Biomechanics* by Chandran [Cha92]

25.4 Anrep Effect

Concerning the **Anrep effect** see:

- Ciba Foundation *The Physiological Basis of Starling's Law of the Heart* [Sym74]

25.5 Gregg Effect

Concerning the **Gregg effect** see:

- Livingston, Halpering and Yin [LHY94]
- Gregg [Gre63]

Part IV

Mathematical Modelling of the Circulatory System

Chapter 26

Introduction

In this Part we attempt to provide an overview of the circulatory system (other than the heart). As usual, we emphasize the associated mathematical problems.
As Lighthill [JL75] p. 199 comments:

> Many of the most important problems in this field, even if the fluid dynamics in them is considered in isolation, are found to raise questions which, in the whole proceding history of research on fluid dynamics, have totally failed to be answered, or in some cases even to be asked!

Some references include:

- *Circulatory System Dynamics* by Noordergraaf [Noo78]. This is a readible and useful description of the subject, with problems (and solutions) at the end of each chapter. One drawback is that the mathematics is often only briefly described.
- *Cardiovascular Fluid Dynamics, 2 vols.*, edited by Bergel [Ber72a], [Ber72b]
- *Blood Flow in the Heart and Large Blood Vessels*, by Sugawara, Kajiya, Kitabataka and Matsuo [SKKM89] which describes many experimental techniques and results.
- *Circulation* by Folkow and Neil [FN71]

They also discuss the measurement of abnormal flows caused by defects such as:

- **aortic regurgitation** in which blood reenters the left ventricle from the aorta. See also Braunwald [Bra92] p. 104.
- **shunts** involving alternative paths for the circulation of blood. See Braunwald [Bra92] p. 906.
- **stenosis**, the "narrowing of a passage in the body", e.g. **aortic stenosis**. See Braunwald [Bra92] p. 1035.

Chapter 27

The Arterial System

The standard reference is *McDonald's Blood Flow in Arteries* the latest edition of which is by Nichols and O'Rourke [NO90].
Other references include:

- Part IV, *Wave Propagation and Arterial Dynamics*, of *Cardiovascular Dynamics and Models* by Brun et al. [BCL88]
- The *Fluid Mechanics of Large Blood Vessels* by Pedley [Ped80].
- Chapter 4 of *Circulatory System Dynamics* by Noordergraaf [Noo78].
- *Arterial System Dynamics* by Li [Li87]
- Rubinow [Rub77]
- Chapter 7, Analysis and Applications of Arterial Flow Dynamics, of *An Introduction to Mathematical Physiology and Biology* by Mazumdar [Maz89]
- Chapter 12 and 13 of *Mathematical Biofluiddynamics* by Lighthill [JL75]
- Chapter 3 of *Biodynamics: Circulation* by Fung [Fun84]

27.1 Introduction

The folw of blood through the arteries represents an interesting mathematical challenge:

- Blood is a complex non-Newtonian fluid.
- The arterial walls are thick elastic containers.
- The arterial walls are often curved.
- The arteries branch repeatedly.
- The flow is time-dependent.

Since the pulsatile flow of the blood in the arteries is readily observed, it aroused early interest. Early contributors included:

- Stephen Hales (a clergyman) who, among other contributions, first measured arterial pressure [Hal]
- Thomas Young (physicist and physician) who first calculated the wave velocity for thin-walled arteries [You08] and [You09].
- Poiseuille (physician and physicist) who studied steady-state viscous flow [?].
- The brothers W. Weber (physicist) and E. H. Weber (physician) [WW25] and [Web50].
- Womersley [Wom57] and Witzig [Wit14].

There are brief historical introductions in:

- *McDonald's Blood Flow in Arteries* Nichols and O'Rourke [NO90] pages 2-6.
- *Biodynamics: Circulation* by Fung [Fun84], Chapter 3. Fung also includes (Table 3:15:1, pages 148 - 149) a tabular summary of the contributions of different authors.

Important results include the following.

The **Poiseuille flow** of a viscous fluid in laminar flow through a circular tube of radius a . The velocity profile is

$$u = -\frac{1}{4\mu}(a^2 - r^2)\frac{dp}{dx}$$

and the mean velocity is

$$u_m = -\frac{a^2}{8\mu}\frac{dp}{dx}.$$

Turbulent flow in a circular tube. The transition to turbulence is governed by the **Reynolds number**

$$R = \frac{UL}{\nu}$$

where U is a typical velocity, L a typical length, and ν the kinematic viscosity ($= \mu/\rho$). For flow in tubes, the flow becomes turbulent if R exceeds the critical value

$$R > R_{crit} = 2300$$

Surprisingly, in the ascending and escending aorta of man and dog, Reynolds numbers greater than 3000 can occur (Fung [Fun84] p. 12).

In 1926 Murray [Mur93] used Poiseuille's formula to analyse the optimum design of blood vessel bifurcation. Murray found that if a blood vessel of radius a_0 splits symmetrically into two vessels of diameter a_1 making angle θ with the original blood vessel, then

$$\begin{aligned} a_1 &= \tfrac{a_0}{2^{1/3}} \\ \cos\theta &= \tfrac{1}{2^{1/3}} \end{aligned}$$

so that $a_1 \doteq 0.794\ a_0$ and $\theta \doteq 37.5°$. These results, known as **Murray's formula** are roughly in agreement with measurements in the human body.

For flow in a thin-walled elastic tube (with Young's modulus E) of inner radius a_i and wall thickness h, the wave speed is found to be (Fung [Fun84] p. 107)

$$c = \sqrt{\frac{Eh}{2\rho a_i}}.$$

This formula was derived by Young in 1808 [You08] but is called the **Moens-Korteweg formula**.

More complicated flows were investigated by Witzig [Wit14] and Womersley [Wom57]. The **Womersley number**

$$\alpha = \frac{D}{2}\sqrt{\frac{\omega}{\nu}}$$

play an important role.

For numerical computations for flow in curved tubes (e.g. the aorta) see Chang and Tarbell [CT85].

There are interesting clinical applications of the above results (McDonald's Blood Flow in Arteries [NO90] contains numerous examples):

- The flow of blood is influenced by the stiffening of arteries with age in man and woman (see [NO90] p. 3)
- It has been postulated, that turbulence increases the haemolysis (loss of haemoglobin) of red blood cells (see [NO90] p. 63.).

Chapter 28

The Pulmonary Circulation

28.1 Introduction

The **pulmonary circulation** ist that part of the cardiovascular system in which blood is pumped from the right ventricle through the pulmonary artery to the lungs, returning through the pulmonary veins to the left artrium.

See:

- Kowalczyk and Kleiber *Modelling and numerical analysis of stresses and strains in the human lung including tissue-gas interaction* [KK94]
- Berne and Levy, [BL92] pages 243-249
- Pedley, *The Fluid Mechanics of Large Blood Vessels* [Ped80] pages 114 and 302
- Nichols and O'Rourke in Chapter 15 in *McDonald's Blood Flow in Arteries* [NO90]
- Levick *"An Introduction to Cardiovascular Physiology* [Lev91]
- *Handbook of Physiology, Section 2, Cardiovascular System, vol. 3, Peripheral Circulation*
- Section 4.2, *Circulatory System Dynamics* by Noordergraaf [Noo78]
- Chapter IV, *Mathematical Aspects of Heart Physiology* by Peskin [Pes75].
- *The Respiratory Physiology of Animals* by Cameron [Cam89]
- Chapter 6, *Mathematics in Medicine and the Life Sciences* by Hoppensteadt and Peskin [HP92]
- Chapter 11, Respiratory Flow Patterns of *Mathematical Biofluiddynamics* by Lighthill [JL75]

Chapter 29

The Venous System

The venous system has been studied comparatively little - there is no equivalent of *McDonalds Blood Flow in the Arteries.*

References include:

- Chapter 6, *Flow in Collapsible Tubes*, of *The Fluid Mechanics of Large Blood Vessels* by Pedley [Ped80]
- Chapter 4, *The Veins*, of *Biodynamics. Circulation* by Fung [Fun84]
- Chapter 5, *Circulatory System Dynamics* by Noordergraaf [Noo78]
- Amoore and Santamore [AS94]

Chapter 30

The Coronary Circulation

Some references on the **coronary circulation** are:

- Part I, *Myocardial Blood Flow* of *Cardiovascular Dynamics and Models* by Brun et al. [BCL88]
- *Fluid Mechanics of Large Blood Vessels* by Pedley [Ped80] contains scattered results. An interesting observation is that oscillations have been observed in the coronary artery of the horse which are not observed in a dog [Ped80] page 41.
- Chapter 14 of *McDonald's Blood Flow in Arteries* by Nichols and O'Rourke [NO90]
- *Mechanics of the Coronary Circulation* by Mates, Nerem and Stein [MNS83]
- Fulton [Ful82]
- *Microcirculation of the Heart. Theoretical and Clinical Problems* by Tillmanns, Kübler and Zebe [TKZ82]

A series of papers on the effects or coronary flow was:

- Kahler, Braunwald, Kelminson, Kedes, Chidsey and Segal *Effect of alterations of coronary blood flow on the oxygen consumption of the nonworking heart* [KBK +63]
- Ross, Klocke, Kaiser, Braunwald *Effect of alterations of coronary blood flow on the oxygen consumption of the working heart* [RKKB63]
- Sarnoff, Gilmore, Skinner, Wallace, Mitchell *Relation between coronary blood flow and myocardial oxygen consumption* [SGS +63]
- Weisberg, Katz and Boyd *Influence of coronary flow upon oxygen consumption and cardiac performance* [WKB63]

In his Ph.D. thesis, Huyghe [Huy86] gives a careful discussion of the influence of myocardial mechanics on the coronary blood flow (pages 1-10 to 1-13) and the influence of the coronary blood flow on myocardial mechanics (pages 1-14 to 1-15).

Huyghe [Huy86] briefly mentions the **garden hose effect** or **erectile properties of the coronary microvasculature** and quotes Vogel, Apstein, Briggs, Gaasch and Ahn [VAB +82].

Chapter 31

The Microcirculation

See:

- Chapter 5, *Microcirculation* in *Biodynamics: Circulation* by Fung [Fun84]
- Chapter 14, *Mathematical Biofluiddynamics* by Lighthill [JL75]

Part V

Mathematical Modelling of the Entire Cardiovascular System

Chapter 32

Introduction

The ultimate goal of modelling the cardiovascular system is of course to model the entire system as a whole. This goal is far from being achieved.

Some references are:

- Chapter 13 of *McDonald's Blood Flow in the Arteries* [NO90]
- The conference proceedings ? edited by Yin [Yin87]
- The thesis of Waldman [Wal83].
- The book of Noordergraaf *Circulatory System Dynamics* [Noo78], especially Chapter 8 *Control* and Chapter 9 *The Closed Cardiovascular System.*

Noordergraaf [Noo78] quotes work in which models containing hundreds of equations have been set up and solved by computer. He makes the following comments ([Noo78] page 296):

> The nature of the problems under scrutiny contains a temptation to write massive systems of equations, easily reaching into the hundreds. Yet, to date, models of the entire circulation have neither answered the questions posed above, nor many of the other questions which were, realistically, the motivation for developing the models in the first place.

Chapter 33

Models of the Cardiovascular System

A few authors have considered the complete cardiovascular cycle, by combining a model of the heart with a simple model of the circulatory system load.
See:

- Arts, Veenstra and Reneman [AVR82]
- Arts, Reneman and Veenstra [ARV79]
- Huyghe [Huy86]
- Chadwick [Cha82]

In two papers, Perl, Horowitz and Sideman [PHS86] and Horowitz, Perl, Sideman and Ritman [HPSR86], a complete canine cardiac cycle was modelled using finite elements. The authors used 192 three-dimensional isoparametric elements. The left ventricle was treated as two layers, the apex elements having 9 nodes, the reminder having 12 nodes. The fibres were modelled as truss elements. The model allows for large deformations and nonlinear constitutive equations. However, the active forces are treated as unknowns and are chosen during each time step so that the experimentally observed ventricular volume is obtained.

An interesting feature of this work is that the authors attempt to assess the importance of four factors: anisotropy, geometric nonlinearity, material nonlinearity, mechanical activation. They do so by dropping each one of these factors in turn and comparing the results. They conclude (Horowitz, Perl, Sideman and Ritman [HPSR86] p. 156) that all factors play a role, but the material nonlinearity is most significant.

Chapter 34

Control

See

- Chapter 8, *Circulatory System Dynamics* by Noordergraaf [Noo78]
- Kappel and Peer [KP93]. Based on the four compartment model of Grodins, Kappel and Peer develop a model for the response of the cardiovascular to a short term submaximal load. Basic mechanisms included in the model are Starling's law of the heart, the Bowditch effect, and autoregulation in the peripheral regions. The model provides a description of data obtained in bicycle ergometer tests.
- Chapters 8 and 9 *Cardiovascular Physiology* by Berne and Levy [BL92]
- *Human Circulation: Regulation During Physical Stress* by Rowell [Row86]
- *Human Cardiovascular Control* by Rowell [Row93] Discusses circulatory adjustment to upright posture (**orthostasis**) and dynamic exercise. Detailed, but does not treat meathematical models.
- Chapter VI *Mathematical Aspects of Heart Physiology* by Peskin [Pes75]
- *Biomechanics and Exercise Physiology* by Johnson [Joh91]

Chapter 35

Design Principles of the Cardiovascular System

Almost everyone who studies the cardiovascular system comes to the conclusion that the system must be optimal[1]. See:

- Fung
- Weibel *The Pathway for Oxygen* p. 58, discusses the question: *Are Animals Built Resonably* [Wei84] and introduces the concept **symmorphosis** on p. 59 and
- Weibel [Wei84], Chapter 13, reviews the respiratory system and particular the smallest mammal, the Etruscan shrew (see p. 399).
- Talk in Alicante

35.1 Optimization of Biological Structures

There are several books in which the **optimal design** of biological structure is considered:

- Gordon *The New Science of Strong Materials or Why You Don't Fall Through the Floor* [Gor76]
- Vogel *Life's Devices: The Physical World of Animals and Plants* [Vog88]
- Wainwright, Biggs, Currey, and Gosline *Mechanical Design in Organisms* [WBCG76]

Epilogue

In this text we have attempted to survey the present state of mathematical modelling of cardiovascular mechanics.

[1]CWC: Find a suitable quotation. E. g. Weibel p. 58

A great deal has been achieved, but many questions await definitive answers. At the risk of arousing criticism and demonstrating ignorance, we venture to make some general and perhaps provocative comments:

- In most numerical work in the literature, the results are shown graphically and it is very difficult for the reader to reproduce the results, since neither the data nor the programs are available. A praiseworthy exception is provided by Peter Hunter and his colleagues [NLSH91] who have made their data available both on diskette and by ftp.

- During the past few years, much work has concentrated on modelling the myocardium and the overall view has been less emphasized. Whereas Chadwick [Cha82] ventured to predict the pressure-volume cycle of the left ventricle, recent workers have taken a much narrower view.

- There is little work which combines the computations of the stress in the heart walls with computations of the blood flow in the ventricles.

- There seems to be a need for greater interaction between physiological and morphological observation and mathematical modelling.

- The cardiovascular system is extremely complex. At present, most modelling is carried out by small groups, Ph.D. students, etc. Full-scale modelling will require coordination of these effort.s

We conclude with an optimistic quotation from Kovacs [Kov91], p. 610.

> It is extremly likely that in the years to come diagnosis, therapy and follow-up of cardiac patients will critically depend on developments achieved through or motivated by current mathematical and theoretical methods.

Appendix A

The SI System

A.1 Physical Units

There is a bewildering range of physical units. Here we use, with very few exceptions, the SI system.

There are a number of books which list conversion factors. We have used Cook [Coo91]. The following introduction to units is along the lines of that given by Schmidt-Nielsen [SN90].

See also:

- Goldman and Bell, 1986, [GB86]
- Mechtly 1973, [Mec73]
- Symbols Committee of the Royal Society, 1975, [otRS71]

Physical quantity	SI base unit	Symbol
length	metre	m
mass	kilogram	kg
time	second	s
thermodynamic temperature	kelvin	K
amount of substance	mole	mol
electric current	ampere	A
luminous intensity	candela	cd

Table A.1: Base units

This appendix provides (1) names of units and their symbols, (2) a brief list of conversion factors, and (3) a list of some physical quantities and the recommended symbols for their designation.

A.2 Units

The SI system uses seven *base units* for seven dimensionally independent basic physical quantities; each base unit is represented by a *symbol*. Use of these symbols is *mandatory* see table A.1. The SI System recognizes several *derived units* (as well as some supplementary units). Although some of these have special symbols, they are derived from and defined in terms of the base units. A few of particular interest in physiology are listed in Table A.2. The following should be noted about the use of these units and symbols:

Physical quantity	SI derived unit	Symbol for unit	Definition in terms of base units
force	newton	N -	$m\ kg\ s^{-2}$
energy, work, quantity of heat	joule	J N.m	$m^2\ kg\ s^{-2}$
power	watt	W J/S	$m^2\ kg\ s^{-3}$
pressure	pascal	Pa N/m^2	$m^{-1}\ kg\ s^{-2}$
viscosity	poise	P 0.10 Ns/m^2	$m^{-1}\ kg\ s^{-1} \times 10^{-1}$
electric potential difference	volt W/A	V	$m^2\ kg\ s^{-3}\ A^{-1}$
electric resistance	ohm	Ω V/A	$m^2\ kg\ s^{-3}\ A^{-2}$
electric charge	coulomb	C -	$s\ A$
frequency	hertz	Hz -	s^{-1}

Table A.2: Derived units

1. The symbols are printed in roman (upright) letters. The symbols are not abbrevations and are not followed by a period, except at the end of a sentence.
2. The plural form of a symbol is unchanged, and the ending -s should not be used.
3. Certain units are derived from proper names (e.g. Volt); the symbols for these units are capital roman (upright) letters (V), but the unit names are not capitalized (volt).

Prefix	Symbol	Equivalent
tera	T	10^{12}
giga	G	10^{9}
mega	M	10^{6}
kilo	k	10^{3}
deci	d	10^{-1}
centi	c	10^{-2}
milli	m	10^{-3}
micro	μ	10^{-6}
nano	n	10^{-9}
pico	p	10^{-12}
femto	f	10^{-15}
atto	a	10^{-18}

Table A.3: Prefixes

There is frequent need for smaller and larger decimal multiples of the units. These are obtained by the use of the prefixes listed in Table A.3. These prefixes may be attached to any SI base or derived unit. (Large multiples of seconds, however, are rarely expressed this way.) The combination of prefix and symbol is regarded as a single symbol, and compound prefixes are not allowed. The decimal sign is usually printed as a point (.) in English, but as a comma (,) in French and German. In the SI system a point or comma should be used only for the decimal design. To facilitate the reading of long numbers the digits may be divided into groups of three, but not separated by commas.

Example: 212 489.143 not 212,489.143

When the decimal sign is placed before the first digit of a number, a zero should always be placed before the decimal sign.

Example: 0.143 872 not .143 872

Products or quotients of two or more units are treated according to the rules of mathematics. Note that the use of more than one solidus (/) is ambiguous. Thus, a specific rate of oxygen consumption can be written: $ml/kg\ s$, or $ml\ (kg\ s)^{-1}$, or $ml\ kg^{-1}s^{-1}$, or $ml/(kg \cdot s)$, or $ml \cdot kg^{-1} \cdot s^{-1}$ not $ml/kg/s$.

A.3 Useful conversion factors

Traditional units are commonly used in those fields of physiology that concern energy (e.g., in the area of metabolic rates) and pressure (e.g., in the area of gas exchange).

- Force
  1 kgf (1 kilogram-force) = 9.80665 N
  1 newton = 0.101971 kgf
- Energy, work (force × distance)
  1 cal = 4.1868 J
  1 joule = 0.238845 cal

  1 litre O_2 (at 0°C, 760 mm Hg), when used in oxidative metabolism, is often equated with 4.8 kcal; thus 1 liter $O_2 \simeq$ 20.083 kJ
- Power (work per unit time)
  1 cal s^{-1} = 4.1868 J s^{-1} = 4.1868 W
  1 kcal h^{-1} = 1.163 W
  1 watt = 0.238845 cal s^{-1} = 14.3307 cal min^{-1} = 859.845 cal h^{-1}
- Pressure (force per unit area)
  1 atm (1 standard atmosphere) = 1.01325* × 10^5 N m^{-2} = 101.325* kPa
  1 at (1 metric atmosphere) = 1 kgf = 0.980665 10^5 Nm^{-2} = 98.0665 kPka 1 mm Hg at 0°C (1 torr) = 1.33322 ×10^2 N m^{-2} = 133.322 Pa
  1 kN m^{-2} = 7.50061 mm Hg = 9.869 ×10^{-3} atm
- Volume
  1 litre is (since 1964) defined as exactly equal to 1 dm^3 (0.001 m^3); thus, ml = cm^3 and $\mu\, l = mm^3$

- Length
  1 inch = 25.4* mm
  1 foot = 0.3048* m

- Mass
  1 pound (avoirdupois) = 0.453592 kg
  1 ounce (avoirdupois) = 0.028495 kg

- Temperature
  The temperature unit of 1 degree Celsius ($°C$) [1] equals the temperature unit of 1 kelvin (K).
  The Celsius temperature is defined as the excess of the thermodynamic temperature over 273.15 K.

A.4 Physical Quantities

Physical quantities	Symbol	Dimensions
Length	l	L
Height	h	= length
Radius	r	= length
Area	A	L^2
Volume	V	L^3
Mass	m	M
Density (m/V)	ρ	ML^{-3}
Pressure	p	$ML^{-1}T^{-2}$
Time	t	T
Velocity	$u,\ v$	LT^{-1}
Frequency	f	T^{-1}
Force	F	MLT^{-2}
Work	W	= energy
Energy	$E,\ W$	ML^2T^{-2}
Power	P	ML^2T^{-3}
Temperature	T	θ

Table A.4: Physical quantities, recommended symbols and dimensions. Alternative symbols are separated by commas.

The names of some physical quantities and *recommended* symbols are given in Table A.4. The symbol for a physical quantity should be a single letter of the Latin or Greek alphabet and should be printed in sloping (italic) type. When necessary, subscripts (and / or superscripts) are used to indicate a specific meaning of a symbol. For example: body temperature, T_b; blood volume, V_{bl}; rate of oxygen consumption, V_{O_2}.

Equations that represent physical or physiological relationships use these and similar symbols for physical quantities. Symbols for units (m, kg, s, etc.) should *never* be used in such equations.

[1] Also incorrectly known as "centigrade".

Appendix B

Dimensional Analysis

B.1 Dimensional Analysis

When a physical phenomenon is observed experimentally and the results are then represented, by an equation or “law”, then the measurements must be made in terms of physical units such as metres or feet, seconds or years, but the particular physical units used are clearly of secondary importance, and one may reasonably expect to be able to express the law in a form which is independent of the actual physical units used. This can be achieved by introducing dimensionless variables and constants. **Dimensional analysis** provides a tool to do this.
Dimensional analysis brings additional benefits:

- In simple cases it is possible to deduce the general form which any law must take.
- By ensuring that the dimensionless parameters are not changed, one can scale a problem without changing the form of the solution.

As an example with biological applications, the nature of the flow of a fluid with velocity u, density ρ, and dynamic viscosity η in a straight tube of diameter d, is determined by the **Reynolds number**

$$Re := \frac{ud\rho}{\eta}.$$

For small values of Re the flow is laminar (smooth), but if $Re > 2000$ the flow can become turbulent.

In animals, one would expect that laminar flow in blood vessels would be favoured by natural selection, but in fact the Reynolds number is often close to 2000 turbulent flow can occur (Y.C. Fung [Fun84] p. 97, [Fun90], p. 179, K. Schmidt-Nielsen [SN84] page 137.)

Dimensional analysis can be systematized as follows:

1. Determine all variables and parameters which occur in a problem.

2. Determine a complete set of dimensionless products Π.

3. Apply **Buckingham's Π-Theorem**:
   Theorem An equation is dimensionally homogenous if and only if it can be expressed in the form $F(\Pi_1, \Pi_2, \ldots, \Pi_n) = 0$ where $\{\Pi_1, \Pi_2, \ldots, \Pi_n\}$ is a complete set of dimensionless products.

To illustrate the approach, consider the pulsating flow of blood in an artery. As determining variables we take:

$$
\begin{array}{rcll}
r & = & \text{radius of artery} & [L] \\
f & = & \text{frequency} & [T^{-1}] \\
\rho & = & \text{density of blood} & [ML^{-3}] \\
\mu & = & \text{(dynamic) viscosity of blood} & [ML^{-1}T^{-1}]
\end{array}
$$

To determine a complete set of dimensionles products, we consider the product

$$r^a f^b \rho^c \mu^d$$

which has the dimensions

$$L^a T^{-b} M^c L^{-3c} M^d L^{-d} T^{-d}.$$

For this product to be dimensionless, we must have

$$
\begin{array}{lcll}
\text{a-3c-d} & = & 0 & [\text{L}] \\
\text{-b-d} & = & 0 & [\text{T}] \\
\text{c+d} & = & 0 & [\text{M}]
\end{array}
$$

from which it follows that

$$
\begin{array}{rcl}
c & = & -d \\
b & = & -d \\
a & = & 3c + d = -2d
\end{array}
$$

Taking, for convenience, $d = -1/2$ we see that $\{\Pi_1\}$ is a base with

$$\Pi_1 = r\sqrt{\frac{f\rho}{\mu}}.$$

Π_1 is called the **Womersley parameter**.

- It is often denoted by

  $$\alpha = r\sqrt{\frac{f}{\nu}}$$

  where $\nu = \mu/\rho$ is the kinematic viscosity.

It follows from Buckingham's Π-theorem that any solution must be of the form

$$F(\alpha) = 0.$$

In his book *Life's Devices* Vogel ([Vog88] page 117) discusses several examples of scaling when analysing biological problems.
References on dimensional analysis include:

- *Dimensional Analysis* by P. W. Bridgman [Bri31]
- *Dimensional Analysis in the Biomedical Sciences* by B. Schepartz [Sch80a].
- Chapter 9 in *Effects of Scale* in *Muscles, Reflexes, and Locomotion* by Thomas A. McMahon [McM84].
- Chapter 1 in *An Introduction to Mathematical Physiology and Biology* by J. Mazumdar [Maz89].
- *Dimensional Analysis and Theory of Models* by Langhaar [Lan51]

Dimensional analysis is a special case of applications of the mathematical concept of **Lie Group** to differential equations. Recent references to this subject include P.J. Olver [Olv93] and G.W. Bluman and S. Kumei [BK89].

Some applications of dimensional analysis to biological problems are:

- In his Ph.D. thesis, Peskin [Pes72], pages 38-42, the flow patterns in the hearts of different mammals are compared.
- Fung [Fun84], page 270 uses dimensional analysis to consider microcirculation in the body.
- Holmes [Hol84] (quoted by G.W. Bluman and S. Kumei [BK89], p. 28) uses dimensional analysis in the modelling of soft tissue.
- Pritchard [Pri93a], [Pri93b] has pointed out that most allometric equations violate the rules of dimensional analysis. He has suggested some new equations which conform to dimensional analysis. These might find application in the modelling of the cardiovascular system.
- Lee [Lee77] in Chapter 6 discusses applications to hemodynamics.
- Günther [Gue75] (quoted by Peters [Pet83]
- In Chapter 9 of *Elastic Mechanisms in Animal Movement* by Alexander [Ale88] uses dimensional analysis to discuss **elastic similarity** and **dynamic similarity**

Appendix C

Elementary Concepts from Physical Chemistry

See:

- Moore *Physical Chemistry*, [Moo72]
- Atkins *Physical Chemistry*, [Atk94]
- Fenn *Engines, Energy and Entropy*, [Fen82]
- Atkins *Elements of Physical Chemistry* [Atk92]
- Atkins *Physical Chemistry. 5th edition* [Atk94]

Appendix D

Physiology

See:

- Guyton, *Textbook of Medical Physiolog*, [Guy91]
- Glaser, *Biophysik*, [Gla86]
- Schmidt and Thews, *Physiologie des Menschen. 25. Auflage.*, [ST93]
- Le Gros, *The Tissues of the Body*, [WELGC75]
- Duerr and Uehlinger, *unbek.*, [Sau81]

Appendix E

Cardiovascular Data

E.1 Man

We consider a "normal" young adult man weighing

$$W = 70kg.$$

The corresponding normal heigth is

$$H = (70 + 100)cm = 1,7m.$$

If we assume a normal height of H = 1,70 m then, according to the **formula of DuBois and DuBois** [DD15] he has a **body surface area** of

$$S = 7.184\ 10^{-3}\ \cdot\ W^{0.425}\ \cdot\ H^{0.725} \approx 1.8m^2$$

(This formula is quoted by Dinnar [Din81] but does not appear in Dubois and Dubois [DD15]. See Kleiber [Kle75] p. 184.)
According to the formula of Carpenter[1] he has a **basal metabolic rate** (BMR) of:

$$\text{BMR } = 2.77 + 0.57W + 0.21H - 0.28 \text{ Age} \approx 67 \text{ kcal/h}$$

for a man of age 40. (But Dinnar [Din81] quotes 72 kcal/h as typical.)

See:

- Altman and Dittmer, *Biology Data Book, vol. III*, [AD74]
- Altman, Dittmer and Grebe *Handbook of Circulation*, [ADG59]
- Milnor, *Cardiovascular Physiology* [Mil90]

E.2 Woman

E.3 Dog

[1] See Dinnar [Din81] p. 135

Appendix F

Mathematical Tools

See:

- *Mathematical Techniques for Biology and Medicine* by Simon [Sim86]

F.1 Differential geometry

Peskin (see e.g. Peskin, [Pes75] has applied differential geometry in several cases.
See:

- *Modern Differential Geometry of Curves and Surfaces* by Gray [Gra93]
- *Tensor Analysis on Manifolds* by Bishop and Goldberg [BG80]
- *Differential Geometry of Curves and Surfaces* by do Carmo [dC94]
- *Riemannian Geometry* by do Carmo [dC92]
- *Differential Forms with Applications to the Physical Sciences* by Flanders [Fla89]
- *Differential Geometry* by Kreyszig [Kre91]
- *Differential Geometry* by Stoker [Sto89]
- *Lectures on Classical Differential Geometry* by Struik [Str81]
- *An Introduction to Differential Topology* [Wil59]
- Schweitzer, Hurder, dos Santos, and Arraut, *Differential Topology, Foliations and Group Actions*, [SHdSA94]
- Eisenhart, *Introduction to Differential Geometry with Use of the Tensor Calculus* [Eis26]
- Eisenhart, *Riemannian Geometry* [Eis50]
- Abraham, Marsden and Ratiu *Manifolds, Tensor Analysis, and Applications* [RAR88]

- Conlon, *Differentiable Manifolds: A First Course* [Con93]
- Kosinski *Differential Manifolds*, [Kos93]
- Klingenberg *Riemannian Geometry*, [Kli95]

F.1.1 Geodesics

The concept of a geodesic has been used in several areas:

- Fibres in the heart (Peskin).
- Chapter 5 "Fibre-wound animals" in *Elastic Mechanisms in Animal Movement* by Alexander [Ale88]. See also Alexander, 1987, [Ale87].
- Horowitz, Perl, and Sideman, 1993, [HPS93]

F.2 Geometric Measure Theory

Concerning **geometric measure theory** see:

- *Geometric Measure Theory. A Beginners Guide* by Morgan [Mor88]

F.3 Topology

The translation of Tamura's book [Tam92] contains a list of recent references on foliations. See:

- Abraham and Marsden, *Foundations of Mechanics*, [AM78]
- Armstrong, *Basic Topology*, [Arm83]
- Arnold, *Catastrophe Theory*, [Arn72]
- Arnold, *Singularity Theory*, [Arn81]
- H. Blaine Lawson, *The Quantitative Theory of Foliations* [Law77]
- Conlon, *Differentiable Manifolds: A First Course* [Con93]
- Fuller, *The writhing number of a space curve*, [Ful71]
- Fuller, *Decomposition of the linking number of a closed ribbon: A problem from molecular biology*, [Ful78]
- Gordon, Moriah and Wajnryb *Geometric Topology*, [GMW94]
- Hempel, *3-Manifolds*, [Hem76]
- Lawson, *The Quantitative Theory of Foliations*, [Law77]
- Nash and Sen *Topology and Geometry for Physicists* [NS92a]
- Schwarz, *Topology for Physicists* [Sch94]

- Tamura, *Topology of Foliations: An Introduction* [Tam92]
- Thurston and Weeks, *The mathematics of three-dimensional manifolds*, [TW84]
- Weeks, *The Shape of Space*, [Wee85]

F.3.1 Knots

If we regard the fibres in the heart wall as twisted, then we may well ask whether they form **knots**. See:

- Adams, *The Knot Book*, [Ada94]
- Livingston, *Knot theory* [Liv93]

F.4 Partial Differential Equations

See:

- Renardy and Rogers, [RR93b]

F.5 Singularities

See:

- Arnold, *Catastrophe Theory* [Arn72]

F.6 Chaos

Some books treating **chaos** include:

- Strogatz *Singular filaments organize chemical waves in three dimensions. 4. Wave Taxonomy* [WS84]

Appendix G

Mechanics

References on **mechanics** are:

- Chetaev, *Theoretical Mechanics* [Che89]
- Goldstein, *Classical Mechanics*, [Gol80]
- Whittaker, *A Treatise on the Analytical Dynamics of Particles Rigid Bodies* [Whi93]

G.1 Applied Mechanics

Under **applied mechanics** we understand elementary mechanis applied to practical engineering problems. See:

- Hillier *Applied Mechanics* [HH95]
- Hibbeler *Engineering Mechanics: Statics and Dynamics* [Hib95]
- Meriam and Kraige *Engineering Mechanics: Dynamics* [MK93]
- Ryder and Bennet

G.2 Thermodynamics

Concerning **thermodynamics** see:

- Rogers and Mayhew *Engineering Thermodynamics: Work and Heat Transfer* [RM92]

G.3 Contiuum Mechanics

Standard classical references on **continuum mechanics** are:

- Truesdell and Noll *The Non-Linear Field Theories of Mechanics. Handbuch der Physik* [TN65]

- Truesdell and Toupin *The Classical Field Theories. Handbuch der Physik, vol. III/1* [TT60]
- Truesdell *The Elements of Rational Mechanics* [Tru65]

Modelling using continuum mechanics is discussed by:

- Fung *A First Course in Continuum Mechanics* [Fun94]
- Allen, Herrera, and Pinder *Numerical Modeling in Science and Engineering*, [IHP88].
- Bowen *Introduction to Continuum Mechanics for Engineers* [Bow89]
- Malvern *Introduction to the Mechanics of a Continuous Medium* [Mal69]
- Truesdell *A First Course in Rational Continuum Mechanics* [Tru91]
- Fung *Foundations of Solid Mechanics* [Fun65]
- Smith *Constitutive Equations for Anisotropic and Isotropic Materials* [Smi94]

G.4 Elasticity

References on **elasticity** include:

- Antman, *Nonlinear Problems of Elasticity* [Ant95]
- Ciarlet *Mathematical Elasticity, vol. I* [Cia88]. The standard introduction for mathematicians.
- Ciarletta and Iesan *Non-classical Elastic Solids* [CI93]
- Crandall, Dahl and Lardner *An Introduction to the Mechanics of Solids* [CDL78]
- Gordon *The New Science of Strong Materials or Why You Don't Fall Through the Floor* [Gor76]
- Green and Adkins *Large Elastic Deformations and Non-Linear Continuum Mechanics* [GA70]
- Green and Zerna, *Theoretical Elasticity* [GZ92]
- Gurtin, *Topics in Finite Elasticity* [Gur81]
- Love, *A Treatise on the Mathematical Theory of Elasticity* [Lov44]
- Malvern, *Introduction to the Mechanics of a Continuous Medium* [Mal69]
- Marsden and Hughes, *Mathematical Foundations of Elasticity* [MH83]
- Maugin *Material in homogeneities in Elasticity* [Mau93]
- Spencer, *Continuum Mechanics* [Spe80]
- Stoker *Nonlinear Elasticity* [Sto68]
- Timoshenko and Goodier *Theory of Elasticity* [TG70]
- Wu, Ting and Barnett *Modern Theory of Anisotropic Elasticity and Applications* [WTB90]

G.4.1 Fibre-reinforced Materials

See:

- Mallick, *Fiber-reinforced Composites* [Mal93]
- Derek Hull *An Introduction to Composite Materials* [Hul81]
- Green and Adkins, chapter VII, Reinforcement by Inextensible Cords *Large Elastic Deformations and Non-Linear Continuum Mechanics* [GA70]
- Christensen, *Mechanics of Composite Materials* [Chr91]

G.4.2 Elastic Solids with Microstructure

See:

- Ciarletta and Iesan *Non-classical Elastic Solids* [CI93]
- Kunin *Elastic Media with Microstructure I* [Kun82] and Kunin *Elastic Media with Microstructure II* [Kun83]
- Capriz *Continua with Microstructure* [Cap85]
- Nowacki *Theory of Asymmetric Elasticity* [Now86]

See also literature on porous solids. (Hornung, [DH93]).

Appendix H

Biomechanics

H.1 General References

See:

- Alexander, *Animal Mechanics*, [Ale83]
- Alexander, *Elastic Mechanisms in Animal Movement*, [Ale88]
- Alexander, *Mechanics of Animal Locomotion*, [Ale92a]
- Biewener, *Biomechanics - Structures and Systems. A Practical Approach*, [Bie92]
- Currey, *The Mechanical Adaptation of Bones*, [Cur84]
- Fung, *A First Course in Continuum Mechanics*, [Fun94]
- Flindt, *Biologie in Zahlen* [Fli95]
- Gans, *Biomechanics: An Approach to Vertebrate Biology* [Gan80]
- Johnson, *Biomechanics and Exercise Physiology* [Joh91]
- McMahon, *Muscles, Reflexes, and Locomotion*, [McM84]
- Valenta, *Biomechanics*, [Val93]
- Vincent, *Structural Biomaterials* [Vin90]
- Vincent, *Biomechanics - Materials. A Practical Approach* [Vin92]
- Vincent and Currey, *The Mechanical Properties of Biological Materials (Symp. Soc. Exp. Biol. 34.)*, [VC80]
- Vogel, Steven, *Life's Devices: The Physical World of Animals and Plants*, [Vog88]
- Wainwright, *Axis and Circumference*, [Wai88]
- Wainwright, Biggs, Currey and Gosline, *Mechanical Design in Organisms*, [WBCG76]

H.2 Biomaterials

See:

- Brown, *Structural Materials in Animals* [Bro75]
- Fung, *Biomechanics: Mechanical Properties of Living Tissues* [Fun93]
- Silver, *Biological Materials: Structure, Mechanical Properties, and Modeling of Soft Tissues* [Sil87]
- Vincent, *Structural Biomaterials* [Vin90]
- Vincent, *Biomechanics - Materials. A Practical Approach* [Vin92]
- Vogel, *Life's Devices: The Physical World of Animals and Plants* [Vog88]
- Yamada, *Strength of Biological Materials* [Yam70]

H.3 Mathematical Biology

See:

- Harrison *Kinetic Theory of Living Pattern* [Har93a]
- Levin, *Lectures on Mathematics in the Life Science, Vol. 9, Some Mathematical Questions in Biology* [Lev77]
- Levin *Frontiers in Mathematical Biology* [Lev94]
- Murray *Mathematical Biology* [Mur93]
- Levin *Frontiers in Mathematical Biology* [Lev94]

H.4 Pattern Formation

Concerning **pattern formation** see:

- Harrison *Kinetic Theory of Living Pattern* [Har93a]
- Murray *Mathematical Biology* [Mur93]

Concerning **phyllotaxis** see:

- Jean, *Phyllotaxis. A systemic study in plant morphogenesis* [Jea93]
- Thompson, *On Growth and Form* [Tho61]

The phenomenon of phyllotaxis refers to the arrangement of leaf or floret primordia in plants according to the Fibonacci sequence $\{1, 1, 2, 3, 5, 8, 13 \ldots\}$ as seen in more than 90% of plants.

H.5 Bioenergetics

See:

- Kleiber, *The Fire of Life* [Kle75]

H.6 Kinesiology

The science of the movement of the body is called **kinesiology** See:

- Rasch,

Appendix I

Computer Graphics

Concerning **computer graphics** see:

- Foley, van Dam, Feiner and Hughes *Computer Graphics. Principles and Practice* [FvDFH90]
- Glassner *Graphics GEMS* [Gla90]
- Holden *Reconstructing the heart* [Hol94]
- Hoehne *3D-Computergrafik in der Medizin. Diagnose glasklar* [Hoe88]
- O'Rourke *Computational Geometry in C* [O'R93]
- Wickham-Jones *Mathematica Graphics: Techniques and Applications.* [WJ94]

Appendix J

Numerical Methods

Concerning **numerical methods** see:

- Adams, *MUDPACK: Multigrid portable FORTRAN software for the efficient solution of linear elliptic partial differential equations* [Ada89]
- Allen, Herrera and Pinder *Numerical Modeling in Science and Engineering* [IHP88]
- Bank, *PLTMG: A Software Package for Solving Elliptic Partial Differential Equations. Users' Guide 7.0* [Ban94]
- Brebbia and Dominguez, *Boundary Elements - An Introductory Course. 2nd edition* [BD92]
- Burkett, *Numerical methods for viscoelasticity* [Bur94]
- Szabo and Babuska, *Finite Element Analysis* [SB91]
- Smith and Criffiths, *Programming the Finite Element Method* [SG88b]
- Crisfield,*Non-linear Finite Element Analysis of Solids and Structures* [Cri91b]
- Zafrany El, *Techniques of the Boundary Element Method* [El93]
- Kim and Karrila, *Microhydrodynamics* [KK91]
- Hirsch, *Numerical Computation of Internal and External Flows. Vol. 2: Computational Methods for Inviscied and Viscous Flows* [Hir90]
- Glowinski and Le Tallec,*Augmented Lagrangian and Operator-Splitting Methods in Nonlinear Mechanics* [GT89]
- Grossmann and Roos, *Numerik partieller Differentialgleichungen* [GR92]
- Langtangen and Nielsen *Getting started with Diffpack. The Diffpack Report Series* [LN94]

J.1 Finite Elements

Concerning **finite elements** see:

- Dhatt and Touzot, *The Finite Element Method Displayed* [DT84]
- MacNeal, *Finite Elements: Their Design and Performance* [Mac94]
- Reddy, *An Introduction to the Finite Element Method* [Red93]
- Schwarz, *Methode der finiten Elemente* [Sch80b]
- Schwarz, *FORTRAN-Programme zur Methode der finiten Elemente* [Sch81]
- Zienkiewicz and Taylor *The Finite Element Method. Vol. 1. Basic Formulation and Linear Problems* [ZT91a]
- Zienkiewicz and Taylor *The Finite Element Method. Vol. 2. Solid and Fluid Mechanics. Dynamics and Non-Linearity* [ZT91b]

Appendix K

Sources

K.1 Sources of Preserved Hearts

The following information about suppliers of models of hearts is taken from Vogel [Vog92] page 40:

All of these vendors offer embalmed cow or calf, sheep or lamb, and pig hearts. They all have horribly expensive anatomical models, but Fisher and Wards have less costly ones as well. For nonfondlers NASCO has a reasonably priced sheep heart sliced open an embedded in plastic, and Wards has freeze dried whole and bisected (as opposed to dissected) sheep hears. Addresses:

- Carolina Biological Supply Co.
  Burlington, NC 27215
  919-584-0381

- NASCO
  901 Janesville Ave, Box 901
  Ft. Atkinson, WI 53538
  414-563-2446

- Fisher Scientific Company
  Educational Materials Division
  4901 W. LeMoyne Street
  Chicago, IL 60651
  312-378-7770

- Ward's Natural Science
  Establishment
  P.O. Box 92912
  Rochester, NY 14692
  716-359-2502

A good source for inexpensive models (but whose catalog may be dangerous to your financial security) is

- Edmund Scientific Co.
  101 E. Glouchester Pike
  Barrington, NJ 08007
  609-573-6259

K.2 Books, Reports, Theses

- BMJ Bookshop, P.O.Box 295, London WC1H 9TE, England.
  Carries stock of medical books.
- National Technical Information Service (NTIS)
  Can provide copies of almost all official technical reports published in the U.S.A.
- University Microfilm (UMI)
  Can provide copies of all Ph.D. theses listed in the journal.

K.3 Journals

The following journals frequently contain information on the cardiovascular system:

- Cardiovascular Research

K.4 Societies

- Cardiovascular System Dynamics Society
  The society holds a biannual conference, the proceedings of which are published. Address:

  J. Yasha Kresh, Ph.D.
  Likoff Cardiovascular Institute
  Hahnemann University, Room 6320 NCB
  Broad & Vine Streets, Mail Stop 110
  Philadelphia, PA 19102-1192
  Tel. (215) 762-1703
  Fax. (215) 762-7222
  email: kresh@cvi.hahnemann.edu

- European Society for Noninvasive Cardiovascular Dynamics:

  The European Society for Noninvasive Cardiovascular Dynamics (ESNICVD) was initiated by the physicist Prof. A.C. Burger in the Netherland 1960. The main goal of the ESNICVD is to unite and to keep together scientists and professionals whose common interest is to promote the knowledge and understanding of cardiovascular dynamics with noninvasive methods in research as well as in medical practice. The congresses which have been held all over Europe for the past 3 1/2 decades serve to keep the scientists from this field together despite the movement towards ever greater specialization.

- Dutch Foundation for Biophysics (Verenigung voor Biofysica) This has a subsection for the Heart and Circulation:
  Verenigung voor Biofysika (Kring Hart en Circulatie)

- British Heart Group

K.5 Deutsche Literatur

1. Bauereisen, *Physiologie des Kreislaufs. 1 + 2* [Bau71]

2. Bowditch, *Ueber die Eigenthuemlichkeiten der Reizbarkeit, welche die Muskelfasern des Herzens zeigen* [Bow71]

3. Braun, *Diagnose und Therapie der Herzkrankheiten* [Bra13]

4. Burton, *Physiologie und Biophysik des Kreislaufs* [Bur69]

5. Caflisch, *Strukturgerechte Analyse der Myokardfaserspannungen* [Caf87]

6. Culclasure, *Anatomie und Physiologie des Menschen. 15 Lehrprogramme. Band 4: Herz- und Kreislaufsystem* [Cul93]

7. do Carmo, *Differentialgeometrie von Kurven und Flaechen* [dC92]

8. Dierberger, *Finite-Elemente-Simulation der Herzdynamik. Das Herzgroessen-Schlagvolumen-Konzept* [Die93]

9. Doerr et al. *Pathologische Anatomie des Herzens und seiner Huellen. I.* [DSU]

10. Faller, *Der Faserverlauf im Bindegewebe des Epikards menschlicher Herzen und funktionelle Deutung* [Fal44]

11. Feneis, *Das Gefuege des Herzmuskels bei Systole und Diastole* [Fen44]

12. Flindt, *Biologie in Zahlen* [Fli95]

13. Frese, *Chaos Theorie und ploetzlicher Herztod* [Fre94]

14. Frank, *Zur Dynamik des Herzmuskels* [Fra95]

15. Glaser, *Biophysik* [Gla86]

16. Grossmann and Roos, *Numerik partieller Differentialgleichungen* [GR92]

17. Hoehne, *3D-Computergrafik in der Medizin. Diagnose glasklar* [Hoe88]

18. Hort, *Untersuchungen ueber die Muskelfaserdehnung und das Gefuege des Myokards in der rechten Herzkammerwand des Meerschweinchens* [Hor57]

19. Hort, *Untersuchungen zur funktionellen Morphologie des Bindegewebsgeruestes und der Blutgefaesse der linken Herzkammerwand* [Hor60]

20. Horvath, *Physikalische Uebungsaufgaben fuer Biologen und Mediziner* [Hor92]

21. Krehl, *Beitraege zur Kenntniss der Fuellung und Entleerung des Herzen* [Kre91]

22. Leonhardt *Taschenatlas der Anatomie. Band 2: Innere Organe. 6. Auflage* [KLP91]

23. Lunkenheimer et al. *Kardiodynamik: Wege zur strukturgerechten Analyse der Myokardfunktion* [LLG85]

24. Mattheck, *Design in der Natur* [Mat93]

25. Meesmann, *Nachweis der diastolischen Sogwirkung der Herzkammern und deren Einfluss auf die intrakardialen Druckablaeufe* [Mee58]

26. Meinzer, *Raeumliche Bilder des Koerperinneren* [Mei93]

27. Puff, *Der funktionelle Bau der Herzkammern* [Puf60]

28. Schmidt und Thews, *Physiologie des Menschen* [ST93]

29. Schwarz, *Methode der finiten Elemente* [Sch80b]

30. Schwarz, *FORTRAN-Programme zur Methode der finiten Elemente* [Sch81]

31. Thomas et al., *Grundlagen der klinischen Medizin. Anatomie, Physiologie, Pathologie, Mikrobiologie, Klinik: Band 1: Herz und Gefaesse* [TGH89]

32. Tigerstedt, *Die Physiologie des Kreislaufes* [Tig23]

33. Torrent Guasp ; Lunkenheimer *Intensivkurs: Funktionelle Anatomie des Herzens. Vom 20.-27.01.1981 bei Dr. Torrent-Guasp in Spanien, Denia, Alicante. Gesprärchsnotizen* [GL81]

34. Trost, *Analysis eines raeumlichen Modells vom Herzmuskel* [Tro78]

35. Wolff, *Das Gesetz der Transformation der Knochen* [?]

K.6 Deutsch-Englisch-Verzeichnis

K.7 Diplomthemen

See:

- Adams *A multigrid algorithm for immersed interface problems* [Ada93]
- Cameron *The Respiratory Physiology of Animals* [Cam89]
- Costa, Hunter, Rogers, Guccione, Waldman, and McCulloch *A three-dimensional finite element method for large elastic deformations of ventricular myocardium. Part I: Cylindrical and Sherical Polar Coordinate* [CHR +94]
- Costa, Hunter, Wayne, Waldman, Guccione, and McCulloch *A three-dimensional finite element method for large elastic deformations of ventricular myocardium. Part II: Prolate spheroidal coordinates* [CHW +94]

- Harrison *Kinetic Theory of Living Pattern* [Har93a]
- Kowalczyk and Kleiber *Modelling and numerical analysis of stresses and strains in the human lung including tissue-gas interaction* [KK94]
- LeVeque and Li *Immersed interface methods for Stokes flow with elastic boundaries or surface tension* [LL94]
- Lin and Taber *A Model for Stress-Induced Growth in the Developing Heart* [LT94]
- Moskowitz,
- Rogers and McCulloch *A collocation-Galerkin finite element model of cardiac action potential propagation* [RM94]
- Yang, Taber and Clark *A Nonlinear Poroelastic Model for the Trabecular Embryonic Heart* [YTC94]

Appendix L

Worksheets

L.1 Questions

This section contains various questions, some of which are points to be discussed with PPL and some of which are possible research topics.

- **Synchronisation of left and right ventricles**
  It is usually stated that the left and right ventricles work at the same time. Is this true? If there is a small time difference, is there an explanation for this?

- **Angular Momentum**
  The heart swivels during each beat. Is this caused by the angular momentum produced by the outflow of blood from the ventricles into the arteries?

- **Global balance**
  Can one derive information from the global balance of forces? See [[HSHY91], page 64 section 3.2.2] and [Mor80].

- **Law of Laplace**
  The inner wall of the left ventricle, the septum?, is concave (see [Lev91], Levick fig. 2.2.c, page 14). Can the law of Laplace still apply? See [Mor80].

- **Peskin's Formula**
  Peskin (Comm. P. Appl. Math. 89) page 99 compares his theory with results of Streeter et al. Is this correct?

- **Peskin's cross-sectional assumption**
  Peskin (Comm. P. Appl.. Math. 89) page 85 assumes that every fibre tube has constant cross-sectional area. Correct?

- **Influence of the pericardium** The pericardium restrains the heart. What is its role? According to Fung (Circulation, p. 34) "opening of the pericardium has an important effect on the shape and size of the heart". He mentions (also p. 34) the difference between surgery carried out with the pericardium open and closed.

- **Heart oscillation** PPL mentioned the measurement of heart beats by placing patients on a table.
- **Flow in the ventricle** How is the flow in the left ventricle influenced by the papillary muscles and by the clearly visible ridges on the inner surface (the endocardium)?
- **Stirling's law - time dependence** In Stirling's experiments, the pressure was changed, and the transitionary data were recorded. (see [[Lev91] p. 70]). The system is apparently damped. Can one model this?
- **Myocardial activation** In Hunter and Smaill p. 152 [[HS89]] experimental and numerical activation times are compared. It would be interesting to try to analyze some of the features such as the closed contours in Figure 27.
- **Heart deformation** Are there any results in the literature which model the heart deformation and blood pressure as functions of time, assuming simple contraction?
- **Systole expansion** During diastole the ventrical wall recoils ([[Lev91] p. 15 line -49]). Why?
- **Electrical activity** Can the upwards movement of the heart be explained by the fact, that the muscle is activated at slightly different times?
- **Right ventricle and atrium** These have not been considered as extensively, even though they handle the same amount of blood.
- **Biot's equations**?
- Coordination of veins and right atrium (PPL fragen).
- "Tectronic" compartments of heart walls (PPL fragen).
- The septum does not distort, but acts as though it were hinged.
- Woods - Role of t?
- Why ?
- Do surface fibres stay on surface?
- Where do "cross fibres" come from / go to?
- What are the "trabi" muscles?
- Are there fibre loops?
- Do chordae tendae really do work?
- What about 2 separate hearts?
- What about unrolling?
- What about apex?
- Magnetism
  1. Magnets? N S

2. rotating (earth)

- Can one think up better constitutive equations for passive myocardium? What about rational functions?
- How do the chorae tendineae transmit the force to the tricuspid and mitral valves? Do the papillary muscles / valves distend? Are the chordae tendineae optimally distributed?

L.2 Availability of Journals etc. in Münster

- **Amer. Heart J.**
  Vol. 79, 1970 - to date, MED ZQ 2170

- **Amer. J. Physiol**
  Split up into Series G, H etc.
  MED 2001 / ZD 1997 6/253 ie
  Institut fr Physiologie,
  Robert-Koch-Str. 27a

- **Annual Review of Physiology**
  MED

- **Anat. Rec**
  MED ZD 3653

- **Biophysical Journal**

Vol. 11	1971	-	to date	019 (Zoologische Institute, Badestr. 9, MS)
Vol. 1	1960/61	-	to date	UB ZD 355

- **Bull. math. Biology**
  ZD 1025

- **Circulation**

Vol. 1	1950	-	68 1983	006=ZQu 1262
Vol. 29	1964	-	date	6/029
Vol. 46	1972	-	date	6/236
Vol. 55	1977	-	72 1982	6/237

- **Circulation Research**
  - Vol. 26, 1970 - to date 029=Med. Klinik, Innere Medizin A-C, Albert-Schweizer-Str. 33, Münster. ZC 16

  - Vol. 9, 1961 - to date 254=Institute Physiologie II, Robert-Koch-Str. 28, Münster.

- **Computer Programs in Biomedicine**
  Vol. 6 1976 - 6/221 (Continued as Computer Methods and Programs in Biomedicine. - Not in Münster).

- **CRC Crit. Rev. Biomed Eng.**
  (Chemical Rubber Co.!). Not available in Münster.

- **Journal of Biomechanical Engineering**
  Not available in Münster.

- **Journal of Biomechanics**
  Vol. 1 1968 - to date 041 = Institut für exp. Biomechanik Domagkstr. 11, Münster.

- **J. Fluid Mechanics**
  ZD 4529

- **Journal of Thoracic Surgery**
  6/236
  Klinik und Poliklinik fr
  Thorax - Herz Gefchirurgie
  Albert-Schweizer-Str. 33

- **Literatur: Medizin**
  WG Kardiovaskuläres System in der UB.

- **Math. Bioscience**
  Not in Münster

- **Progress in Biophysics and molecular biology**
  - Vol. 16, 1966 - to date 019=Zoologische Institute, Badestr. 9, Münster.

- **Scand. J. Thoracic and Cardiov. Surg.**
  either ZD 1398 (all)
  or b/236 **18 - 21**

- **Scientific American**
  ZC4Ø7

- **Virchows Archiv**

Vol. 171	1903	-	Vol. 343 (1967)	UB ZD	
Vol. 207	1912	-	Vol. 343 (1967)	043 (Gerhard-Domagk-Institut für Pathologie, Domagkstr. 17, MS.	MED ZD 1983

- **Z. fuer Biologie**
  Vol. 1 (1865) - Vol.

- **Gegenbauers morph. J.**
  MED ZD 3597

- **Journal of Physiology** (London)
  MED Z 11311 and UB?

L.3 Literature Searched

1. **Bull. Math. Biology 55**
   1993 (ZD 1025)

2. **Cardiovascular Research 28**
   Jan. - June 1994

3. Circulation Research
   72 Jan.-June 1993
   - all -
   Must copy these pages:
   p. 631, 744, 795, 939, 297, 579, 607, 768, 1044

4. **Dissertation Abstracts (DA)** 1991 **51**
   No. 7, No. 8, No. 9, No. 10

5. **DA 1992 52** - all -

6. Index to Theses UK.
   411992 Part 1, Part 2, Part 3

7. **Am. J. Physiology**

8. **Am. J. Physiology**
   266:
   No.1 1994 Jan.
   NO.2 1994 Feb.
   No.3 1994 March

   262: all 1992.

L.4 Ph.D. Theses

The following theses have not been ordered (as yet):

1. "Finite analytic numerical solution of laminar and turbulent flow in a two-dimensional artificial heart model."
   S. H. Kim (Sang Hyun Kim),
   University of Iowa, 1991, 52 No. 7, p. 3743 B, DA 9136949.
   Advisor: K. B. Chandran
2. "The effects of right heart loading on left ventricular performance of in situ ejecting hearts.",
   E.B. Lankford,
   Johns Hopkins, 1991, 284 p., 52 No. 6, p. 33170B, DA 9132687.
   Advisor: not given
3. "Hemodynamics and wall shear rate measurements in the abdominal aorta of dogs.",
   K. C. White,
   Penn. State, 1991, 52 No. 4, p. 2167B, 214 p., DA 9127452.
   Advisor: John M. Tarbell
4. "Models of cardiac muscle contraction and relaxation.",
   J. L. Palladino,
   University of Pennsylvania, 1991, 51 No. 8, p. 3957B, 290 p., DA 9101196.
   Advisor: Abraham Noordergraaf
5. "Reconstructing fluid flow inside the heart from multiple x-ray image sequences.",
   N. H. Cornelius,
   Carnegie Mellon, 52 No. 12, p. 6523B, 167 p., DA 9216021.
   Advisor: not given
6. "Mathematical models of components of the cardiovascular system. Arterial baroreceptors and heart rate control",
   T. F. Itani, Northwestern, Nov. 1991, 52 No. 5, p. 2665B, 124 p., DA 9129007.
   Advisor: not given

Look if theses by following are available:

- Costa, Kevin D.
- Taber, Larry A.
- Burkett, D. A., DA 54, No. 6, 5964
- LeGrice, Ian J. *A Finite Element Model of Myocardial Structure: Implications for Electrical Activation in the Heart* [LeG92]
- Rustum Choksi, not in DA vol. 54.

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Appendix M

Biomathematics

General references on Biomathematics are:

- Winter *On the Stem curve of a tall palm in a strong wind* [Win93]

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