Faculty of Engineering

Department of Biomedical Engineering

Graduation Project

Left Ventricular Assist Devices

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ABSTRACT

The number of patients that are waiting for heart transplants far exceed the number of available donor hearts. Left Ventricular Assist Devices are mechanical alternatives that can help and are helping several patients. They work by taking blood from the left ventricle and ejecting that blood into the aorta. In the University of Louisville they are developing a similar device that will take the blood from the aorta instead of the ventricle. This new device they call an Artificial Vasculature Device. In this thesis the arterial system and AVD are modeled and a simple control algorithm for the AVD proposed. The arteries are modeled as a tube with linear resistance and inertia followed by a chamber with linear compliance and last a tube with linear resistance. The model is identical to the 4-element Windkessel model. The aortic valve is modeled as a drum that appear when the valve closes and disappear when it opens. The left ventricle is modeled as a compliance chamber with a constant compliance profile. The values for the resistances, inertias and compliances are identified using pressure and flow measurements from the ventricle and aortic root from a healthy patient. The AVD is modeled using common modeling structures for servo motors and simple structures for tubes and pistons. The values for the AVD could not be measured and identified so they are fetched from preliminary motor and part specifications. The control algorithm for the AVD uses a wanted load to create a reference aortic flow. This wanted aortic flow is then achieved by using a PI controller. With these models and controller the interaction between the modeled arterial system and AVD is investigated.

ABBREVIATIONS

VAD = Ventricular Assist Device.

AVD = Artificial Vasculature Device.

Notations

Rf1 = Fluid resistance in the tube from the aorta to the AVD.

Lf1 = Fluid inertia of the blood in the tube from the aorta to the AVD.

fp = Friction between the piston and container in the AVD.

mp = Mass of the piston in the AVD.

la = Length of the arm between the motor and the piston in the AVD.

Lw = Inductance in the windings in the servo motor in the AVD.

Rw = Resistance in the windings in the servo motor in the AVD.

b = Friction coefficient in the servo motor in the AVD.

J = Inertia in the servo motor in the AVD.

r = Gyration coefficient from current to torque

Ac = Area of the base of the cylinder container in the AVD.

btot = Total friction coefficient for the servo motor, piston and tube.

Jtot = Total inertia for the servo motor, piston and tube.

u = Voltage that drives the servo motor.

PA = Pressure in the aorta at the tube insertion point.

f = Frequency of the servo motor.

i = Current in the servo motor.

Qp = Flow in the tube to the AVD.

Vmin	=	Minimum volume of the AVD.
Vmax	=	Maximum volume of the AVD.
V	=	Volume of the AVD.
Tstat	=	Static friction in the servo motor in the AVD.
Ff_p_stat	=	Static friction between the piston and the container in the AVD.
Tstat_tot	=	Total static friction in the AVD.
Tdyn_const	=	The part of the dynamic friction in the AVD that is constant.
r	=	Gyrator factor in the servo motor in the AVD.
mbt	=	Mass of the blood in the tube to the AVD.
At	=	Area of the tube to the AVD.
?	=	Parameters for identification.
?c	=	Chosen parameters after identification.
e	=	Prediction error.
y(t|?)	=	Predicted value at time ‘t’ using parameters ?
t	=	Time.
y(t)	=	Measured value at time ‘t’.
QA	=	Flow at the root of the aorta.
Qend	=	Flow at the capillaries.
R1	=	Resistance in the beginning of the arteries.
C1	=	Compliance in the arteries.
P1	=	Pressure produced by the compliance in the models.

L1	=	Fluid inertia in the beginning of the arteries.
dAg	=	Guessed diameter of the aorta.
lAg	=	Guessed length of the aorta.
Pend	=	Pressure at the capillaries.
lAi	=	Identified length of the aorta.
R2	=	Resistance in the latter part of the arteries
X	=	Constant that relates the capillary flow and pressure to each other.
PLv	=	Pressure in the left ventricle.
PLv*	=	Approximated pressure in the left ventricle.
RA	=	Resistance in the root of the aorta.
LA	=	Fluid inertia in the root of the aorta.
lrAi	=	Identified length of the “root of the aorta”.
CV	=	Compliance of the closed aortic valve.
RV	=	Resistance of the closed aortic valve.
PV	=	Pressure after the aortic valve.
PVi	=	Pressure after the aortic valve’s compliance.
CLv	=	Compliance of the left ventricle.
VLv	=	Volume of the left ventricle.
RAV	=	Resistance of the closed added valve.
CAV	=	Compliance of the closed added valve.
RAVo	=	Resistance of the opened added valve.

QB = Flow after the added valve.

R1W = Wanted resistance instead of R1+RA.

L1W = Wanted fluid inertia instead of I1+IA.

C1W = Wanted compliance instead of C1.

R2W = Wanted resistance instead of R2+X.

QAW = Wanted aortic flow.

uI = Intake part of the voltage.

uE = Ejection part of the voltage.

u = Total voltage sent to the AVD

QPWE = Wanted flow into the AVD under the ejection phase

APPENDIX

Biomedical engineering

Biomedical engineering (BME) is the application of engineering principles and design concepts to medicine and biology for healthcare purposes (e.g. diagnostic or therapeutic). This field seeks to close the gap between engineering and medicine: It combines the design and problem solving skills of engineering with medical and biological sciences to advance healthcare treatment, including diagnosis, monitoring, and therapy.

Biomedical engineering has only recently emerged as its own study, compared to many other engineering fields. Such an evolution is common as a new field transitions from being an interdisciplinary specialization among already-established fields, to being considered a field in itself. Much of the work in biomedical engineering consists of research and development, spanning a broad array of subfields (see below). Prominent biomedical engineering applications include the development of biocompatible prostheses, various diagnostic and therapeutic medical devices ranging from clinical equipment to micro-implants, common imaging equipment such as MRIs and EEGs, regenerative tissue growth, pharmaceutical drugs and therapeutic biologicals.

Notable subdisciplines of biomedical engineering can be viewed from two angles, from the medical applications side and from the engineering side. A biomedical engineer must have some view of both sides. As with many medical specialties (e.g. cardiology, neurology), some BME sub-disciplines are identified by their associations with particular systems of the human body, such as:

Cardiovascular technology - which includes all drugs, biologics, and devices related with diagnostics and therapeutics of cardiovascular systems

Neural technology - which includes all drugs, biologics, and devices related with diagnostics and therapeutics of the brain and nervous systems

Orthopaedic technology - which includes all drugs, biologics, and devices related with diagnostics and therapeutics of skeletal systems

Those examples focus on particular aspects of anatomy or physiology. A variant on this approach is to identify types of technologies based on a kind of pathophysiology sought to remedy apart from any particular system of the body, for example:

Cancer technology - which includes all drugs, biologics, and devices related with diagnostics and therapeutics of cancer

But more often, sub-disciplines within BME are classified by their association(s) with other more established engineering fields, which can include (at a broad level):

Biochemical-BME, based on Chemical engineering - often associated with biochemical, cellular, molecular and tissue engineering, biomaterials, and biotransport.

Bioelectrical-BME, based on Electrical engineering and Computer Science - often associated with bioelectrical and neural engineering, bioinstrumentation, biomedical imaging, and medical devices. This also tends to encompass optics and optical engineering - biomedical optics, bioinformatics, imaging and related medical devices.

Biomechanical-BME, based on Mechanical engineering - often associated with biomechanics, biotransport, medical devices, and modeling of biological systems, like soft tissue mechanics.

One more way to sub-classify the discipline is on the basis of the products created. The therapeutic and diagnostic products used in healthcare generally fall under the following categories:

Biologics and Biopharmaceuticals, often designed using the principles of synthetic biology (synthetic biology is an extension of genetic engineering). The design of biologic and biopharma products comes broadly under the BME-related (and overlapping) disciplines of biotechnology and bioengineering. Note that "biotechnology" can be a somewhat ambiguous term, sometimes loosely used interchangeably with BME in general; however, it more typically denotes specific products which use "biological systems, living organisms, or derivatives thereof." [2] Even some complex "medical devices" (see below) can reasonably be deemed "biotechnology" depending on the degree to which such elements are central to their principle of operation.

Pharmaceutical Drugs (so-called "small-molecule" or non-biologic), which are commonly designed using the principles of synthetic chemistry and traditionally discovered using high-throughput screening methods at the beginning of the development process. Pharmaceuticals are related to biotechnology in two indirect ways: 1) certain major types (e.g. biologics) fall under both categories, and 2) together they essentially comprise the "non-medical-device" set of BME applications. (The "Device - Bio/Chemical" spectrum is an imperfect dichotomy, but one regulators often use, at least as a starting point.)

Devices, which commonly employ mechanical and/or electrical aspects in conjunction with chemical and/or biological processing or analysis. They can range from microscopic or bench-top, and be either in vitro or in vivo. In the US, the FDA deems any medical product that is not a drug or a biologic to be a "device" by default (see "Regulation" section). Software with a medical purpose is also regarded as a device, whether stand-alone or as part of another device.

Combination Products (not to be confused with fixed-dose combination drug products or FDCs), which involve more than one of the above categories in an integrated product (for example, a microchip implant for targeted drug delivery).

Tissue engineering

Tissue engineering, like genetic engineering (see below), is a major segment of Biotechnology - which overlaps significantly with BME.

One of the goals of tissue engineering is to create artificial organs (via biological material) for patients that need organ transplants. Biomedical engineers are currently researching methods of creating such organs. Researchers have grown solid jawbones and tracheas from human stem cells towards this end. Several artificial urinary bladders actually have been grown in laboratories and transplanted successfully into human patients. Bioartificial organs, which use both synthetic and biological components, are also a focus area in research, such as with hepatic assist devices that use liver cells within an artificial bioreactor construct.

Micromass cultures of C3H-10T1/2 cells at varied oxygen tensions stained with Alcian blue.

Genetic engineering

Genetic engineering, recombinant DNA technology, genetic modification/manipulation (GM) and gene splicing are terms that apply to the direct manipulation of an organism's genes. Genetic engineering is different from traditional breeding, where the organism's genes are manipulated indirectly. Genetic engineering uses the techniques of molecular cloning and transformation to alter the structure and characteristics of genes directly. Genetic engineering techniques have found success in numerous applications. Some examples are in improving crop technology (not a medical application, but see Biological Systems Engineering), the manufacture of synthetic human insulin through the use of modified bacteria, the manufacture of erythropoietin in hamster ovary cells, and the production of new types of experimental mice such as the oncomouse (cancer mouse) for research.

Neural engineering

Neural engineering (also known as Neuroengineering) is a discipline that uses engineering techniques to understand, repair, replace, or enhance neural systems. Neural engineers are uniquely qualified to solve design problems at the interface of living neural tissue and non-living constructs.

Pharmaceutical engineering

Pharmaceutical engineering is sometimes regarded as a branch of biomedical engineering, and sometimes a branch of chemical engineering; in practice, it is very much a hybrid sub-discipline (as many BME fields are). Aside from those pharmaceutical products directly incorporating biological agents or materials, even developing chemical drugs is

considered to require substantial BME knowledge due to the physiological interactions inherent to such products' usage. With the increasing prevalence of "combination products," the lines are now blurring among healthcare products such as drugs, biologics, and various types of devices.

Medical devices

This is an extremely broad category—essentially covering all health care products that do not achieve their intended results through predominantly chemical (e.g., pharmaceuticals) or biological (e.g., vaccines) means, and do not involve metabolism.

A medical device is intended for use in: the diagnosis of disease or other conditions, or in the cure, mitigation, treatment, or prevention of disease,

Two different models of the C-Leg prosthesis

Some examples include pacemakers, infusion pumps, the heart-lung machine, dialysis machines, artificial organs, implants, artificial limbs, corrective lenses, cochlear implants, ocular prosthetics, facial prosthetics, somato prosthetics, and dental implants.

Biomedical instrumentation amplifier schematic used in monitoring low voltage biological signals, an example of a biomedical engineering application of electronic engineering to electrophysiology.

Stereolithography is a practical example of medical modeling being used to create physical objects. Beyond modeling organs and the human body, emerging engineering techniques are also currently used in the research and development of new devices for innovative therapies, treatments, patient monitoring, and early diagnosis of complex diseases.

Medical devices are regulated and classified (in the US) as follows (see also Regulation):

Class I devices present minimal potential for harm to the user and are often simpler in design than Class II or Class III devices. Devices in this category include tongue depressors, bedpans, elastic bandages, examination gloves, and hand-held surgical instruments and other similar types of common equipment.

Class II devices are subject to special controls in addition to the general controls of Class I devices. Special controls may include special labeling requirements, mandatory performance standards, and postmarket surveillance. Devices in this class are typically non-invasive and include x-ray machines, PACS, powered wheelchairs, infusion pumps, and surgical drapes.

Class III devices generally require premarket approval (PMA) or premarket notification (510k), a scientific review to ensure the device's safety and effectiveness, in addition to the general controls of Class I. Examples include replacement heart valves, hip and knee joint implants, silicone gel-filled breast implants, implanted cerebellar stimulators, implantable pacemaker pulse generators and endosseous (intra-bone) implants.

Medical imaging

Medical/biomedical imaging is a major segment of medical devices. This area deals with enabling clinicians to directly or indirectly "view" things not visible in plain sight (such as due to their size, and/or location). This can involve utilizing ultrasound, magnetism, UV, other radiology, and other means.

An MRI scan of a human head, an example of a biomedical engineering application of electrical engineering to diagnostic imaging. Click here to view an animated sequence of slices.

Imaging technologies are often essential to medical diagnosis, and are typically the most complex equipment found in a hospital including:

Fluoroscopy

Magnetic resonance imaging (MRI)

Nuclear medicine

Positron emission tomography (PET) PET scansPET-CT scans

Projection radiography such as X-rays and CT scans

Tomography

Ultrasound

Optical microscopy

Electron microscopy

Implants

An implant is a kind of medical device made to replace and act as a missing biological structure (as compared with a transplant, which indicates transplanted biomedical tissue). The surface of implants that contact the body might be made of a biomedical material such as titanium, silicone or apatite depending on what is the most functional. In some cases implants contain electronics e.g. artificial pacemaker and cochlear implants. Some implants are bioactive, such as subcutaneous drug delivery devices in the form of implantable pills or drug-eluting stents.

Artificial limbs: The right arm is an example of a prosthesis, and the left arm is an example of myoelectric control.

A prosthetic eye, an example of a biomedical engineering application of mechanical engineering and biocompatible materials to ophthalmology.

Bionics

Artificial body part replacement is just one of the things that bionics can do. Concerned with the intricate and thorough study of the properties and function of human body systems, bionics may be applied to solve some engineering problems. Careful study of the different function and processes of the eyes, ears, and other the way for improved cameras, television, radio transmitters and receivers, and many other useful tools. These developments have indeed made our lives better, but the best contribution that bionics has made is in the field of biomedical engineering. Biomedical Engineering is the building of useful replacements for various parts of

the human body. Modern hospitals now have available spare parts to replace a part of the body that is badly damaged by injury or disease. Biomedical engineers who work hand in hand with doctors build these artificial body parts.

Clinical engineering

Clinical engineering is the branch of biomedical engineering dealing with the actual implementation of medical equipment and technologies in hospitals or other clinical settings. Major roles of clinical engineers include training and supervising biomedical equipment technicians (BMETs), selecting technological products/services and logistically managing their implementation, working with governmental regulators on inspections/audits, and serving as technological consultants for other hospital staff (e.g. physicians, administrators, I.T., etc.). Clinical engineers also advise and collaborate with medical device producers regarding prospective design improvements based on clinical experiences, as well as monitor the progression of the state-of-the-art so as to redirect procurement patterns accordingly.

Their inherent focus on practical implementation of technology has tended to keep them oriented more towards incremental-level redesigns and reconfigurations, as opposed to revolutionary research & development or ideas that would be many years from clinical adoption; however, there is a growing effort to expand this time-horizon over which clinical engineers can influence the trajectory of biomedical innovation. In their various roles, they form a "bridge" between the primary designers and the end-users, by combining the perspectives of being both 1) close to the point-of-use, while 2) trained in product and process engineering. Clinical Engineering departments will sometimes hire not just biomedical engineers, but also industrial/systems engineers to help address operations research/optimization, human factors, cost analysis, etc. Also see safety engineering for a discussion of the procedures used to design safe systems.

Schematic representation of a normal ECG trace showing sinus rhythm; an example of widely used clinical medical equipment (operates by applying electronic engineering to electrophysiology and medical diagnosis).

Regulatory issues

Regulatory issues have been constantly increased in the last decades to respond to the many incidents caused by devices to patients. For example, from 2008 to 2011, in US, there were 119 FDA recalls of medical devices classified as class I. According to U.S. Food and Drug Administration (FDA), Class I recall is associated to "a situation in which there is a reasonable probability that the use of, or exposure to, a product will cause serious adverse health consequences or death"

Regardless the country-specific legislation, the main regulatory objectives coincide worldwide.[7] For example, in the medical device regulations, a product must be:

1) Safe and

2) Effective

3) For all the manufactured devices

A product is safe if patients, users and third parties do not run unacceptable risks of physical hazards (death, injuries, …) in its intended use. Protective measures have to be introduced on the devices to reduce residual risks at acceptable level if compared with the benefit derived from the use of it.

A product is effective if it performs as specified by the manufacturer in the intended use. Effectiveness is achieved through clinical evaluation, compliance to performance standards or demonstrations of substantial equivalence with an already marketed device.

The previous features have to be ensured for all the manufactured items of the medical device. This requires that a quality system shall be in place for all the relevant entities and processes that may impacts safety and effectiveness over the whole medical device lifecyle.

The medical device engineering area is among the most heavily regulated fields of engineering, and practicing biomedical engineers must routinely consult and cooperate with regulatory law attorneys and other experts. The Food and Drug Administration (FDA) is the

principal healthcare regulatory authority in the United States, having jurisdiction over medical devices, drugs, biologics, and combination products. The paramount objectives driving policy decisions by the FDA are safety and effectiveness of healthcare products that have to be assured through a quality system in place as specified under 21 CFR 829 regulation. In addition, because biomedical engineers often develop devices and technologies for "consumer" use, such as physical therapy devices (which are also "medical" devices), these may also be governed in some respects by the Consumer Product Safety Commission. The greatest hurdles tend to be 510K "clearance" (typically for Class 2 devices) or pre-market "approval" (typically for drugs and class 3 devices).

In the European context, safety effectiveness and quality is ensured through the "Conformity Assessment" that is defined as "the method by which a manufacturer demonstrates that its device complies with the requirements of the European Medical Device Directive". The directive specifies different procedures according to the class of the device ranging from the simple Declaration of Conformity (Annex VII) for Class I devices to EC verification (Annex IV), Production quality assurance (Annex V), Product quality assurance (Annex VI) and Full quality assurance (Annex II). The Medical Device Directive specifies detailed procedures for Certification. In general terms, these procedures include tests and verifications that are to be contained in specific deliveries such as the risk management file, the technical file and the quality system deliveries. The risk management file is the first deliverable that conditions the following design and manufacturing steps. Risk management stage shall drive the product so that product risks are reduced at an acceptable level with respect to the benefits expected for the patients for the use of the device. The technical file contains all the documentation data and records supporting medical device certification. FDA technical file has similar content although organized in different structure. The Quality System deliverables usually includes procedures that ensure quality throughout all product life cycle. The same standard (ISO EN 13486) is usually applied for quality management systems in US and worldwide.

Implants, such as artificial hip joints, are generally extensively regulated due to the invasive nature of such devices.

In European Union, there are certifying entities named "Notified Bodies", accredited by European Member States. The Notified Bodies must ensure the effectiveness of the certification

process for all medical devices apart from the class I devices where a declaration of conformity produced by the manufacturer is sufficient for marketing. Once a product has passed all the steps required by the Medical Device Directive, the device is entitled to bear a CE marking, indicating that the device is believed to be safe and effective when used as intended, and, therefore, it can be marketed within the European Union area.

The different regulatory arrangements sometimes result in particular technologies being developed first for either the U.S. or in Europe depending on the more favorable form of regulation. While nations often strive for substantive harmony to facilitate cross-national distribution, philosophical differences about the optimal extent of regulation can be a hindrance; more restrictive regulations seem appealing on an intuitive level, but critics decry the tradeoff cost in terms of slowing access to life-saving developments.

RoHS II

Directive 2011/65/EU, better known as RoHS 2 is a recast of legislation originally introduced in 2002. The original EU legislation "Restrictions of Certain Hazardous Substances in Electrical and Electronics Devices" (RoHS Directive 2002/95/EC) was replaced and superseded by 2011/65/EU published in July 2011 and commonly known as RoHS 2. RoHS seeks to limit the dangerous substances in circulation in electronics products, in particular toxins and heavy metals, which are subsequently released into the environment when such devices are recycled.

The scope of RoHS 2 is widened to include products previously excluded, such as medical devices and industrial equipment. In addition, manufacturers are now obliged to provide conformity risk assessments and test reports – or explain why they are lacking. For the first time, not only manufacturers, but also importers and distributors share a responsibility to ensure Electrical and Electronic Equipment within the scope of RoHS comply with the hazardous substances limits and have a CE mark on their products.

IEC 60601

The new International Standard IEC 60601 for home healthcare electro-medical devices defining the requirements for devices used in the home healthcare environment. IEC 60601-1-11 (2010) must now be incorporated into the design and verification of a wide range of home use and point of care medical devices along with other applicable standards in the IEC 60601 3rd edition series.

The mandatory date for implementation of the EN European version of the standard is June 1, 2013. The US FDA requires the use of the standard on June 30, 2013, while Health Canada recently extended the required date from June 2012 to April 2013. The North American agencies will only require these standards for new device submissions, while the EU will take the more severe approach of requiring all applicable devices being placed on the market to consider the home healthcare standard.

Training and certification

Biomedical engineers require considerable knowledge of both engineering and biology, and typically have a Master's (M.S.,M.Tech, M.S.E., or M.Eng.) or a Doctoral (Ph.D.) degree in BME (Biomedical Engineering) or another branch of engineering with considerable potential for BME overlap. As interest in BME increases, many engineering colleges now have a Biomedical Engineering Department or Program, with offerings ranging from the undergraduate (B.Tech,B.S., B.Eng or B.S.E.) to doctoral levels. As noted above, biomedical engineering has only recently been emerging as its own discipline rather than a cross-disciplinary hybrid specialization of other disciplines; and BME programs at all levels are becoming more widespread, including the Bachelor of Science in Biomedical Engineering which actually includes so much biological science content that many students use it as a "pre-med" major in preparation for medical school. The number of biomedical engineers is expected to rise as both a cause and effect of improvements in medical technology.

In the U.S., an increasing number of undergraduate programs are also becoming recognized by ABET as accredited bioengineering/biomedical engineering programs. Over 65 programs are currently accredited by ABET.

In Canada and Australia, accredited graduate programs in Biomedical Engineering are common, for example in Universities such as McMaster University, and the first Canadian undergraduate BME program at Ryerson University offering a four-year B.Eng program. The Polytechnique in Montreal is also offering a bachelors's degree in biomedical engineering.

As with many degrees, the reputation and ranking of a program may factor into the desirability of a degree holder for either employment or graduate admission. The reputation of many undergraduate degrees are also linked to the institution's graduate or research programs, which have some tangible factors for rating, such as research funding and volume, publications and citations. With BME specifically, the ranking of a university's hospital and medical school can also be a significant factor in the perceived prestige of its BME department/program.

Graduate education is a particularly important aspect in BME. While many engineering fields (such as mechanical or electrical engineering) do not need graduate-level training to obtain an entry-level job in their field, the majority of BME positions do prefer or even require them. Since most BME-related professions involve scientific research, such as in pharmaceutical and medical device development, graduate education is almost a requirement (as undergraduate degrees typically do not involve sufficient research training and experience). This can be either a Masters or Doctoral level degree; while in certain specialties a Ph.D. is notably more common than in others, it is hardly ever the majority (except in academia). In fact, the perceived need for some kind of graduate credential is so strong that some undergraduate BME programs will actively discourage students from majoring in BME without an expressed intention to also obtain a masters degree or apply to medical school afterwards.

Graduate programs in BME, like in other scientific fields, are highly varied, and particular programs may emphasize certain aspects within the field. They may also feature extensive collaborative efforts with programs in other fields (such as the University's Medical School or other engineering divisions), owing again to the interdisciplinary nature of BME. M.S. and Ph.D. programs will typically require applicants to have an undergraduate degree in BME, or

another engineering discipline (plus certain life science coursework), or life science (plus certain engineering coursework).

Education in BME also varies greatly around the world. By virtue of its extensive biotechnology sector, its numerous major universities, and relatively few internal barriers, the U.S. has progressed a great deal in its development of BME education and training opportunities. Europe, which also has a large biotechnology sector and an impressive education system, has encountered trouble in creating uniform standards as the European community attempts to supplant some of the national jurisdictional barriers that still exist. Recently, initiatives such as BIOMEDEA have sprung up to develop BME-related education and professional standards. Other countries, such as Australia, are recognizing and moving to correct deficiencies in their BME education. Also, as high technology endeavors are usually marks of developed nations, some areas of the world are prone to slower development in education, including in BME.

Licensure/certification

Engineering licensure in the US is largely optional, and rarely specified by branch/discipline. As with other learned professions, each state has certain (fairly similar) requirements for becoming licensed as a registered Professional Engineer (PE), but in practice such a license is not required to practice in the majority of situations (due to an exception known as the private industry exemption, which effectively applies to the vast majority of American engineers). This is notably not the case in many other countries, where a license is as legally necessary to practice engineering as it is for law or medicine.

Biomedical engineering is regulated in some countries, such as Australia, but registration is typically only recommended and not required. In the UK, mechanical engineers working in the areas of Medical Engineering, Bioengineering or Biomedical engineering can gain Chartered Engineer status through the Institution of Mechanical Engineers. The Institution also runs the Engineering in Medicine and Health Division.

The Fundamentals of Engineering exam - the first (and more general) of two licensure examinations for most U.S. jurisdictions—does now cover biology (although technically not

BME). For the second exam, called the Principles and Practices, Part 2, or the Professional Engineering exam, candidates may select a particular engineering discipline's content to be tested on; there is currently not an option for BME with this, meaning that any biomedical engineers seeking a license must prepare to take this examination in another category (which does not affect the actual license, since most jurisdictions do not recognize discipline specialties anyway). However, the Biomedical Engineering Society (BMES) is, as of 2009, exploring the possibility of seeking to implement a BME-specific version of this exam to facilitate biomedical engineers pursuing licensure.

Beyond governmental registration, certain private-sector professional/industrial organizations also offer certifications with varying degrees of prominence. One such example is the Certified Clinical Engineer (CCE) certification for Clinical engineers.

Founding figures

Leslie Geddes (deceased)- Professor Emeritus at Purdue University, electrical engineer, inventor, and educator of over 2000 biomedical engineers, received a National Medal of Technology in 2006 from President George Bush for his more than 50 years of contributions that have spawned innovations ranging from burn treatments to miniature defibrillators, ligament repair to tiny blood pressure monitors for premature infants, as well as a new method for performing cardiopulmonary resuscitation (CPR).

Y. C. Fung - professor emeritus at the University of California, San Diego, considered by many to be the founder of modern Biomechanics.

Robert Langer - Institute Professor at MIT, runs the largest BME laboratory in the world, pioneer in drug delivery and tissue engineering.

Herbert Lissner (deceased) - Professor of Engineering Mechanics at Wayne State University. Initiated studies on blunt head trauma and injury thresholds beginning in 1939 in collaboration with Dr. E.S. Gurdjian, a neurosurgeon at Wayne State's School of Medicine. Individual for whom the American Society of Mechanical Engineers' top award in Biomedical Engineering, the Herbert R. Lissner Medal, is named.

1 Introduction

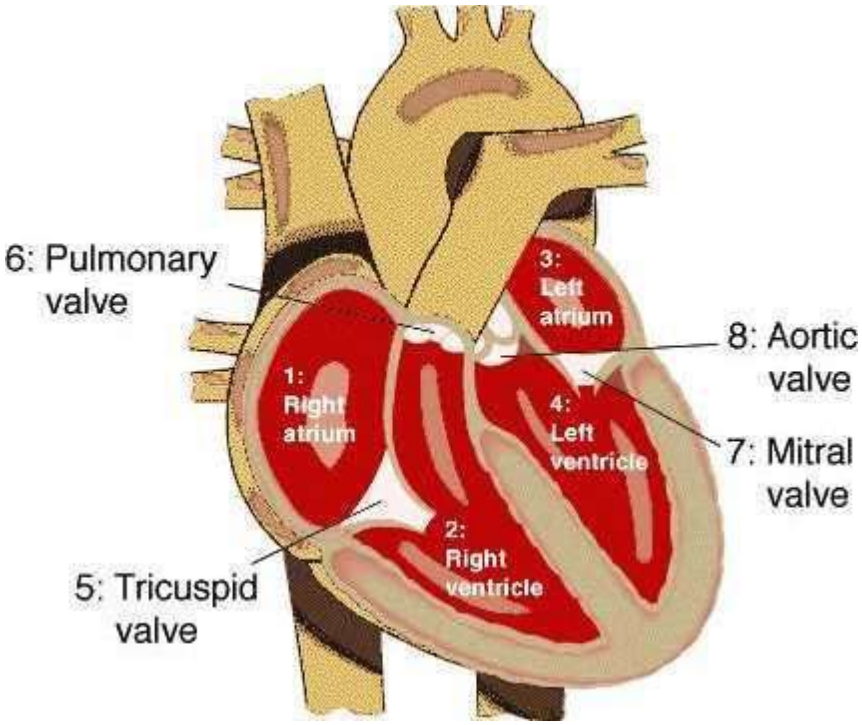
1.1 Background

Congestive heart failure is the cause of 39,000 deaths a year and is a contributing factor in another 225,000 deaths. Pharmacological therapies can prolong the life of a patient and even cure in many cases, but for many this treatment is not enough. An estimated 30 000 to 60 000

people each year in the US alone could benefit from having heart transplants and for all those there are less than 3000 donor hearts available 1.In those cases the hearts have become too weak to eject blood there are mechanical alternatives such as Ventricular Assist Devices (VAD) to help alleviate the shortage 2. They are primarily used as bridges to transplants, implanted in patients who would otherwise not survive until a heart is available. A VAD partly takes over the pumping by assisting one or both ventricles of the heart.

The ventricles are the parts of the heart that eject the blood out of the heart. Oxygenated blood comes from to the lungs, gets stored in the left atrium, transferred to the left ventricle and pushed out into the aorta by the contraction of the muscles in the wall of the left ventricle. When the left ventricle is filling with blood the aortic valve is closed to prevent backflow from the aorta and when the left ventricle is ejecting blood the mitral valve is closed to prevent backflow into the left atrium. The aorta branches out into a multitude of arteries that lead the blood to the capillaries where the oxygen gets transferred to the cells. The non oxygenated blood then gets pumped trough the veins, stored in the right atrium, transferred to the right ventricle and pushed into the lungs by the right ventricle in the same way as the left ventricle does. In the lungs the blood gets oxygenated and finally returns to the left atrium. Figure 1 shows the heart with named parts.

The heart with named parts.



There are many reasons for a ventricle to become weaker than it needs to be but basically they can be divided into two. The first is that the strength of the ventricle has deteriorated from sickness or injury. The other is that the load of the blood vessels has increased from clogging. The effect of the weaker ventricle is that the volume in it increases as it can't eject what it wants. This increase causes the pressure in the ventricle to rise as well which enables it to eject more. Eventually an equilibrium is reached with a higher than normal ventricle volume.

It is more work to push blood down the aorta than to the lungs so the left ventricle has a harder job and therefore is the one that most often is in need of assistance. Because the left ventricle fails more often than the right one there are more left VADs than right ones and it is the primary research subject. A VAD assists a ventricle by taking blood from that ventricle and ejecting that blood into the blood vessel leading from the ventricle; the left VAD takes blood from the left ventricle and ejects into the aorta. The assistance results in that the ventricle experiences a lower load which in turn makes it easier for it to pump and reduces its volume.

VADs can be divided into external and internal depending on if the actual device is implanted inside the body or not. With external ones the tubes that lead the blood to and from the

device pierce the skin. With internal ones the tubes does not need to go through the skin but the device still needs to be in contact with the outside world for power supply and control reasons.

Generally the more of the device that is implanted inside the body and the fewer things pierce the skin the better the quality of life becomes for the patient, but also the more expensive it becomes.

Another way of dividing VADs is into continues and pulsatile depending on how they work. Continues VADs keep the blood flowing at a constant speed through the tubes. Pulsatile ones take in blood while the ventricle ejects, stores it and then ejects it while the ventricle is filling up. There are internal and external ones of both types.

With progresses made the newer VADs are more and more being considered as end state solutions and not just bridges to transplants. Coatings that have a lesser risk of being rejected and fewer things piercing the skin that can cause infections mean that the VADs can be implanted longer. Better constructions with less wear means that the VADs life time is longer and it doesn't need to be replaced as often or at all. Smaller batteries with longer life time and less energy consuming constructions mean that the patients can go longer between recharges and has to carry less weight which improves the quality of life. Many patients are ineligible for heart transplants due to other afflictions so for these a permanent VAD is the best solution.

While waiting for transplants and being assisted by VADs a small number of patients have recovered from their illnesses and have had their devices explanted without getting a heart transplant. That a few patients' hearts can recover by them self while "resting" under the assistants of a VAD suggests that more patients can be cured in this way.

1.2 Purpose of thesis

That people might be cured under the assistance of a VAD has prompted the University of Louisville and professor Steve Koenig to start building an Artificial Vasculature Device so that they can study the effects of different "rest" and "rehabilitation" conditions for the heart3. The idea is to alter the load seen by the heart in a similar way a VAD does by implanting the AVD in parallel with the arteries. By lowering the load the hearts ventricle does not have to push as hard to eject and it can rest and by raising the load again the hearts gets more exercise. This way the heart can be rehabilitated in a similar fashion to the rehabilitation of other muscles when they have been injured. The purpose of this thesis is to build a preliminary computer model in order to gain a better understanding of how the AVD will work and interact with the body. Building the model will include modeling the arterial system, modeling the AVD and constructing a simple control algorithm for the AVD. As the device has not been built and real measurements can't be obtained the model will only give a rough understanding of how the actual system will work. The detailed values will be wrong but the general behavior should be correct enough. After the construction of the device is done the model can be improved from observations to include interactions that could not be foreseen.

The computer model will be implemented in MatlabR and SimulinkR. SimulinkR is chosen because it is simple and graphical and therefore might be easier to understand for someone not used to computer programming.

1.3 Thesis outline

The 'Introduction' section includes some background to why the AVD is being designed and also the purpose of this thesis.

The 'Modeling' section includes models of the arterial system and the AVD. It also tells something about how these models where achieved.

The ‘Controllers’ section is about the controller; description of the design of the controllers, proof of stability and plots from simulations to show how the effect of the controller and AVD. The pre sampling filtering is also discussed.

The ‘Simulations with different wanted loads’ section contains plots from different simulations.

The ‘Result’ section tells of the results of the built model and simulations using it.

The ‘Concluding remarks’ section sums up the most important results of this thesis and also contains what further work can be done with this thesis.

Lastly the ‘Appendix’ contains the MatlabR code and SimulinkR schematics to identify the model parameters and run the simulations.

2 Modeling

This chapter contains explanations of how the models were constructed and how the numerical values for them were obtained. To model the AVD attached to the aorta the system is divided into three separate systems; one of the AVD alone, one of the unassisted arterial system and one of the valve that is added in the aorta. These three systems are modeled separately and then connected to achieve a model of the total system.

All models are based on physical modeling, which means that the transfer functions have variables and constants that can be traced to combinations of ideal physical attributes such as flow, mass and resistance. The biggest advantage with using this type of modeling is that everything can be explained. Small subsystems can be compared to the actual physical subsystems they represent. Using black boxes might produce better simulated signals compared to the data but they can’t be divided and compared to the physical systems. Also if a physical model can produce accurate signals in simulations it’s a validation that the beliefs of how the actual system works are correct.

The equations that make up the physical model are derived using bond graphs4; these are a way to graphically build up and present physically based models. When the bond graph

includes everything that is thought to be significant it is easily translated into differential equations.

The numerical values of the model parameters can either be obtained from measurements of the individual constants that make up the parameters or from identification experiments on bigger systems. When identifying parameters from experiments the structure derived from the physical model has to be conserved otherwise the parameters can't be traced back to the physical constants. If a parameter's value can be obtained using both methods and the measured value is much different from the identified one something is wrong. Either the measurements are faulty or the model needs to be adjusted. If the values on the other hand are close then this is a validation that the model is correct.

2.1 Description of the proposed AVD

The proposed Artificial Vasculature Device is being designed to work with the left ventricle and assist it rather than the right ventricle. The decision to work with the left ventricles is because they fail much more often than the right ventricles. In any case there should not be a big problem to adjust or alter the device to assist right ventricles if this is needed in future studies.

The AVD is very similar to a pulsatile Left VAD in that they both take in blood when the left ventricle is ejecting causing the heart to see a lesser load, storing the blood and ejecting when the ventricle is filling up again. Both types eject their blood into the aorta, so they both require that a hole is made there and a tube inserted. Both types make the left ventricle see a lesser load and "rest" but the VAD takes the blood in passively while the AVD will be able to adjust the blood taken in actively. Another difference is where the blood is taken from; the Left VAD takes its blood from the left ventricle while the AVD takes its blood from the aorta. This means that the Left VAD requires a hole to be made in the left ventricle and a tube inserted there. The AVD on the other hand doesn't need a hole to be made there at all. With the AVD the same hole made for the ejection tube can be used for the intake tube, the tubes just need to be connected just before the insertion point in the aorta. To make taking blood from the aorta work well an extra valve needs to be implanted in the aorta down flow of the intake tube insertion point. The valve

stops backflow in the aorta when the AVD is taking in blood. The picture below in figure 2 shows a pulsatile Left VAD implanted in a human body, the AVD would be implanted in the same way except for that the intake tube could be taken away completely or moved to be inserted into the aorta beside the ejection tube.

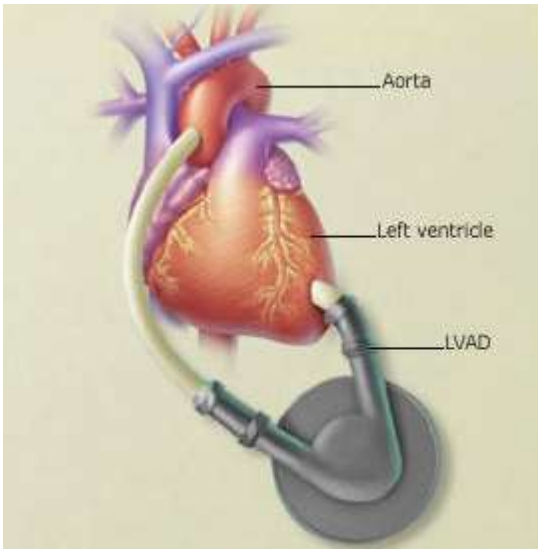


Figure 2: Implanted pulsatile left ventricular assist device.

The proposed AVD will be constructed by modifying the commercially available HeartMate. The HeartMate is a pulsatile Left VAD that has successfully been used to assist failing left ventricles for several years. The intake and ejection tube will both remain with the difference that the intake tube will be inserted into the aorta beside the ejection tube instead of the left ventricle. The original HeartMateTM has passive mechanical valves in the intake and ejection tubes to prevent backflow from the aorta to the device and from the device to the left ventricle. Even with these two valves another valve has to be added inside the aorta down flow of the tubes to prevent backflow from the arteries to the intake tube. With the two tubes still working as one intake and one ejection there will still be circulation of the blood that goes into the AVD. The circulation means that all the parts of blood taken from the aorta will within a few beats return to the aorta. If the valves in the tubes were removed and the AVD only took in a small amount of blood then that exact blood would be the blood that gets ejected and the rest of the blood in the tubes would remain. Blood remaining in place like that is not good for the body.

The AVD prototype will primarily be used in research on animals so whether or not it is internal or external doesn't matter. Even if it can be fully implanted it might be easier to leave it external so it can be tampered with easier.

2.2 Modeling the AVD

The proposed AVD consists of two tubes that connect the AVD to the aorta for intake and ejection of blood, a container for storing blood, a piston to regulate the container volume, a servo motor to move the piston and an arm that links the piston to the servo motor. The model for this AVD is presented in figure 3. There is also a possibility to add gears for the servo motor.

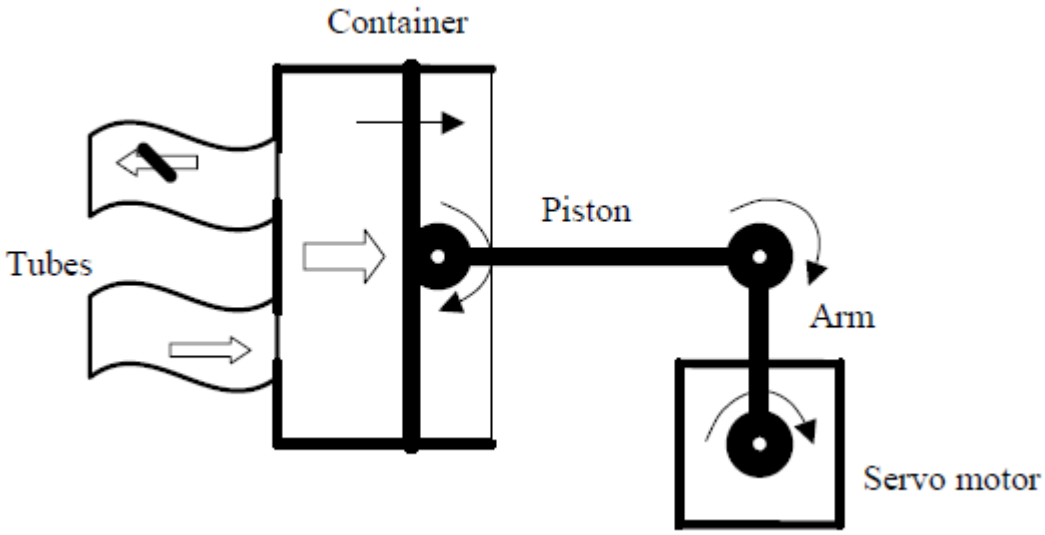


Figure 3: Model of the AVD.

It is assumed that the valves in the tubes are still there and that the added valve is placed down flow of them both. The two tubes therefore function as one without valve so the model only needs to contain one tube. As the AVD has not yet been built no measurements can be preformed on it. Instead the model is based on commonly made approximations and realistic numerical values for the physical constants, the servo motors characteristics are fetched from a

motor that is being considered to be used for the prototype; the A0400-102-4-000 by ‘Applied Motion Products’6.

2.2.1 Developing a model

The approximations made are first that the tube and the container both are perfect cylinders with rigid walls and that the blood is non compressive. This means that the filling of the container is linear and that there are no capacitive (flow storing) elements. The fluid friction ‘Rft‘ and fluid inertia ‘Lft‘ of the blood in the tube and the friction coefficient between the piston and container ‘fp‘ and the mass of the piston ‘mp‘ are all included in the model. The next approximation made is that the arm that links the servo motor to the piston gives a linear transformation of torque to force and angular velocity to velocity and that it has no mass. Other more linear and better solutions for the transfer from motor to piston are possible but using an arm is simplest both to make and explain in a model

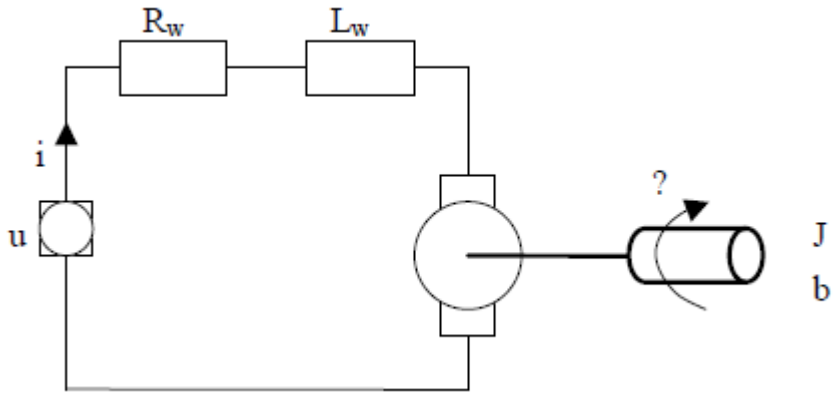


Figure 4: Model of the servo motor.

The servo motor is as a whole approximated as most servo motors and the model for it is presented in figure 4. The model has inductance ‘Lw’ and resistance ‘Rw’ in the windings,

friction coefficient ‘b’ and inertia ‘J’ in the rotor and that the gyration coefficient ‘r’ from voltage to angular frequency and torque to current is linear7.

If static friction and limits in the containers volume are ignored a linear model can be constructed using the physical constants and relationships above. The model of the whole AVD is presented in figure

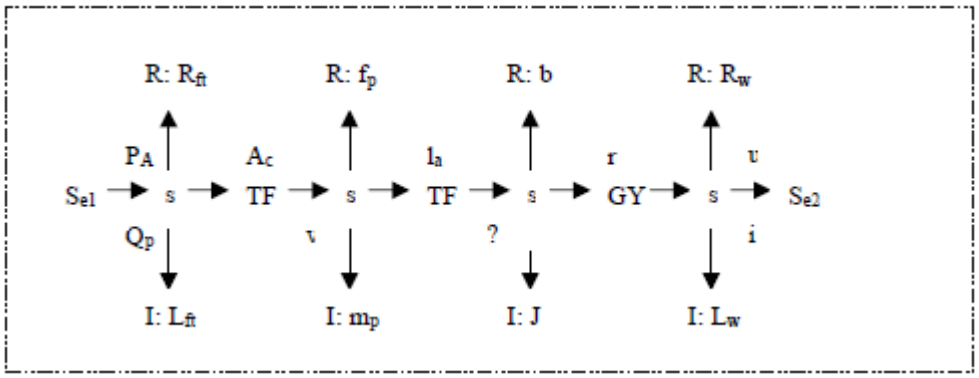


Figure 5: Bond graph of the AVD

To make the linear model causal all the different resistive and inductive (effort storing) elements of the motor, piston and blood are added into one resistive and one inductive element as shown below.

$$b_{tot} = b + f_p(l_a)^2 + R_{ft}(l_a A_c)^2$$
$$J_{tot} = J + m_p(l_a)^2 + L_{ft}(l_a A_c)^2$$

The model is causal if the voltage to the motor ‘u’ and the pressure at the end of the tubeinserted in the aorta ‘PA‘ are taken as the inputs (sources) and ‘Qp‘ the flow in the tubethe output. The causal version of the bond graph is presented in figure 6.

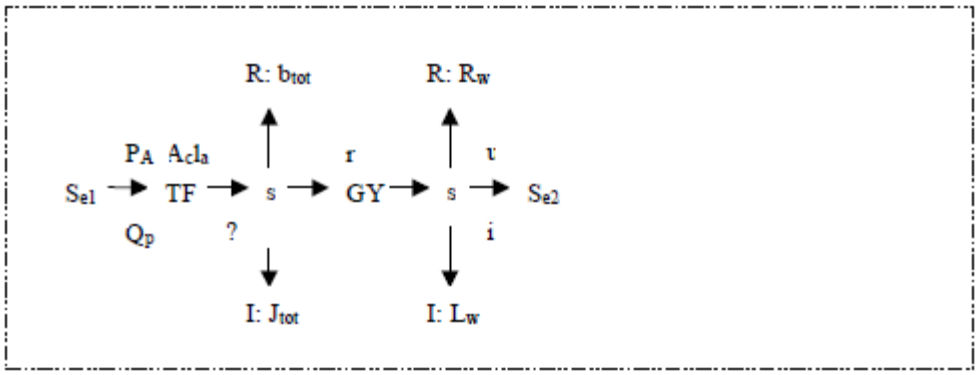


Figure 6: Causal bond graph of the AVD

The equations for the causal system are as follows;

$$\frac{d\omega(t)}{dt} = \frac{P_A(t) l_a A_c - i(t) r - \omega(t) b_{tot}}{J_{tot}}$$

$$\frac{di(t)}{dt} = \frac{\omega(t) r - u(t) - i(t) R_w}{L_w}$$

$$Q_p(t) = \omega(t) l_a A_c \text{ (SI-units)}$$

In the equations '? ' is the angular velocity of the servo motor and 'i' the current in the servo motor.

The linear and causal model can now be expanded to include the limits in the container's volume and the static friction. That the physical container must have a maximum volume could easily be ignored by assuming that the container is big enough to hold any possible stroke volume. That it must have a minimum volume is on the other hand not as easy to ignore. The assumption that the AVD never ejects fully and that it works with a buffer to avoid that the piston hits the end of the container can be made. Doing this requires that the controller that ejects the blood can guarantee it won't allow the piston to hit the end of the container, which makes for a more complex controller. It would have to stop at approximately the same spot every time

without drifting, do it without generating too much backflow and still be fast enough. This might very well be what will be desired of the controller in the end product; the piston hitting the end of the container might cause wear and there will be blood left in the tube anyway no matter how well the container is emptied. However, a computer model is better the more things it can explain and it shouldn't depend on the design of the controller, especially not if the model will be used when designing this controller. A model that includes a minimum volume 'Vmin' and can explain the piston hitting the end of the container is therefore preferred. The minimum volume is included by not allowing a negative flow when the volume 'V' is or reaches zero. Also the internal state of the stored effort caused by the inertia is set to zero and kept there until a positive flow is produced. Even though the maximum volume 'Vmax' could be ignored as stated above there is no reason to when it is easily included in the same manner as the minimum volume.

The differences being that a positive flow is not allowed when the volume is at the maximum and that the internal state is forced to and kept at zero until a negative flow is produced. The limited volume changes the equation for the angular velocity given above. The new equation for the angular velocity is presented below.

$$\frac{d\omega(t)}{dt} = \begin{cases} 0\ ,\ V(t) \le V_{min}(t)\ \&\ \dfrac{P_A(t) l_a A_c - i(t) r - \omega(t) b_t}{J_t} \le 0 \\ 0\ ,\ V(t) \ge V_{max}(t)\ \&\ \dfrac{P_A(t) l_a A_c - i(t) r - \omega(t) b_t}{J_t} \ge 0 \\ \dfrac{P_A(t) l_a A_c - i(t) r - \omega(t) b_t}{J_t}\ ,\ otherwise \end{cases}$$

The static friction in the motor 'Tstat' and between the piston and the container 'Ff_p_stat' added together into one static friction 'Tstat_tot' in the same way that the resistances and inertias were when creating the linear and causal model. It is assumed that the static friction between the blood and the tube is zero and therefore it is ignored.

$$T_{stat\_tot} = T_{stat} + F_{f\_p\_stat}(l)^2$$

The total static friction is included in the model when the motor is still by adding an opposite torque of the same size but less than the maximum static friction to the dynamic

friction. When the motor is moving a different torque is added in the opposite direction of the angular velocity. This torque represents the part of the dynamic friction that is constant 'Tdyn_const'. It is set to the same value as the maximum static friction to minimize the discontinuities when the motor starts and stops moving. The equations for the static friction are shown below.

$$total\_friction(t) = b_{tot}\omega(t) + \max(\min(P_A(t)l_a A_c - i(t)r, T_{stat\_tot}), -T_{stat\_tot}),$$
$$\omega(t) = 0$$
$$total\_friction(t) = b_{tot}\omega(t) + sign(\omega(t))T_{dyn\_const},\ \omega(t) \neq 0$$
$$T_{dyn\_const} = T_{stat\_tot}$$

2.2.2 Finding the numerical values

The numerical values for the different physical constants need to be found using different ways than measurements as stated above. For the servo motor there is an easy and accurate way; using the manufacturer's datasheet. The datasheet gives values of an average motor of the same series as proposed to be used in the AVD prototype; the A0400-102-4-000 by 'Applied Motion Products'6. Any specific motor would only be marginally different from the average one. In the datasheet all but one of the needed values are given; the winding resistance and inductance, the motors inertia ('Rotor Inertia' in the datasheet), the motors static friction ('Friction Torque' in the datasheet) and the gyrator factor 'r' ('Voltage constant' in the datasheet). The value not given is the dynamic friction. This is instead taken from the data sheet of a similar motor; the N-2304-1 by 'Rockwell Automation'8. The two motors have about the same inertia and static friction so the dynamic friction should be in the same range. The motor constants are shown in table 1.

Physical constant	Numerical value
Winding resistance, R_w	2.4 [Ohm]
Winding inductance, L_w	9.0 [mH]
Inertia, J	$3.6*10^{-5}$ [kg m^2]
Static friction, T_{stat}	0.04 [N m]
Dynamic friction, b	0.034 [N m / krpm] = $3.25*10^{-4}$ [N m / (rad / s)]
Voltage constant, r	43.6 [V / krpm] = 0.42 [V / (rad / s)]

For the rest of the AVD the values have to be based on assumptions. The inertia of the blood in the tube is found by seeing the blood in the tube as a weight consisting of two pistons between fluids with no mass. The mass of the weight is the same as the mass of the blood ‘mbt’ inside the tube in the original physical configuration. This mass is then transformed into fluid inertia in the same way that the fluid inertia was transformed into inertia when moving it to make the model causal above. The transformation goes the other way now so the mass is divided by the square of the base area of the tube ‘At’. The mass of the blood have now been moved back to the fluid and the pistons can be removed which leaves the original configuration with the correct inertia value. By approximating that blood weighs 1kg per liter and by assuming a likely tube size the mass of the blood inside it can easily be calculated. With a 15 cm long tube ‘lt’ that has a base area of 1,77*10-4 m2 (diameter 0.015 m) the blood’s mass becomes 26,5 g. The equations to generate the fluid inertia of the blood in the tubes are given below.

$$L_{ft} = \frac{m_{bt}}{A_t^2}$$

$$m_{bt} = 1000 l_t A_t$$

These values give a fluid inertia of around 8*105 kg/m4. When this is moved to the inertia of the motor it becomes 0,018 kgm2. This value is much larger than the value of the motor’s inertia of 3,6*10-5 kgm2.

The piston’s mass is given the reasonable value of 100 g. The true value should not be much larger and as shown below the value doesn’t really matter in comparison with the bloods fluid inertia so it can be considered accurate enough.

With all the inductive values found they can be compared by moving them to the motor’s inertia as described in section ‘2.2.1. Developing a model’. The inertia values are shown in table 2.

Moved inertia Numerical value

Moved inertia	Numerical value
Motor inertia, J	$3.6*10^{-5}$ [kg m^2]
Piston inertia moved	$4*10^{-5}$ [kg m^2]
Tube blood inertia moved	0.018 [kg m^2]

Table 2: Moved inertia values.

The blood's fluid inertia clearly is the dominant of the three. The reason the pistons mass and the motors inertia is so much smaller when compared to the bloods fluid inertia is the difference in area between the tube and the container.

The differences in the inertias of the different parts are used when guessing the frictions. The area difference and arm length that makes the blood's fluid inertia so much larger than the motors inertia should also make the blood's friction much larger than the motors and pistons. Since the piston inertia is about the same size of the inertia in the motor the friction is also made about the same size. The friction of the blood in the tube is made 100 times larger than the motor's even though the inertia is 1000 times larger, this cause with a smooth tube the friction should be small. The resistance values are shown in table 3.

Moved resistance	Numerical value
Motor resistance, b	$3.25*10^{-4}$ [N m / (rad / s)]
Piston resistance moved	$4*10^{-4}$ [N m / (rad / s)]
Tube blood resistance moved	0.028 [N m / (rad / s)]

2.2.3 Comments

The values for the piston and blood are very uncertain; the values for the friction of these parts are just guesses. Measurements need to be made to get accurate parameter values in the model. However; even though the values are not perfect the model should still give a reasonably good approximation of the behavior of the AVD. Also, trying different values in the model and

running simulations can help in designing the AVD. From the calculations above it is clear that the design of the tube has great impact on the behavior and that the motor has much less of an impact. A wider tube gives less friction and inertia seen by the motor but it also means more stored blood.

Inserting gears for the motor changes the way the friction and inertia gets moved to the motor. The different values get closer in size with gears that mimic a shorter arm.

2.3 Modeling the systemic arterial system

The modeling of the systemic arterial system is done by defining physical models and then seeing these as greybox models. By using greybox models it is possible to conserve the structure derived from the physical modeling. The parameter values are identified using measured data from a healthy human. This measured data includes aortic pressure, aortic flow and left ventricle pressure.

To obtain a good model of the systemic arterial system with the AVD attached it is necessary to have one model of the part of the aorta that is up-flow and one model of part of the aorta that is down-flow of the AVD tube insertion. This is possible to make if the tube is assumed to be inserted at the same point where the aortic pressure measurements were made. The model of the part of the aorta that is down-flow of the AVD tube is therefore achieved by modeling the part of the aorta that is down-flow of the aortic pressure measurements and the up-flow model is achieved by modeling the part that is between the two pressure measurements. To handle the nonlinearity of the aortic valve the whole arterial system is divided into two models; one for when the valve is opened and one for when the valve is closed.

By adding the AVD to the systemic arterial system and producing a lesser load for the heart the left ventricle pressure and aortic flow should both be affected so neither one can be used as a driving factor for the total model. The only thing that can be done is to assume that the left ventricle compliance profile remains the same and use this as a driving factor. Any changes to the compliance profile due to the lesser load are impossible to predict and can really only

be investigated by letting the heart pump in to different lower loads. This means that to keep the profile the same is as valid an assumption as any other without data from different load conditions.

2.3.1 Greybox identification

When identifying parameters in a model derived from physical modeling it is important that the structure is conserved. This is achieved by using greybox models that have the parameters '?=[?1, ?2 ,,, ?N]T' that can be fixed or let lose. The parameters that are let lose to be identified can also be linked to each other so that a physical constant that appears in more than one place still only receives a single value. The parameter values chosen '?c' when identifying are those that minimize the prediction error 'e' for the prediction 'y(t|?)' of the measured value 'y(t)' according to the following equations4.

$$\varepsilon(t,\theta) = y(t) - \hat{y}(t \mid \theta)$$
$$\theta_c = \arg\min_{\theta} \frac{1}{N} \sum_{t=1}^{N} \varepsilon^2(t,\theta)$$

The MatlabR command 'pem' in the Identification Toolbox is used to calculate this9. Since this program iterates to find the minimizing values it is possible that it ends up in the wrong local minima if several exists. The initial estimations of the parameters determine which minima the program will end up in.

2.3.2 Identification data

The measurements used in the identification are obtained from a person being operated on for a different reason than heart problems. Therefore the data can be considered to represent a healthy person. The measured data is the pressure in the left ventricle, the pressure at the root of the aorta and the flow at the root of the aorta. The root of the aorta signifies a place in the beginning of the aorta but after the aortic valve. Each of the three data points are measured with 5999 samples. The measurements are shown in figure 7.

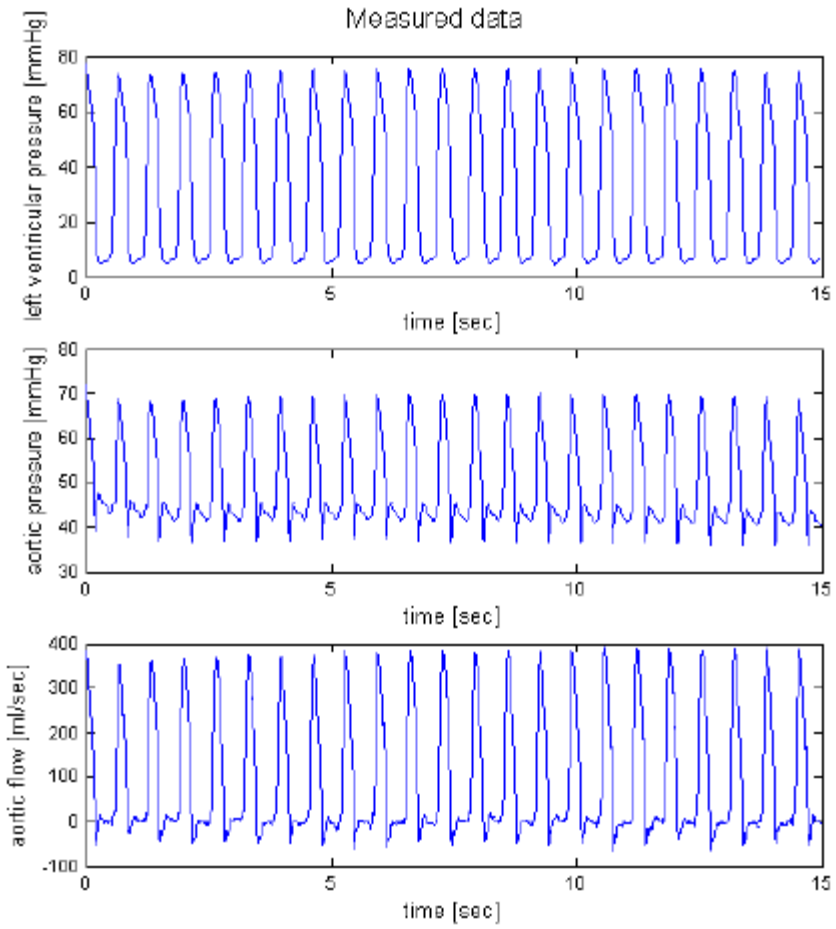


Figure 7: Measured data used for identification.

The unit of the pressure measurements is mmHg and for the flow measurements ml/sec which aren't SI units like the AVD equations use. The model for the arterial system is made as a stand alone model using these units so there needs to be unit conversions made for the signals going between the models. The pressure from the arterial model is multiplied with 133 to be converted into the SI unit N/ m2. The flow from the AVD in m3/sec is multiplied with 106 to be converted into the unit used in the arterial model. The sampling rate used was 400 Hz and the signals were filtered with an 8th order 60 Hz linear-phase low pass filter before sampling. The filter used has a high order and therefore does not at all affect frequencies a little bit lower than the cut off frequency of 60 Hz. From the DFT plots in figure 8 it is clear that most of the energy is found in frequencies well below 60 Hz and that it reduces with increasing overtones. That the original signals had high energy contents in frequencies over 60 Hz is therefore most unlikely and the measured data can be considered accurate.

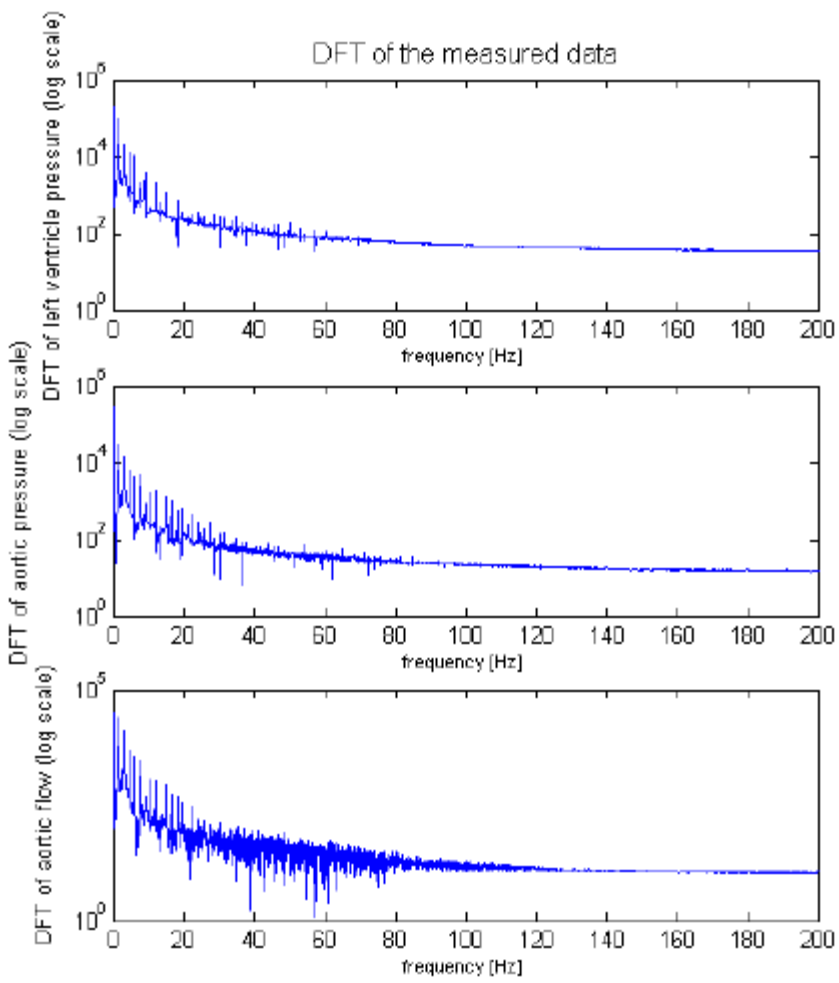


Figure 8: DFT of the measured data used for identification.

Data that is used for identifications most often have trends, such as the mean value, removed so that the identification is made easier and more correct. In this case however removing the mean value of the data would give the wrong levels when integrating and an incorrect model10. The first data point is in the middle of an ejection. In order to make it easier to find initial values of the states another starting point is chosen that is between beats. The chosen new starting point is sample 166, thereby discarding the first 165 samples.

2.3.3 Validation methods

The data presented above is divided into estimation data and validation data. The estimation data is used for identifying the models and the validation data is then used to validate and compare different models. Then the data is divided roughly in half with care taken to make the validation data start at the same point in a beat as the estimation data. When validating the models and comparing different ones the following methods will be used; Fit, loss function, FPE and correlation coefficient. Also the simulated signals are compared to the measured ones to make sure the model in fact produces the correct signals.

2.3.3.1 Fit

The fit is the percentage of the measured output that is explained by the model. It is calculated by dividing the largest prediction error by the largest difference between measured value and the mean of the measured values. This value is then subtracted from 1 and multiplied with 100 to make a percentage, the command 'norm' gives the absolute of the largest difference between values at the same time. The closer this value is to 100 the better the test says the model is.

$$Fit = 100(1 - \frac{norm(\hat{y} - y)}{norm(y - mean(y))}),$$

2.3.3.2 Loss function

The loss function is a measurement of the total error. It is calculated by adding the squares of all the prediction errors and dividing by the number of measurements. The lower this value is the better the model is according to this test.

$$Loss\_function = \frac{1}{N}\sum_{t=1}^{N}\varepsilon^2(t,\theta)$$

2.3.3.3 Correlation coefficient

The correlation coefficient is a measurement of the correlation between the measurements and the predictions. The closer this value is to 1 the better the test says the model is.

$$Correlation\_coefficient = \frac{Covariance\{\hat{y}, y\}}{\sqrt{Variance\{\hat{y}\}Variance\{y\}}}$$

2.3.4 Modeling the arteries

The first part of modeling the arterial system is to model the part that is down-flow of the root aorta measurement. This part is what will be down-flow of the AVD when it is connected. To make the model for this linear it is assumed that the arteries are passive and can be approximated using resistive, inductive and capacitive elements. Two beats of the aortic pressure 'PA' and aortic flow 'QA' are given in figure 9 below. The figure clearly show that the flow is delayed in comparison with the pressure and that the pressure should therefore be the input to the model and the flow the output.

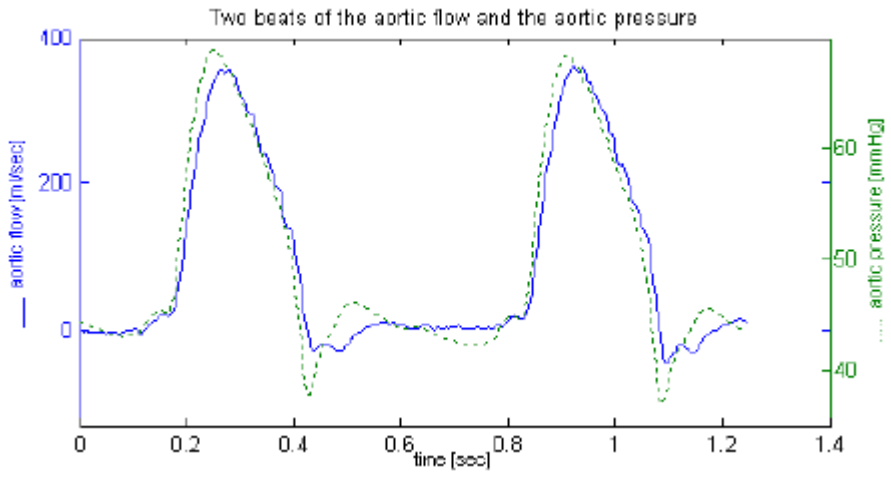


Figure 9: Two beats of the aortic pressure and aortic flow from the validation data.

Using physical modeling, the arteries are approximated with a system of ideal tubes and containers. The tubes have linear resistance and the blood in them can have fluid inertia. The containers have linear compliance and no resistance. In the physical arteries this compliance would be generated by stretching the artery walls. The ideal tubes and containers are described by the following equations. 'Px' denotes pressures, 'Qx' flows, 'Rfx' resistance,compliance in the equations. 'Lfx' fluid inertia and 'Cfx'

$$Q(t) = \frac{P_{high}(t) - P_{low}(t)}{R_{fx}}$$

$$\frac{dQ(t)}{dt} = \frac{P_{high}(t) - P_{low}(t) - Q(t)R_{fx}}{L_{fx}}$$

Equation for container;

$$\frac{dP(t)}{dt} = \frac{Q_{high}(t) - Q_{low}(t)}{C_{fx}}$$

The tube equations gives flow when between two pressures and the container equations give pressure when between two flows so by linking tubes and containers in series after each other causal models are obtained. Three of these types of model structures are defined and compared below. Their numerical values are identified using the estimation data which is the first half of the measured data and compared using the validation data which is the second half of the measured data.

2.3.4.1 Model A

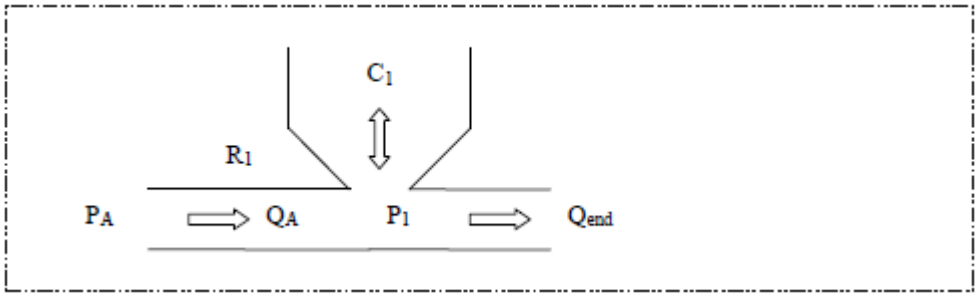
To complete the input signals to the models discussed above one more is needed; one that represents the other end of the system with respect to the left ventricular pressure. Since the measured data does not include measurements further down the arteries a simple solution is to assume a constant flow 'Qend' somewhere down the arteries. This assumptions is not only simple it are also based on the physical cardio vasculature system. At the end of the arteries are

the capillaries and there the blood is divided into a multitude of very thin streams and “filtered” to let the cells obtain nutrients.

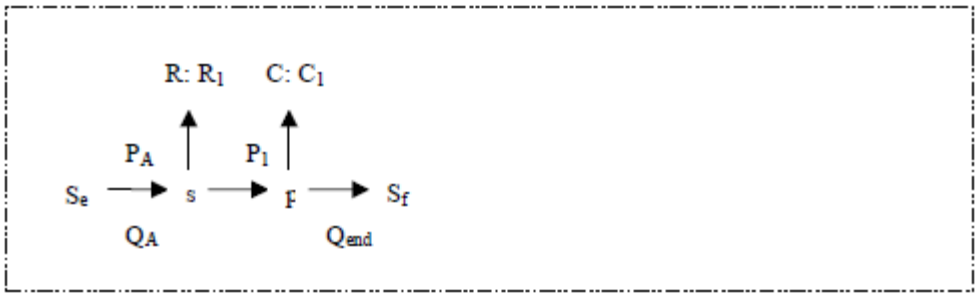
With so many capillaries working individually at different distances form the heart the average flow of them must be constant or close to constant so long as the heart rate is the same. The value of the constant flow is calculated as the average of the aortic flow so that the volume of blood in the system is the same at the beginning as at the end of the estimation, resulting in the following equation;

$$Q_{end} = \frac{1}{N}\sum_{t=1}^{N} Q_A(t)$$

Between the aortic pressure and the end flow a tube without inertia and a container is put in series. The first tube relates to the resistance ‘R1’ in the beginning of the arteries. The container relates to any compliance ‘C1’ in the whole of the arteries. Inertia in the beginning of the arteries is ignored. Any resistance or inertia at the end of the arteries and capillaries are not important since the flow there is considered to be constant. A schematic picture of model A is shown below in figure 10.



The system is explained by the following equations;, P1 is the pressure produced by the compliance in the container.



$Q_A(t) = \frac{P_A(t) - P_1(t)}{R_1}$, P_1 is the pressure produced by the compliance in the container.

$\frac{dP_1(t)}{dt} = \frac{Q_A(t) - Q_{end}}{C_1}$

The parameters that are let loose to be identified by minimizing the prediction error are; ‘R1’, ‘C1’, ‘P1(0)’. (0) stands for the initial value of that state at time 0.

Several different initial values for the loose parameters were tested and either the program reached the local minima that gives the parameter values used it or ended up far from them with very high loss functions (calculated with the estimation data as comparison). The local minima found are therefore considered to be the global one.

The identified model gives the simulated aorta flow plotted in figure 12 when driven by the validation data. The plot also includes the original validation aorta flow as a comparison. Only the first two beats are displayed to make the two flows easier to distinguish from each other. That the model so well predicts the aortic flow is a strong indication that the parameter values represent the global minima.

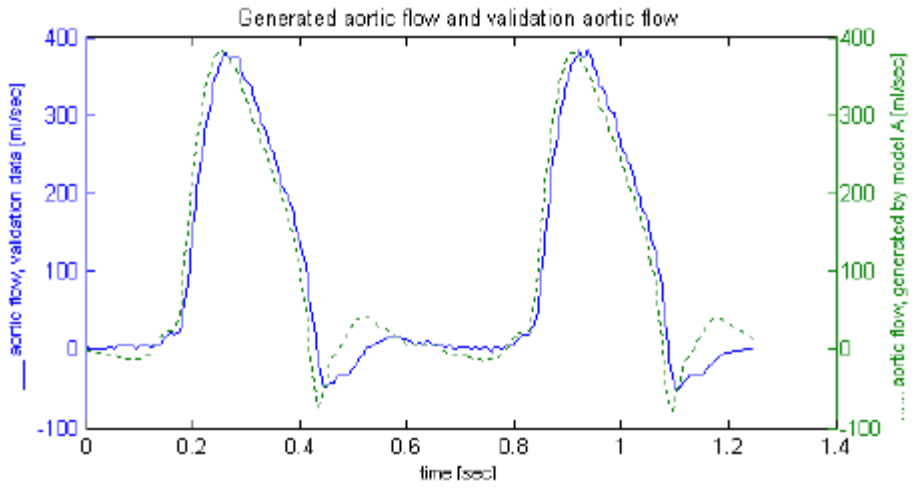


Figure 12: Comparison between generated aortic flow by model A and validation aortic flow.

Validation method	Value
Fit	79.5
Loss function	824.6
Correlation coefficient	0.979

The prediction of the aortic flow by this model compared to the measured flow is quite

good, but there are two major differences; the predicted flow drops sooner and levels out with far more ripple. Both of these things and the fact that the measured flow is delayed compared to the measured pressure all hints at that inertia should be included in the model. Inertia slows down fast changes like the turn from level to increasing flow, from increasing to dropping flow and the ripple when the flow levels out.

2.3.4.2 Model B

The difference between this model and model A is that inertia 'L1' is added to the blood in the tube. This means that this model of the arteries is a tube with inertia followed by a container. The input and output signals are the same as in model A; aortic pressure and a constant end flow as inputs and the aortic flow as output. Model B is given in figure 13 below.

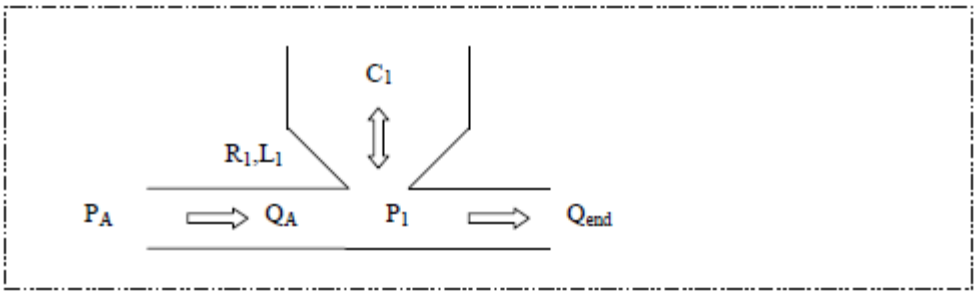
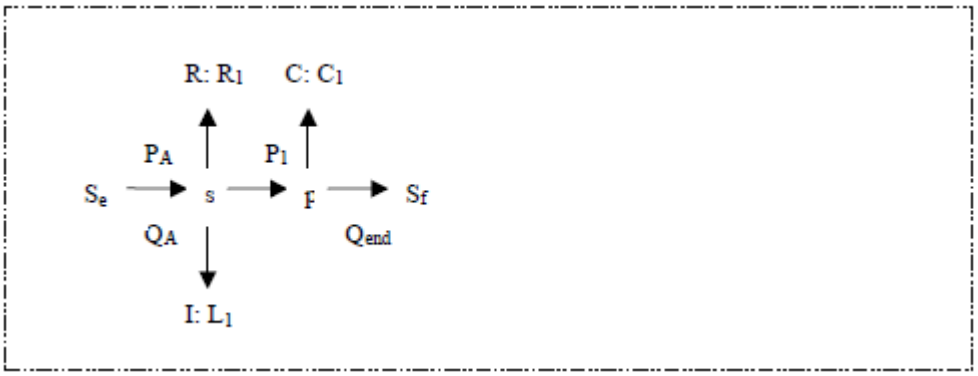


Figure 13: Schematic picture of model B.

The parameters that are let loose to be identified by minimizing the prediction error are; 'R1', 'L1', 'C1', 'P1(0)'.

The initial value of the state aortic flow, 'QA(0)', is fetched from the first data point of the aortic flow in the estimation data.



$\frac{dQ_A(t)}{dt} = \frac{P_A(t) - P_1(t) - Q_A(t)R_1}{L_1}$, P_1 is the pressure produced by the compliance in the container.

$\frac{dP_1(t)}{dt} = \frac{Q_A(t) - Q_{end}(t)}{C_1}$

The parameters that are let loose to be identified by minimizing the prediction error are; 'R_1', 'L_1', 'C_1', '$P_1(0)$'.

The initial estimates for 'R1' and 'P1(0)' are take from the identification of their counterparts in model A. The initial estimate for the inertia L1 is approximated by

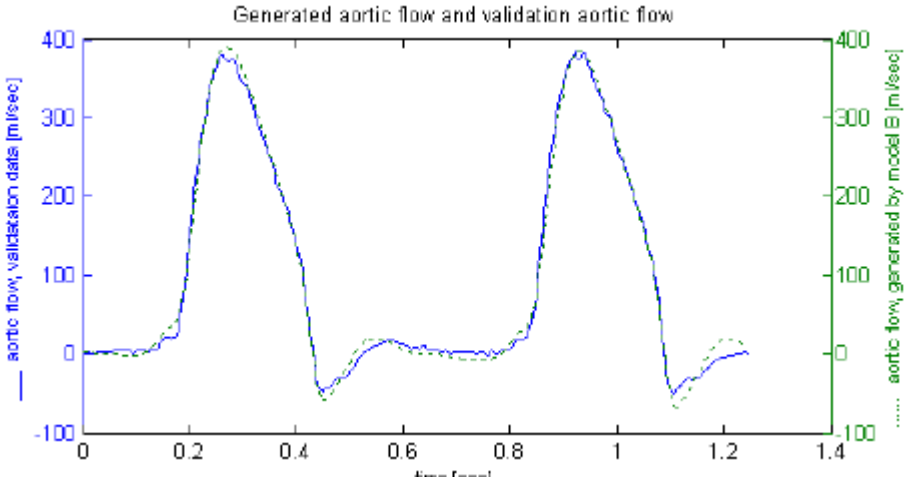
guessing reasonable values for the diameter ‘dAg‘ (0.02 m) and length ‘lAg‘ (0.2 m) of the aorta. This is then used to calculate the fluid inertia of the blood in a tube with that size. It is done in the same way the fluid inertia of the blood in the tube was calculated and the equations are given below.

$$L_{1\_init} \approx \frac{1000 l_{Ag} 2^2}{\pi d_{Ag}^2}, \text{ in SI units.}$$

$$L_{1\_init} \approx \frac{1}{133 \cdot 10^6} \frac{1000 l_{Ag} 2^2}{\pi d_{Ag}^2}, \text{ in the arteries' model units.}$$

Using the identified value for the counterpart of ‘C1‘ in model A gives a terrible loss function which means the wrong local minima was found. By making the start estimate 10 times as big a minima that gives much better results is found. The physical interpretation of making ‘C1‘ bigger is making the walls stiffer which makes sense because now some of the dynamics are explained by the inertia. Several other initial estimates were used but none gave a better loss function the ones described.

The identified model gives the simulated aorta flow plotted in figure 15 when driven by the validation data. The plot also includes the original validation aorta flow as a comparison. Only the first two beats are displayed to make the two flows easier to distinguish from each other. That the model predicts the aortic flow so well is also a strong indication that the parameter values represent the global minima.



The validation tools give the values in table 5;

Validation method	Value
Fit	91.7
Loss function	134.8
Correlation coefficient	0.997

Table 5: Validation values for model B.

When comparing the plots of the predicted flow from model B and model A it is clear that model B produce better results. The flow doesn't drop too soon and the ripple when the flow levels out is also much better. All of the validation numbers are also much better for model B than model A. Based on the better looking plot and the better validation numbers it is determined that fluid inertia must be included and that model A is discarded.

The prediction results by model B are very good and it is highly unlikely that a better model can be constructed without adding many more parameters. These added parameters would make the identification process more unstable as more local minima would exist. It would also be a high uncertainty whether the parameters actually represent their physical constants or would include noise characteristics. The type of model that includes a tube with fluid inertia and resistance and a container with compliance is therefore decided to be best solution. However, model B does have one fault; the constant end flow requires that the heart rate remains the same and the model can therefore not handle a heart that sta

2.3.4.3 Model C

To handle a change in heart beat the input of the constant end flow in model B is changed to an end pressure 'Pend'. This also requires that a tube is added after the container and the end pressure to keep the model causal.

This tube represents the end of the arteries and these have branched out a lot making the area quite large which in turn makes the fluid inertia quite small (compared with the calculation of the inertia in the AVD tube). Since the fluid inertia is small it is possible to ignore it and use a

tube without inertia and only the resistance ‘R2’ in the model. The pressure in the capillaries can probably be considered to remain constant as long as the heart rate remains the same. How the pressure would change with a change in heart rate can’t be determined from the available data. With a constant heart rate there might still exist small pressure changes in the capillaries but when averaged out over all of them it should be constant or very close to constant. The way chosen to describe the end pressure is by assuming that there is a point where the average pressure equals the average flow time a constant ‘X’, according to the equation below.

$$P_{end}(t) = Q_{end}(t)X$$

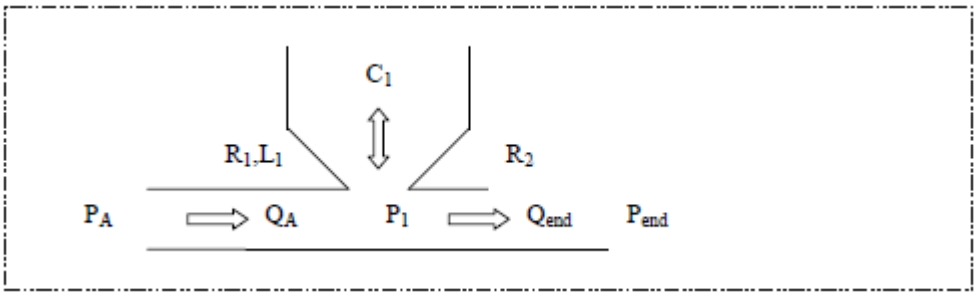


Figure 16: Schematic picture of model C.

The bond graph for the model is presented in figure 14.

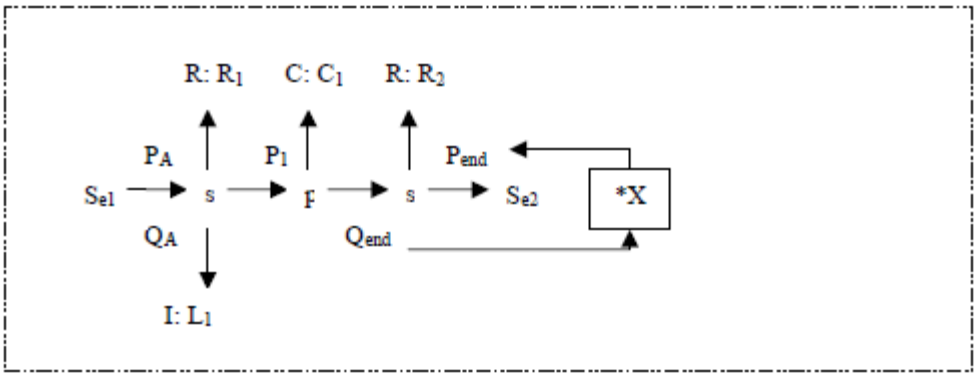


Figure 17: Bond graph for Model C.

$\frac{dQ_A(t)}{dt} = \frac{P_A(t) - P_1(t) - Q_A(t)R_1}{L_1}$, P_1 is the pressure produced by the compliance in the container.

$$\left.\begin{aligned} \frac{dP_1(t)}{dt} &= \frac{Q_A(t) - Q_{end}(t)}{C_1} \\ Q_{end}(t) &= \frac{P_1(t) - P_{end}(t)}{R_2} \\ P_{end}(t) &= Q_{end}(t)X \end{aligned}\right\} \Leftrightarrow \frac{dP_1(t)}{dt} = \frac{Q_A(t)(R_2 + X) - P_1(t)}{(R_2 + X)C_1}$$

The system is explained by the following equations;

These are the same equations as if the pressure was zero at the end of the tube and it had the constant plus the resistance as its resistance but the body does not have zero pressure. If the constant and resistant is added and thought of as a resistance and the pressure set to zero like mentioned above the model is identical to the ‘four element Windkessel’ model.

The Windkessel model is well known in the medical industry. The name has it’s origin in the Windkessel model presented by Otto Frank in an 1899 paper11. The model he presented has later become known as the two element Windkessel model because it has two elements; a container and a tube without inertia. The model has been extended in several different ways but the name Windkessel is still the common name used. With zero pressure somewhere at the capillaries negative pressure is needed in the veins to get the blood back to the heart and it would be hard to implement such a model. It is easier to add a model of the veins similar to the arterial one if the pressure is higher than zero at the capillaries.

The parameters that are let loose to be identified by minimizing the prediction error are; ‘R1’, ‘I1’, ‘C1’, ‘R2’+’X’, ‘P1(0)’.

The initial value of the state aortic flow, ‘QA(0)’, is fetched from the first data point of the aortic flow in the estimation data (same as for model A).

The initial estimation of the loose parameters are in part fetched from the identified values of their counterparts in model B. Only the new constant ‘R2’+’X’ is not represented in the other model and have to have its initial estimation guessed. It is given the value that was identified for ‘R1‘ in model B. With these initial estimations the model gives about the same loss function as model B. This is considered a strong indication that the local minima found is the global one.

The identified model gives the simulated aorta flow shown in figure 18 when driven by the validation data. The figure also includes the original validation aorta flow as a comparison. Only the first two beats are displayed to make the two flows easier to distinguish from each other. That the model so well predicts the aortic flow is also a validation that the parameter values represent the global minima.

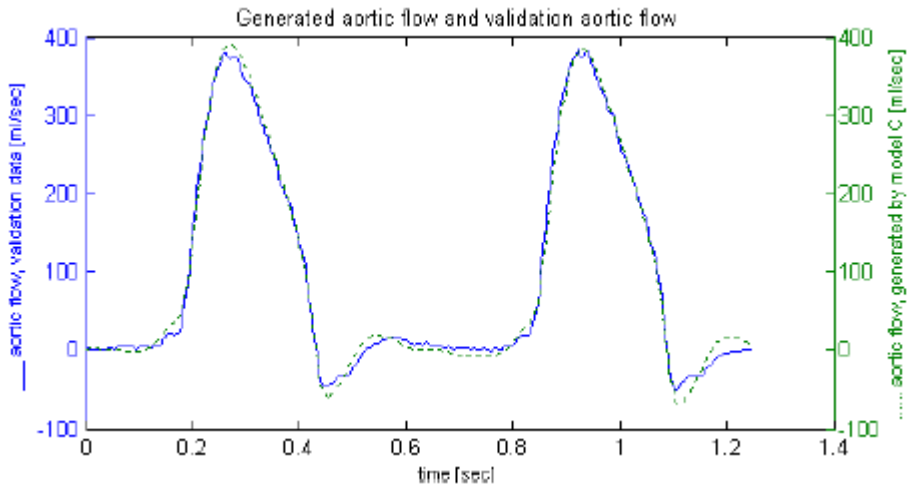


Figure 18: Comparison between generated aortic flow by model C and validation aortic flow.

Validation method	Value
Fit	91.4
Loss function	145.7
Correlation coefficient	0.997

The validation tools give the values in table 6;

$$l_{Ai} = \frac{\pi d_{Ag}^2 133 \cdot 10^6 L_1}{2^2 1000}$$

Table 6: Validation values for model C.

The plots and validation numbers from model C and model B are very similar. The validation numbers from model B are a bit better, but model C can handle a change in heart beat passively and this type of model is well known in the medical industry so model C is the model chosen.

Another validation for the model is given by calculating the length 'lAi' of a tube with a guessed aorta diameter of 2.3 cm and the inertia that was identified according to the equation below.

The identified inertia and same guessed aorta diameter as before gives that the aorta length after the aortic pressure measurement is 6,82 cm which is believable when considering that the true aorta branches out very fast.

2.3.5 Expanding the model to include the root of the aorta

The model above covers the part of the arteries that is down-flow of the aorta measurement. The part that is up-flow is a short piece of the aorta, the aortic valve and the left ventricle. Same as in the artery model the decision whether to have the flow or pressure as input in the up-flow end is based on which is delayed compared to the other. The flow is the same aorta flow that was used in the artery model and the pressure is the left ventricle pressure 'PLv'. As can be seen in figure 19 the flow is even more delayed here compared to the pressure so the left ventricle pressure is seen as an input and the aortic flow as the output.

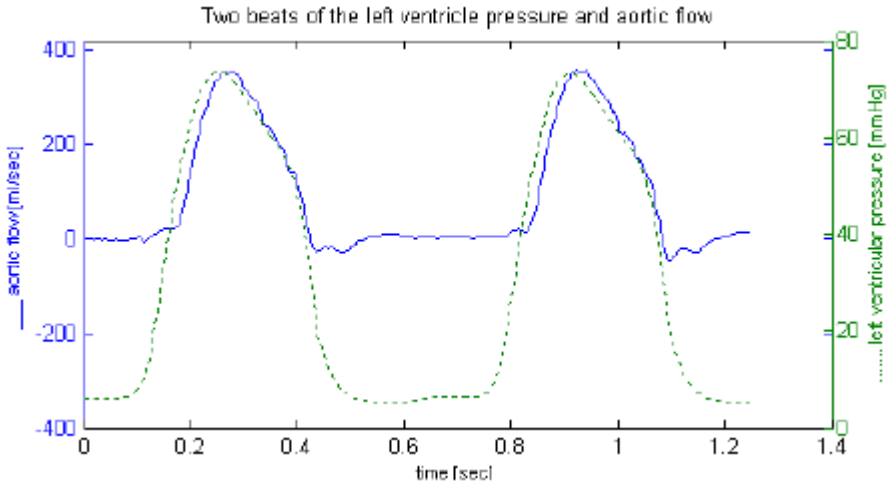


Figure 19: Two beats of the left ventricle pressure and aortic flow from the validation data.

The left ventricle pressure measurements can however only be used as they are if the effects of the valve could be linearized. This can't be done over a full beat so instead the left ventricle pressure is modified to include them. This is done using the maximum of the two pressure measurements for every sample 'PLv*' according to the equation further down. This is an approximation of having the pressure be the left ventricle pressure when the valve is opened and having it be the aortic pressure when it is closed. The changes between the two will not happen exactly when the valve opens and closes but it will be close enough and the resulting pressure function will be continues.

$$P_{Lv}^{*} = \max(P_{Lv}, P_{A})$$

By ignoring any stretching in the aorta wall and excluding the valve the added part of the aorta can be seen as a rigid tube. The tube is assumed to have resistance 'RA' and the blood in it to have fluid inertia 'LA'. The values of the resistance and fluid inertia are identified using the same model structure as in the artery model. The differences between the systems are that the new model has a little more resistance and inertia in the first tube and that the driving pressure is changed to the modified left ventricle pressure. By keeping the values of all the other parameters fixed the increases can be identified.

The schematics of model C when it has been expanded to include the aortic root are given below in figure 20.

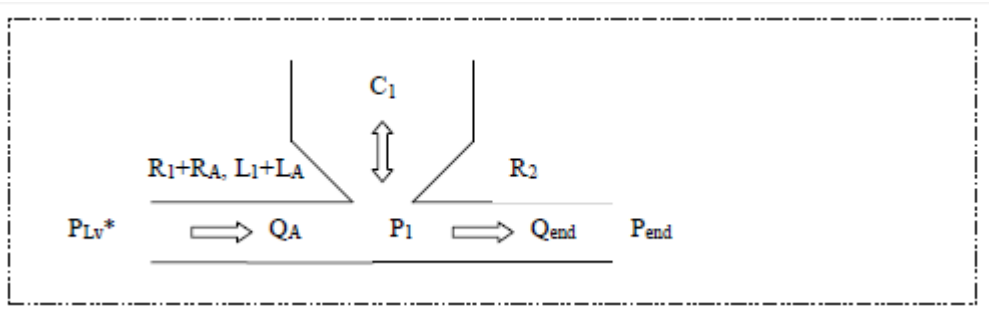


Figure 20: Schematic picture of model C, including the model of the root of the aorta.

The bond graph of the model is presented in figure 21.

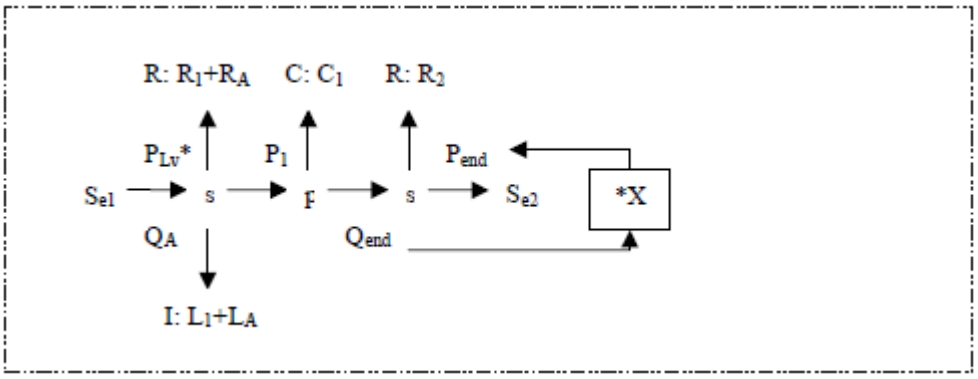


Figure 21: Bond graph for model C, including the root of the aorta.

The system is explained by the following equations;

$$\frac{dQ_A(t)}{dt} = \frac{P_{Lv}^*(t) - P_1(t) - Q(t)(R_1 + R_A)}{L_1 + L_A}$$, P_1 is the pressure produced by the compliance in the container.

$$\frac{dP_1(t)}{dt} = \frac{Q_A(t)(R_2 + X) - P_1(t)}{(R_2 + X)C_1}$$

The parameters that are let loose to be identified by minimizing the prediction error are; ‘RA‘ and ‘LA’. This is done by identifying values for ‘R1’+’RA‘ and ‘L1’+’LA‘ and then

subtracting the ‘R1‘ and ‘L1‘ values identified in the arteries model. The other parameter values are also taken from the arteries model.

The initial value for the state aortic flow, ‘QA(0)’, is fetched from the first data point of the aortic flow in the estimation data (same as for model C and A). The initial value of the pressure in the container is taken from the identified value in the artery model.

The initial estimation of the loose parameters is fetched from the identified values in the artery model. This means that the initial estimate of both the resistance and the fluid inertia in the added part of the aorta are zero.

The identified model gives the simulated aorta flow in figure 22, when driven by the validation data. The figure also includes the original validation aorta flow as a comparison. Only the first two beats are displayed to make the two flows easier to distinguish from each other.

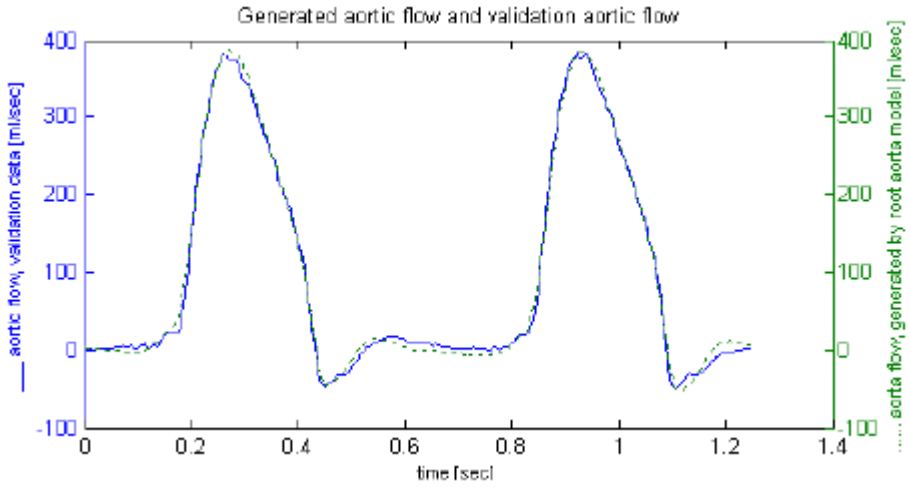


Figure 22: Comparison between generated aortic flow by model C and aortic root model and validation aortic flow.

The validation tools give the values in table 7;

Validation method	Value
Fit	93.2
Loss function	91.5
Correlation coefficient	0.998

Table 7: Validation values for model C, including the root of the aorta.

The validation values are even better here than in the chosen artery model and the plot just as good. Another validation is given by calculating the length 'lrAi' of a tube with the same guessed aorta diameter as before (2.3 cm) and the inertia that was identified. The equation for this calculation is given below.

$$l_{rAi} = \frac{\pi d_{Ag}^2 133 \cdot 10^6 L_A}{2^2 1000}$$

The identified inertia and same guessed aorta diameter as before gives that the aorta pressure measurements were made 2,16 cm from the opening to the left ventricle. This should be about the right length considering the diameter.

All of the validation methods above support that the identified values of the added part of the aorta are correct.

2.3.6 Model of the aortic valve

The effects of the aortic valve were in the resistance and fluid inertia identification earlier included in the left ventricle pressure. As stated there the effects can't be linearized over a full beat. However, they can be linearized if the beat is divided into two parts; namely valve opened and valve closed.

When the valve is open any effects other than those already taken into account are ignored. The resistance that the opened valve contributes to is already identified together with the resistance in the root of the aorta.

The closed valve is seen as a stretchable drum that covers the opening in to the aorta. The drum is linearized and modeled with compliance and resistance which gives it the same characteristics as a piston with a spring and damper (ideal resistance) behind. The ideal drum is described by the following equation;

'Px' denotes pressures, 'Qx' flows, 'Rx' resistance and 'Cx' compliance in the equation for a drum below.

$\frac{dP_{high}(t)}{dt} = \frac{dP_{low}(t)}{dt} + \frac{Q(t)}{C_x} + \frac{dQ(t)R_x}{dt}$, the flow 'Q' is assumed to be going from 'Phigh' to 'Plow'.

The aortic valve is included in the arteries model by placing it between the left ventricle and the root of the aorta, figure 23 shows how. The pressure 'PV' created by the valve from the compliance 'CV' and resistance 'RV' is now the pressure that is seen by the root of the aorta.

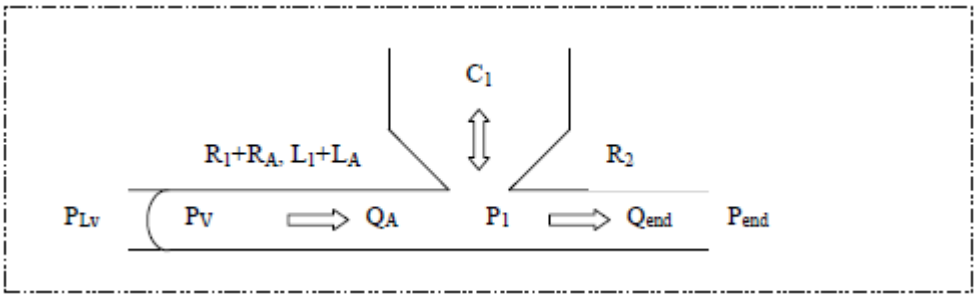


Figure 23: Schematic picture of model C, including aortic root and closed aortic valve.

The bond graph of the model is given in figure 24.

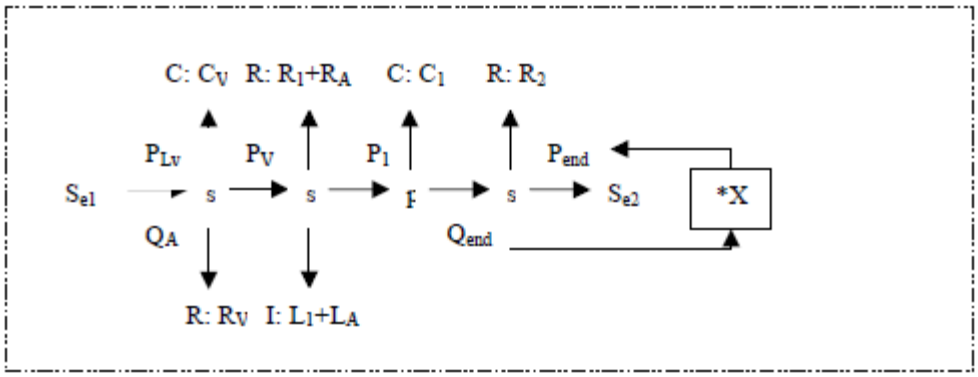


Figure 24: Bond graph for Model C, including aortic root and closed aortic valve.

The system is explained by the following equations

$$\frac{dP_V(t)}{dt} = \frac{dP_{Lv}(t)}{dt} - \frac{Q(t)}{C_V} - \frac{dQ_A(t)R_V}{dt}$$

$$\frac{dQ_A(t)}{dt} = \frac{P_V(t) - P_1(t) - Q_A(t)(R_1 + R_A)}{L_1 + L_A}$$, P_1 is the pressure produced by the compliance in the arteries (in the model it is the container).

$$\frac{dP_1(t)}{dt} = \frac{Q_A(t)(R_2 + X) - P_1(t)}{(R_2 + X)C_1}$$

The identification of the values is preformed by using the artery and aorta model and adding the drum to the root of the aorta. The friction is added to the resistance in the root of the aorta since it comes in at the same place in the equations. The pressure ‘PVi’ that the spring creates is added to the pressure of the left ventricle to get the pressure that the root of the aorta and the resistance of the valve experiences. In this identification the left ventricle pressure is taken from the measurements as they are. However, since identification only covers the part of the beats when the valve is closed only those samples are taken from the measurements. The sample points when the valve is closed are picked out from the measurements by looking at their plots. The valve is assumed to close when the flow drops below zero after the ventricle ejects and open again approximately when the aorta pressure exceeds the left ventricle pressure. The equations describing this are;

$$\frac{dP_{Vi}(t)}{dt} = \frac{dP_{Lv}(t)}{dt} - \frac{Q_A(t)}{C_V}$$

$$\frac{dQ_A(t)}{dt} = \frac{P_{Vi}(t) - P_1(t) - Q_A(t)(R_1 + R_A + R_V)}{L_1 + L_A}$$

, 'P_1' is the pressure produced by the compliance in the arteries (the container in the model).

$$\frac{dP_1(t)}{dt} = \frac{Q_A(t)(R_2 + X) - P_1(t)}{(R_2 + X)C_1}$$

The parameters that are let loose to be identified by minimizing the prediction error are 'RV' and 'CV'. This is done by identifying values for 'RV'+'R1'+'RA' and 'CV' and then subtracting the 'R1' and 'RA' values identified in the arteries model to get 'RV'. The other parameter values are taken from the arteries and aortic root models earlier.

The initial value of the state aortic flow is given the average flow of the first flow measurement in every closed valve interval. The initial value of the pressure in the arteries container is taken from a simulation of the artery and aorta model and is an estimate of the average pressure when the valve should open. If the container's initial pressure value is let loose to be identified it is given a non-physiological value that it too high (around 80 mmHg which is higher than any measured pressure).

The initial estimation of the loose constant 'RV' is zero. The initial estimation of 'CV' is given the value of the arteries' compliance times 10, as the valve should be stiffer than the whole of the arteries.

The identified model gives the simulated aorta flows plotted in figure 25 when driven by the validation data interval that gives the best Fit value and in figure 26 when driven by the validation data interval that gives the worst Fit value. The values from the validation tools are shown in Table 8.

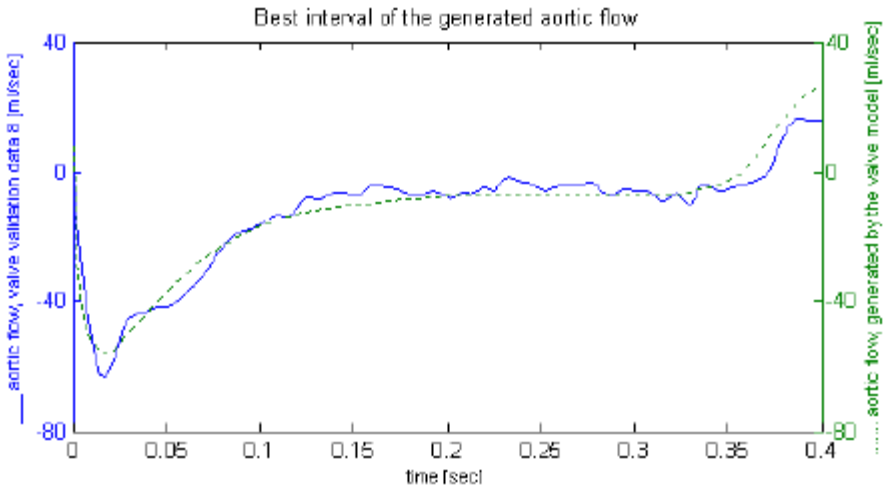


Figure 25: Comparison between generated aortic flow by the valve model and validation aortic flow for the interval that gives the best Fit value.

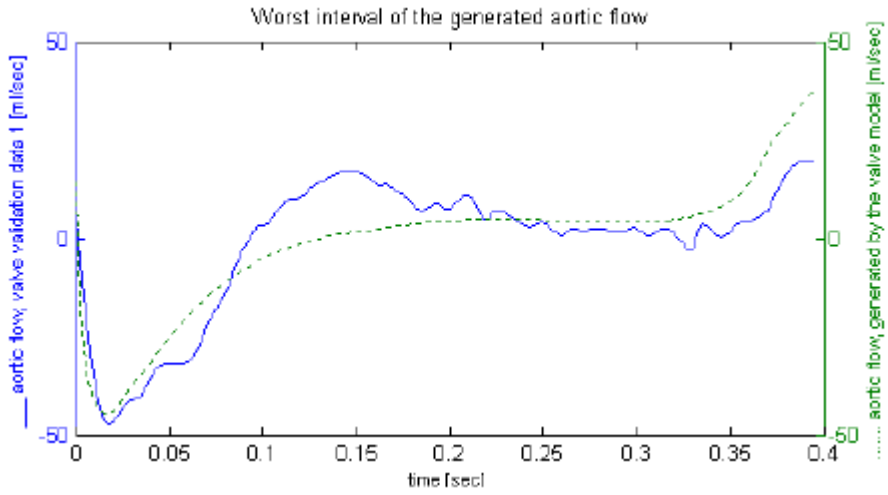


Figure 26: Comparison between generated aortic flow by valve model and validation aortic flow for the interval that gives the worst Fit value.

Validation method and interval	Value
Fit for validation interval 1	51.6
Fit for validation interval 2	61.3
Fit for validation interval 3	56.6
Fit for validation interval 4	55.4
Fit for validation interval 5	69.4
Fit for validation interval 6	53.1
Fit for validation interval 7	52.2
Fit for validation interval 8	75.5
Fit for validation interval 9	63.5
Fit for validation interval 10	58.9
Average loss function	39.0
Average loss function for stiff valve	295.0

Table 8: Validation values for valve model.

The plots are not perfect nor are the Fit values great. Also, the fact that the identification wanted the container pressures to be higher than it should physiologically hints at that the model does not fully describe the valve. But both from the plot and the Loss function values it is clear that this model is much better than just using a totally stiff valve as with a saturation model. Also a totally stiff valve would keep the flow constant at zero while it was closed and not at all react to the effects caused by the AVD.

If the valve were a flat piston like the model suggests it would move an average of 8,6 mm when it is most displaced. The distance is an average of all the maximum displacements in the validation data's closed valve intervals and calculated as shown in the equation below. No measurements on valve stretching are available so the calculated length can't be compared to the real one. But the stretching length calculated is about a third of a normal aorta diameter so it is reasonable to model the valve in this way. closed and the summation is over all the samples of the aortic flow where the valve is closed.

$$movement = \frac{2^2}{\pi d_{ag}^2} \frac{10^{-6}}{N_{closed}} \cdot \sum_{valve-closed} Q_A$$, 'N$_{closed}$' is the number of times the valve is closed and the summation is over all the samples of the aortic flow where the valve is closed.

The change between the opened and closed valve states is done by only allowing the pressure difference between the left ventricle pressure and the aortic root pressure that is added by the valve to be positive. This means that the valve can only make the pressure in the aortic

root higher than the pressure in the left ventricle, not the other way. Also to keep the integration of the flow pushing the valve from 'windup' when the valve is closed it isn't allowed to be negative. The equations for the valve are given below.

$P_T(t) = P_{Lv}(t) + \max(0, \Delta P(t))$, '? P' is the pressure generated by the aortic valve in the model.

$$\frac{d\Delta P(t)}{dt} = \begin{cases} 0 \ , \ Q_{AV}(t) \geq 0 \ and \ \Delta P \leq 0 \\ -\frac{Q_A(t)}{C_V} - \frac{dQ_A(t)R_V}{dt} \ , \ otherwise \end{cases}$$

2.3.7 Model of the left ventricle

Different types of pressures have been used as inputs to the models in all the identification processes earlier. However, in the final model however this will not work. Changing the load seen by the heart by adding the AVD alters the pressure profile of the left ventricle and the flow profile in the aorta. If the pressure profile that was measured is used and the load made by the AVD is very low then the heart would eject all its blood and even try to eject more so it can't be used as the input to the final model. Since no measurements were available on a heart affected by different loads the best assumption that can be made is that the compliance profile of the left ventricle remains the same even with a change in load. With this input the left ventricle will get reduced volume if the load is lowered. This is the same kind of response as when the load is lowered using a VAD. If the heart muscle is considered to be a piston with a spring attached to it and the left ventricle as a linear container then the heart compliance is the spring constant (stiffness of the spring). The spring 'constant' changes according to how hard the heart muscles contract. Keeping the compliance profile the same is the same as keeping the spring constant profile the same.

To calculate the compliance profile 'CLv' the profile of the volume of the left ventricle 'VLv' is needed. No measurements of the volume were available. Instead the volume profile is approximated by using the measured outflow of the ventricle, approximating the inflow to the

ventricle by shifting the aortic flow and assuming a start volume. One full beat is about 264 samples and the shift used is 132 samples; half of a full beat. The start volume of the ventricle is made so that the volume just before ejection is 120ml. This assumption is based on the fact that the measurement values are low and the person has to be small and therefore should have a small heart. The equations for compliance profile is calculated are given below.

$V_{Lv}(t) = 120 + Q_A(t - \frac{T_b}{2}) - Q_A(t)$, 'Tb' is the time elapsed during one beat.

The compliance profile is the volume divided by the pressure for every sample.

$$C_{Lv}(t) = \frac{V_{Lv}(t)}{P_{Lv}(t)}$$

The compliance is calculated using the values at that time instant and values from other time points can not be used to validate it. This means that there is no way of validating this model without data with different loads from the same patient.

Assuming that the inflow looks just like the aorta flow might give the wrong inflow profile, but the valve is closed during the time the inflow is high so it does not affect the pressure in the aorta that much. The guess of the ventricle volume before ejection might also be a little wrong and a different value would change the compliance, but the pressure generated would be the same without a lesser load and should only be slightly different with a lesser load so a small error would not have that big an affect.

2.3.8 Comments

To verify how good the final model of the systemic arterial system works the generated data is compared to the original measured data. In figure 27 a comparison of the validation left ventricular pressure and generated left ventricle pressure is shown. In figure 28 a comparison of the validation aortic pressure and generated aortic pressure is shown. In figure 29 a comparison of the validation aortic flow and generated aortic flow is shown. Only the first two beats are displayed to make the predicted data and the measured validation data easier to distinguish from each other.

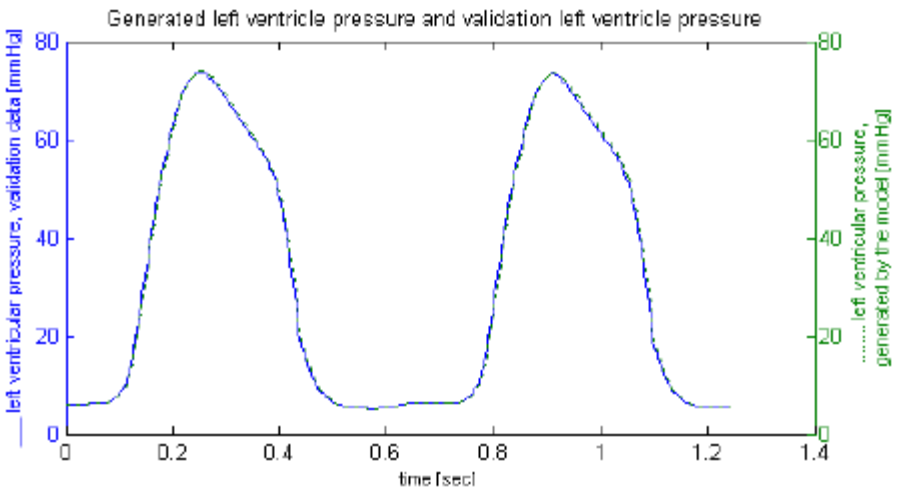


Figure 27: .

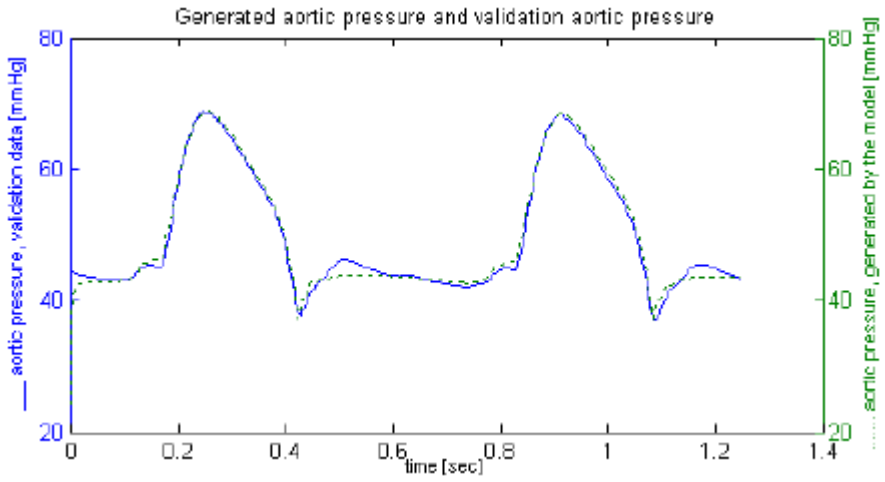


Figure 28: Comparison between generated aortic pressure by the total arterial model and validation aortic pressure.

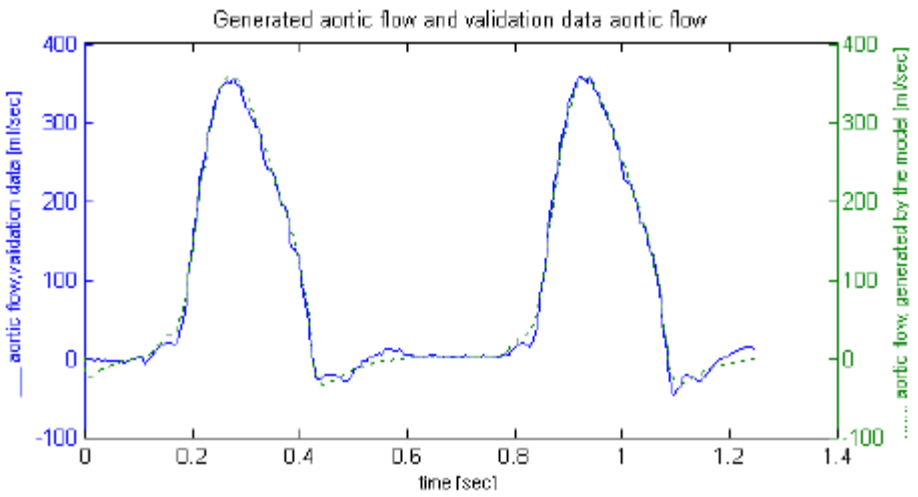


Figure 29: Comparison between generated aortic flow by the total arterial model and validation aortic flow.

The generated data is very close to the original validation data during the time when the aortic valve is opened. During the time when the valve is closed the generated aortic pressure and flow are somewhat different from the measured, but these differences are most likely caused by the model of the valve. The model of the valve clearly isn't perfect, but it does a much better job than just using a saturation model. The valve model produce the same kind of dip in the flow when the ventricular pressure drops below the aortic pressure and even the same little rise in the flow just before the ventricular pressure exceeds the aortic pressure.

As stated above the model of the systemic arterial system describes the dynamics very good if the load is not changed with an AVD. The input signal used for the identification is periodic but it is built up of several frequencies so it should excite most of the dynamics in the system so they should have been modeled. However, how a load change with an AVD would change the dynamics is impossible to know from the available data. The arteries could be active and alter their resistance or compliance with the change in flow and pressure that will occur. Even more likely is that the ventricles compliance profile isn't conserved, but it would change with a change in the load. To know how things would change with a change in load

measurements need to be taken with a working AVD. The model can then be updated with this data.

2.4 Modeling the added valve

No data on the valve that will be used together with the AVD is available so the best model of it that can be constructed is a copy of the aortic valve. The same compliance and resistance values that were identified for the closed aortic valve are used for the closed added valve. The resistance of the closed added valve 'RAV' is given the value of the closed aortic valve. The compliance of the closed added valve 'CAV' is given the value of the closed aortic valve. The resistance the open aortic valve has can not be identified separately from the rest of the resistance in the root of the aorta so the only thing that can be done is to guess the relation between them. The relationship used is 1:1, which means the resistance of the opened added valve 'RAVo' is given half the value of the resistance of the root of the aorta;

$$R_{AVo}(t) = \frac{R_A}{2}$$

2.4.1 Comments

The model of the added valve is a copy of the model for the aortic valve while the valve that actually will be added will most likely be an artificial one. Artificial valves must have different dynamics than biological ones but they are built to emulate the biological ones so they should be at least similar.

2.5.1 Connecting the models

The models that need to be connected are the models for the AVD, the arteries, the root of the aorta, the valve, the added valve and the left ventricle. The AVD model, the arteries model and the aortic root model all connect to the same point in the aorta, where the blood branches out to the AVD and the original arteries. All three of them need the pressure at that junction 'PA' as inputs to be causal. It is not possible for all of them to get the pressure as input; one of them has

to generate the pressure for the other two. The one that is chosen to do this is the aortic root model because it is simplest to modify. Instead of taking the pressure at both ends and generating the flow through it as defined for a tube with inertia, it takes the derivative of the aortic flow and the pressure after the aortic valve to generate the pressure at the junction according to the following equation.

$$\frac{dQ_A(t)}{dt} = \frac{P_V(t) - P_A(t) - Q_A(t)R_A}{L_A} \Leftrightarrow P_A(t) = P_V(t) - \frac{dQ_A(t)L_A}{dt} - Q_A(t)R_A$$

Having a pure derivative makes the computation in MatlabR of the model unstable so an approximation is used as shown below (the approximation is given in Laplace which is the format the equations are inputted in SimulinkR).

$$P_A(t) = P_V(t) + Q_A(t)(L_A \frac{s}{0.001s+1} + R_A)$$

Modifying the model of the root of the aorta like this does not alter the simulation results much. This is validated by comparing the flow generated by the root of the aorta and arteries model when the resistances and inertias are added together and the modified model when they are separated. The modified left ventricle pressure that was used in the identification of the root of the aorta is used as input to the two models. As can be sen in figure 30 the two flows are very close to identical.

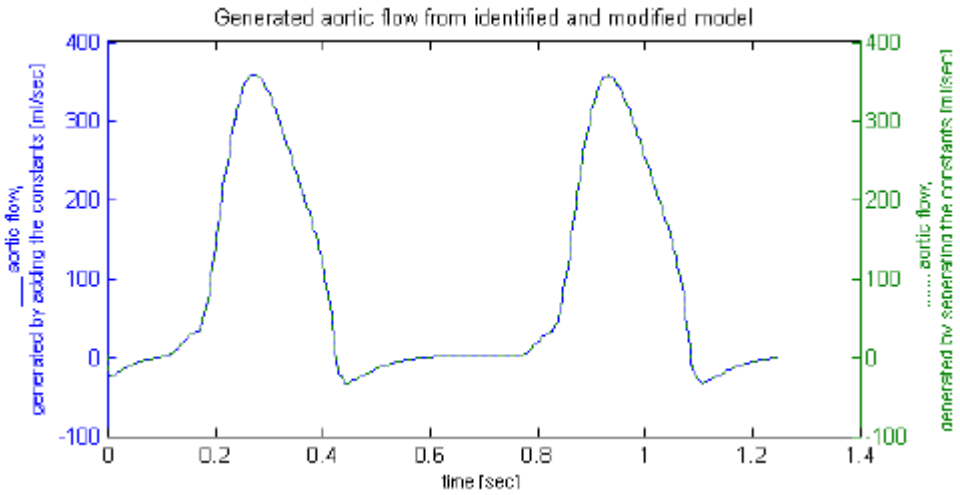


Figure 30: Comparison between the identified model where the fluid inertia and resistance parameters are added and the modified one where they are separated.

The aortic valve model is added up flow of the root of the aorta model. When it is open it does nothing. When it is closed it adds pressure to the ventricle pressure seen by the root of the aorta. The pressure added is generated from the compliance and resistance of the drum.

The added valve is assumed to be implanted just down flow of the AVD tube insertion so the model is inserted just up flow of the arteries. The model works in the same way as the aortic valve model and adds pressure to the junction point seen by the arteries model. The resistance the opened added valve has is added to the resistance of the arteries.

The added valve and the AVD makes the flow from the left ventricle ‘QA’ different from the flow that goes into the arteries. In the modeling before the same notation was used for both these flows but now they need different notations so they are not confused with each other. The flow to the arteries through the added valve will be called ‘QB’.

The left ventricle model is inserted up flow of the aortic valve model. From the compliance profile and the volume of blood it has stored it produces the pressure that drives the flow of the blood.

2.5.2 Inputs to the total model

The AVD model has two inputs; the voltage to the motor and the pressure at the end of the tube. The pressure at the end of the tube is generated by the systemic arterial model so only the voltage will remain an input to the final model. What voltage to give to the AVD is decided by the controller and how it decides this is presented in the chapter.

The systemic arterial model has two inputs; the compliance profile to the left ventricle and the inflow of blood to the ventricle. The compliance profile and inflow that is used is the

same that was used in section ‘2.3.7 Model of the left ventricle‘. Using these inputs will create errors of unknown importance. The compliance profile might change with a change in load. The inflow to the ventricle defiantly changes with a change in load but the changes are most likely transient. A large load change would create transient effects in both the outflow and inflow to the ventricle but after a few beats an equilibrium would be reached. At this equilibrium the volume of blood ejected and taken into the ventricle should be the same as before the change in load. So even if the outflow and inflow profiles changed the amount is the same and the inflow profile should still be viable as an input since it fills the ventricle with the right amount of blood.

2.6 Simulations from the total model

To understand how the final model works the generated aortic flow is compared to the original measured aortic flow. First the added valve is inserted in the model. The simulation with the added valve is shown in figure 31.

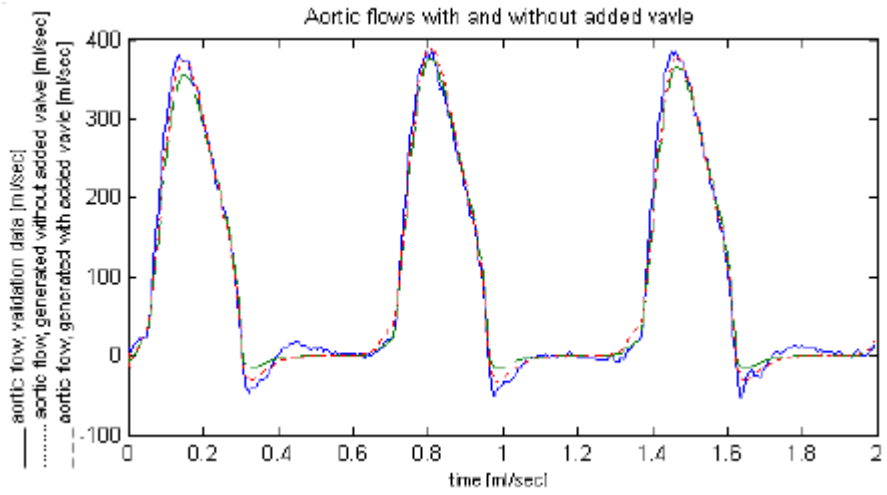


Figure 31: Comparison between generated aortic flow by the total arterial model with and without the added valve and the measured aortic flow.

The first effect of the added valve on the system is that it adds resistance when it is opened, which can be seen in that the peak flow is slightly lower. The second effect is that now there are two valves working together to keep the blood from flowing back into the ventricle.

The cooperating valves are stronger than the single valve so the back flow becomes less which is seen in that the dip when the valves close is smaller than it was before.

The second change is inserting the AVD in the model. The voltage to the AVD is at a constant level that is low enough so that some blood gets diverted into it and still high enough that it ejects all the blood it receives. Simulating this shows a few of the impacts of the AVD on the arterial system and what needs to be considered when designing the controller. The simulation result is shown in figure 32 for the flows and figure 33 for the pressures.

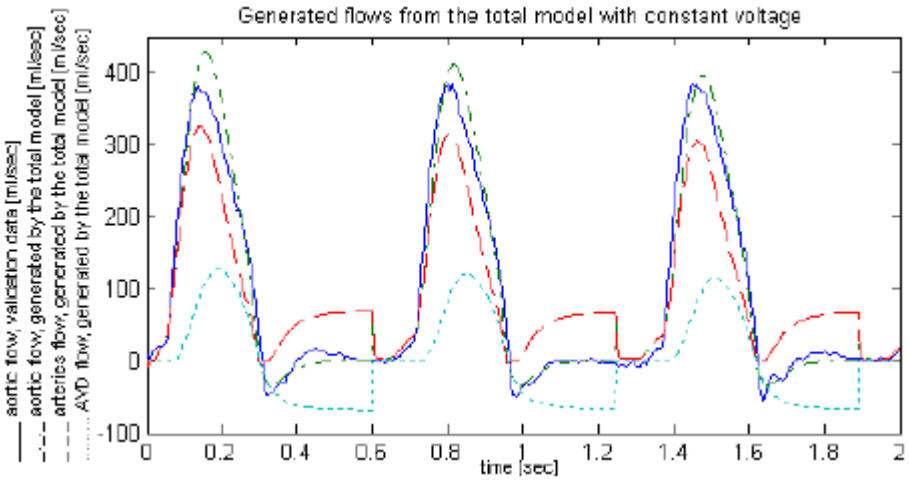


Figure 32: Validation aortic flow and the generated aortic flow, arterial flow and AVD flow when the voltage is set to constant -6 V.

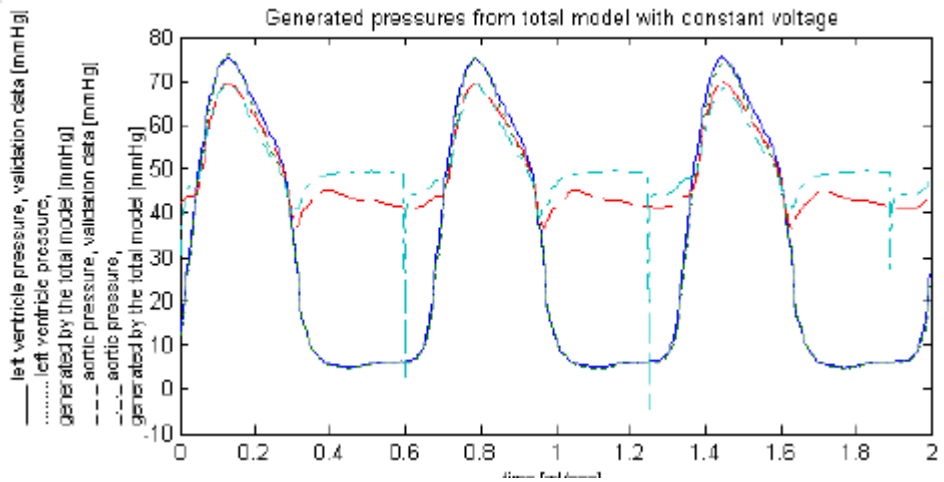


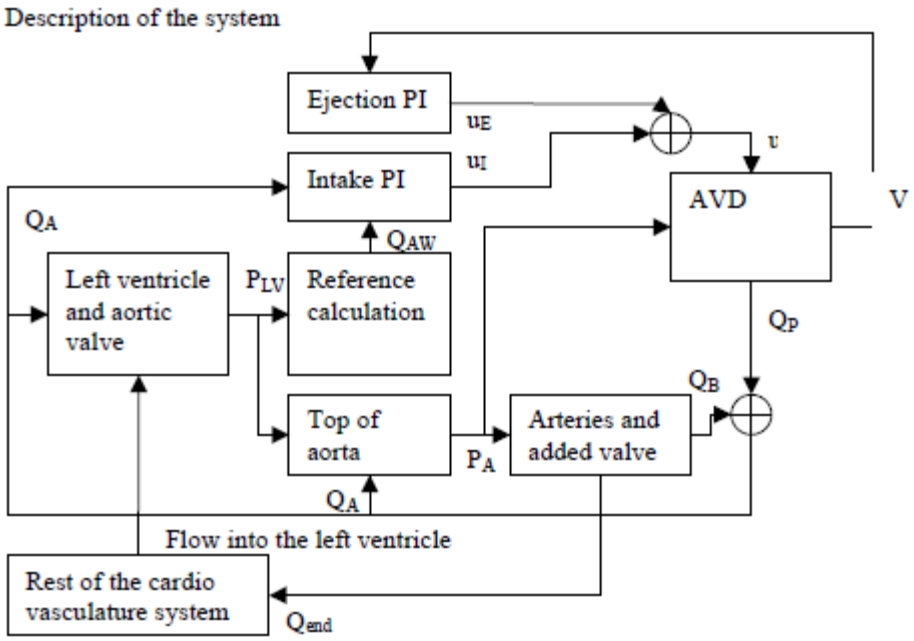
Figure 33: Measured left ventricle and aortic pressure plus the generated left ventricle and aortic pressure when the voltage is set to constant -6 V.

With the AVD taking in blood the load seen by the ventricle is lower and it ejects more blood in the first two beats which can clearly be seen. The amount in the following beats approach the normal, although with a slightly different profile because the load is different. Another affect of inserting the AVD that can be seen is the jump in aortic flow when it finishes ejecting. With a constant voltage as used here the flow out of the AVD will suddenly stop when it becomes empty. The blood flowing through the aorta has inertia that will then cause a drop in pressure in the aorta which will in turn cause a surge of blood from the ventricle. This pressure drop and surge could be harmful since the patient will be assisted by the device for months which means a great amount of beats.

3 Controllers

The objective of the controller is to make the load seen by the left ventricle be whatever is wanted, to eject the stored blood and to keep the filling and emptying synchronized with the ventricle. The objectives are divided in time so the controller can also be divided in time; create the correct load when the ventricle is ejecting and then eject the stored blood when the ventricle is filling up again. This division gives a total system with arterial system and AVD as shown in figure 34.

The only control signal available to achieve all this is the voltage to the AVD. With a constant high voltage the AVD won’t take any blood in at all and the ventricle will see the normal load. With a constant low voltage the AVD will drain the ventricle which will see no load at all and no blood will be returned to the arteries. With a constant medium voltage the AVD takes some blood in and ejects all of it when the ventricle is filling up which means a lower load should have been seen by the ventricle. This means that it should be possible to create any load in between the too high one and too low one.



For the controller to calculate the correct voltage it needs accurate online measurements from the arterial system and the AVD. The controller is constructed with the assumption that it can get any measurement it wants. However, to lower costs the number of measuring points is minimized. When the controller is constructed it is also assumed that there is no noise in the measurements; the effects of these are handled afterwards. Even if the measurements are considered ideal in that they have no noise they still need to be sampled for the digital controller to be able to use them so sampling is handled while constructing the controller. The sampling rate is assumed to be the same as for the identification data gathering, 400 Hz, even if the pre-sampling filters might not be the same.

3.1 Intake part of controller

The wanted load is a copy of the model of the systemic arterial system but with the desired parameter values; ‘R1W’ for the R1+RA arterial model parameters, ‘L1W’ for the L1+LA arterial model parameters, ‘C1W’ for the C1 arterial model parameter, ‘R2W’ for the R2+X arterial model parameters. These desired parameter values are therefore the true reference signals for the first goal. However, the only feedback the ventricle in the model

gets from the arteries is the aortic flow so if the flow response is changed the ventricle will experience that as a different load.

By generating the aortic flow the wanted load would have generated 'QAW' the ventricle will experience that as if the wanted load was the actual load. To calculate what this reference aortic flow should be the pressure in the ventricle and the wanted load are needed. The pressure is assumed to be an available measurement. The wanted load is a copy of the model of the systemic arterial system but with the reference parameter values and no AVD, added valve or separated constants in the aortic root model. Also the wanted load model has a simpler representation of the valve; a saturation that doesn't let the wanted aortic flow become negative. The equations for the wanted aortic flow is given below.

$$\frac{dQ_{AW}(t)}{dt} = \begin{cases} 0 \ , \ Q_{AW}(t) \le 0 \ \& \ \dfrac{P_{LV}(t) - P_{1W}(t) - R_{1W}Q_{AW}(t)}{L_{1W}} < 0 \\ \dfrac{P_{LV}(t) - P_{1W}(t) - R_{1W}Q_{AW}(t)}{L_{1W}} \ , \ otherwise \end{cases}$$

P_{1W} is the pressure produced by the compliance in the container.

$$\frac{dP_{1W}(t)}{dt} = \frac{Q_{AW}(t)R_{2W} - P_{1W}(t)}{R_{2W}C_1}$$

The equation for generating the wanted aortic flow is then transformed into discrete time since the controller works with discrete time. The transformation is done by using zero order hold approximation with a sample rate of 400 Hz7. The equations for the discrete wanted aortic flow are given below.

$$Q_{AW}[t] = \sum \frac{1}{400} \cdot \begin{cases} 0 \ , \ Q_{AW}[t-1] \le 0 \ \& \ \dfrac{P_{LV}[t-1] - P_{1W}[t-1] - R_{1W}Q_{AW}[t-1]}{L_{1W}} < 0 \\ \dfrac{P_{LV}[t-1] - P_{1W}[t-1] - R_{1W}Q_{AW}[t-1]}{L_{1W}} \ , \ otherwise \end{cases} ,$$

$$P_{1W}[t] = \sum \frac{1}{400} \frac{R_{2W}Q_{AW}[t-1] - P_{1W}[t-1]}{R_{2W}C_1}$$

The way to adjust the aortic flow is to alter the AVD flow. If for example the aortic flow is too small then the flow into the AVD is increased by altering the voltage to the motor.

$u_I[t] = P_I e_I[t] + I_I \sum e_I[t]$, eI is the error with the aortic flow.
$e_I[t] = Q_{AW}[t] - Q_A[t]$

The value of the voltage during the intake phase 'uI' is calculated by using a PI feedback on the aortic flow. The reference aortic flow is calculated as described above and compared to the measured aortic flow which means that the aortic flow needs to be measured.

The values for the gains in the PI are chosen as the ones that seem to best allow the controller to follow the wanted flow in simulations.

3.2 Ejection part of controller

The goal of the ejection part of the controller is to empty the AVD without disturbing the intake part. Also, the AVD flow should not be too high right before the AVD becomes empty to avoid a too big surge of blood from the ventricle. It's possible that a too high surge might damage the aortic valve since it would happen at every beat and the AVD would be implanted during a long time. Ejecting blood only marginally affects the aortic flow and through that the intake part so as long as the ejection is finished well before the intake starts the ejection part won't affect the intake part.

The ejection is preformed using the voltage the intake part sends and adding an ejection voltage 'uE'. The two voltages added together is the voltage 'u' that is sent to the AVD, according to;

$$u[t] = u_I[t] + u_E[t]$$

The ejection voltage starts getting added when the aortic flow is negative which means the intake part is finished. The ejection voltage then stops getting added when the AVD is empty or the aortic flow is positive again. The AVD should be empty before the aortic flow becomes positive but just in case it isn't the ejection voltage is stopped if the flow becomes positive, so

the intake part can work uninhibited. The next ejection part of the cycle can hopefully eject the blood that was left in the previous faulty ejection. If it can't and the volume builds up then something is seriously wrong with the AVD but all that will happen is that the AVD fills up and that the ventricle is forced to pump on it's own without assistance. Starting and stopping the adding of the ejection voltage is the same as forcing it to zero when it is stopped.

The value of the ejection voltage is calculated by using a PI feedback on the AVD flow 'QP'. The reference AVD flow 'QPWE' is calculated from the volume in the AVD 'V' by a constant plus the volume times a second constant. The first constant is to make sure all the blood gets ejected by having a minimum reference flow. The second makes the reference flow proportional to the volume so that a higher flow is wanted when the volume is high. The values of the gains in the PI are chosen so that the flow doesn't jump too high when the AVD has taken in a lot of blood, that the surge of blood from the ventricle is small when the AVD becomes empty and so that all the blood still gets ejected. The equations for generation the ejection voltage is presented below;

$$u_E[t] = \begin{cases} P_E e_E[t] + I_E \sum e_E[t] \ , \ opened \ aortic \ valve \\ 0 \ , \ closed \ aortic \ valve \end{cases}$$, e_E is the AVD flow error.

$$e_E[t] = \begin{cases} Q_{PWE}[t] - Q_P[t] \ , \ opened \ aortic \ valve \\ 0 \ , \ closed \ aortic \ valve \end{cases}$$

$$Q_{PWE}[t] = -(10V[t] + 150)$$

Whether the aortic valve is opened or closed is determined by looking at the aortic flow;if it has been under a certain value for all of the last nine samples it is considered closedand if not it is considered opened. Hence;

$$\max(Q_A[t], Q_A[t-1], \ldots, Q_A[t-8]) \begin{cases} > 25 \ \Rightarrow \ opened \\ \leq 25 \ \Rightarrow \ closed \end{cases}$$

The measurement of the AVD flow that is needed is achieved using an approximation of the derivative of the volume. The measurement of the volume is in turn achieved by measuring the position of the AVD piston and using the known area of the AVD container. This might be a more noisy way than measuring the AVD flow directly but for the ejection the

important thing is that the volume becomes empty so some noise won't make that much difference and this way the AVD flow won't need to be measured at all.

$$Q_P[t] = \frac{100z - 100}{z - 0.7788} V[t]$$

To insure that there is no drift and that every ejection starts in the same manner the integration part of the ejection controller is reset before every new ejection starts.

3.3 Filtering the measurements

The controllers above need the measurements of the left ventricle pressure, the aortic flow and the AVD position. To lower the level of noise and lessen the effects of aliasing the measured signals need to be filtered before they are sampled. In this computer model the filter design chosen is standard Bessel filters since these have linear phase. The 8th order 60 Hz lowpass filter that was used for this purpose when the identification data was gathered is great for offline use; it should remove almost all noise over 60 Hz and just about eliminated the alias effects. However the higher the order and lower the cut of frequency the longer the delay is in analog filters. This does not matter when the data is used offline for identifications but when it is used online for the purpose of giving accurate information to the AVD it becomes crucial. To show the effect of the delay of different filters simulations of the whole system were preformed. To isolate the effects of delay from those derived from noise the measurements are seen as correct with zero noise. Simulation results with different order filters as well as no filter as a reference are showed in figure 35.

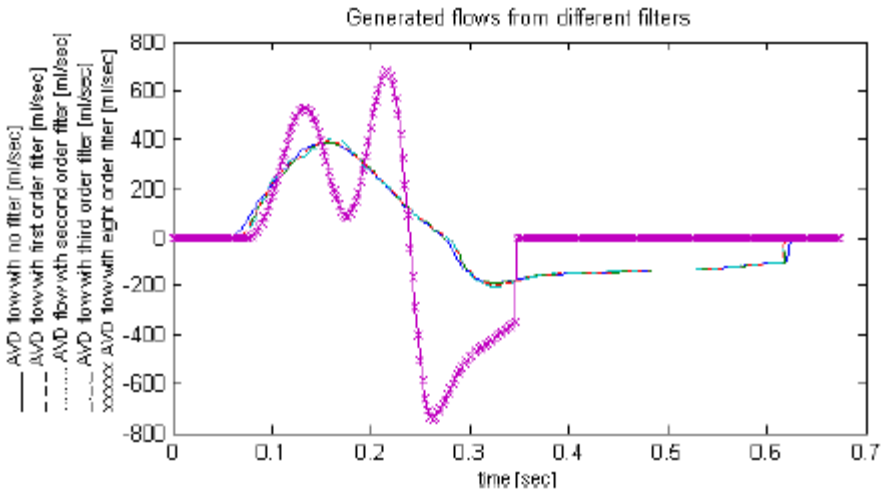


Figure 35: AVD flows with different pre sample filters. 8th order Bessel 60 Hz low pass filter is clearly too slow to be used for this application. Even when the order is lowered to 3 it is still to slow and the effects can clearly been seen. The results with the 2nd order filter are adequate and with the 1st order one they are good. Using a higher cut off frequency would improve the speed of all the filters.

The cost of using a faster filter is that its ability to filter away noise is worse. To know which order and cut off frequency would be optimum for speed and noise reduction the correct noise characteristics of the measured signals are needed.

3.4 Stability

To investigate the stability of the system it is divided into two parts in time; one for each controller. By assuming that the two parts don't somehow work together to create an unstable system the total system is stabile if the two parts are stabile. The ejection controller almost reaches a steady state and also it ends with the container being empty each time so it should not be able to affect the intake controller to such a degree that instability is created.

When the AVD is taking in blood the ejection controller is not contributing so only the intake controller affects the stability. When the AVD is ejecting the aortic flow is close to constant. This makes the intake controller's output close to constant when compared to the

ejection controller's output. So when the AVD is ejecting the stability is only affected by the ejection controller.

The division in time also works to cancel the nonlinearity of the aortic valve, when the AVD is taking in blood the valve is opened and when it is ejecting blood the valve is closed.

3.4.1 Stability of the intake controller

The system uses the compliance of the left ventricle as a driving input which makes it nonlinear. Also the rest of the system is circular and over determined; the pressure in the left ventricle affects the aortic flow through the equations of the body but the pressure is affected by the volume which is the integral of the aortic flow. Even if the arterial system without the AVD can be assumed to be stabile (a normal heartbeat is quite repetitive and doesn't exhibit any signs of instability) the introduction of the AVD might ruin it. It is not hard to increase the P gain so that the system become unstable as shown in figure 36.

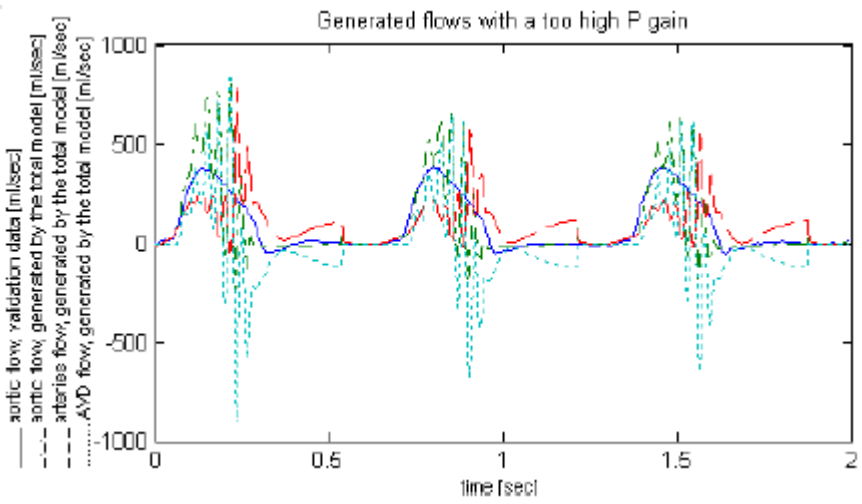


Figure 36: Aortic flow from when the system is unstable.

With a low enough P gain that instability is removed which suggests that the system is stabile from beat to beat due to the AVD involvement. The fact that there are so many different VADs on the market also suggests that the system is stable.

3.4.2 Stability of the ejection controller

The ejection controller gains are chosen so that the piston in the AVD only moves in one direction during its phase. This means that the only way the system can be unstable is if the piston and flow accelerates in that one direction. However the piston will eventually reach the end of the container when the AVD is empty and it will not start filling again until the intake phase. This means that the system in reality is stabile even if it was accelerating. In addition even if the AVD was not emptied the ejection part of the controller is reset before the next ejection phase so no instability can grow over several cycles.

3.5 Comments

The constructed controller is very simple and it doesn't make the load seen by the left ventricle exactly what is wanted but it is close enough as shown in figure 37.

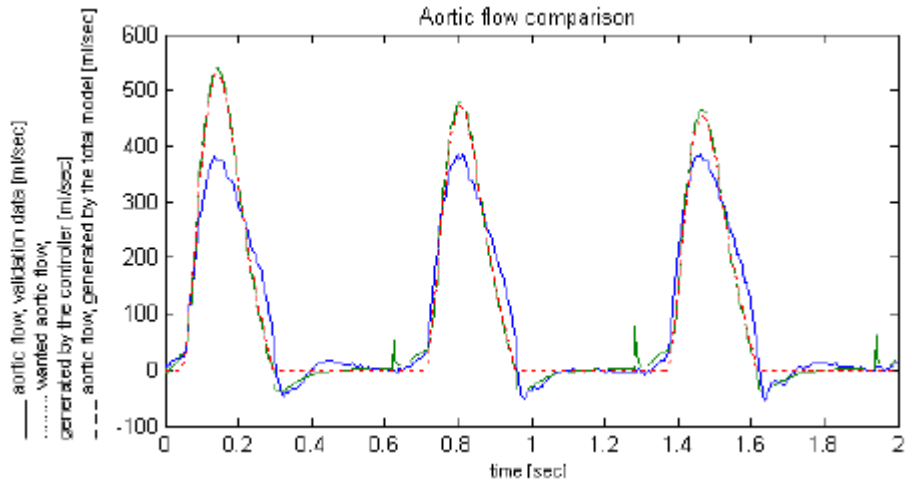


Figure 37: Aortic flows when the wanted load is given by putting the initial resistance to 60% of the normal one.

The most important thing with the finished AVD is not that it can generate the exact load wanted but that it can generate several different loads and that it is consistent. The controller does not make assumptions on the compliance profile of the heart, the actual load of the arteries or even the dynamics of the AVD. Making such assumptions and using prediction or repetition in the controller should make for better results as long as the heart rate is constant. However the heart rate does change and could do so quickly so the predictive or repetitive controller would have to be able to handle that and very fast.

The constructed controller should not be affected by this at all other than the ejection might need to be made faster with a faster heart rate so the AVD can be emptied in time.

4 Simulations with the controller

With the controller the AVD can be used to make the left ventricle see what ever lower loads than the normal one that is wanted. Simulations were made with the load being a copy of the normal one except for the resistance in the beginning of the arteries. This resistance was set at 80% and 40% of the normal one. The generated results are shown in figures 38, 39 and 40 for when the resistance is

80%. The results when the resistance is 40% are shown in figures 41, 42 and 43.

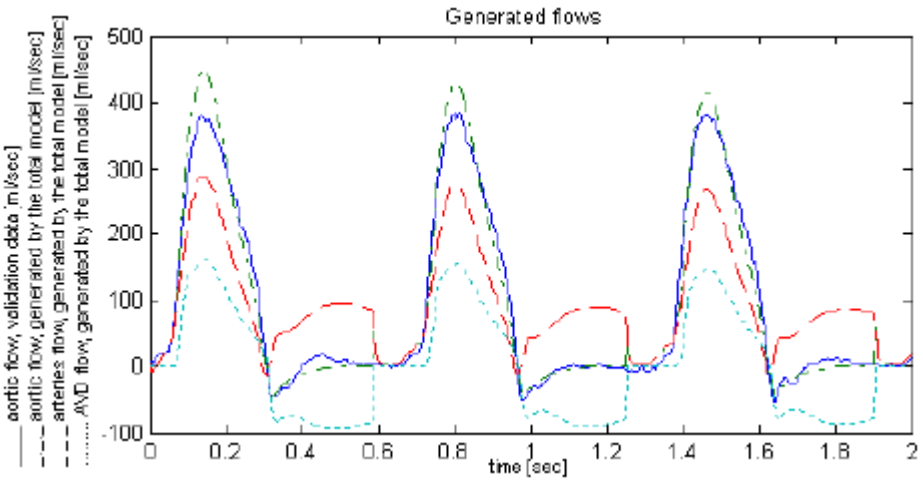


Figure 38: Generated flows with resistance at 80% of normal.

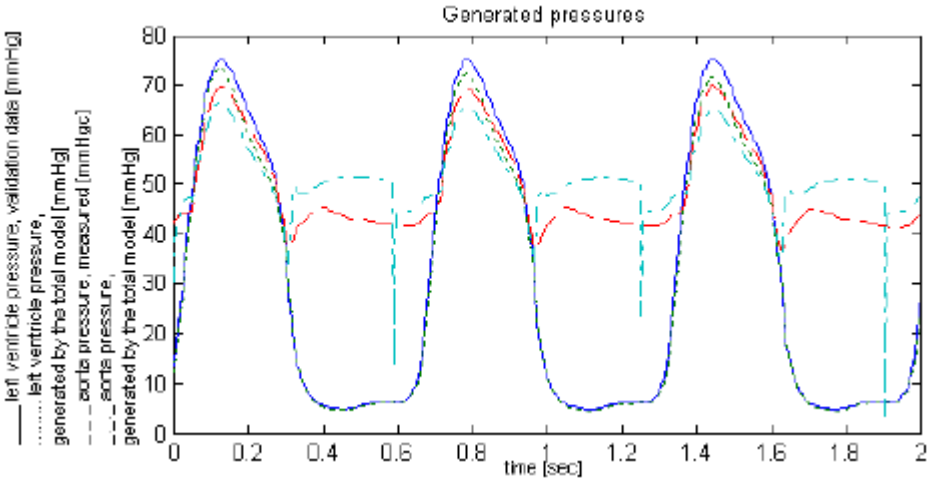


Figure 39: Generated pressures with resistance at 80% of normal.

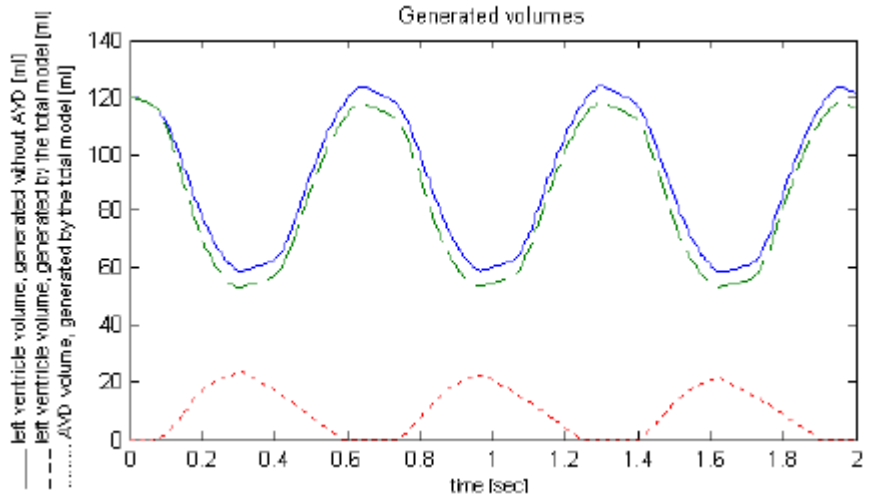


Figure 40: Generated volumes with resistance at 80% of normal.

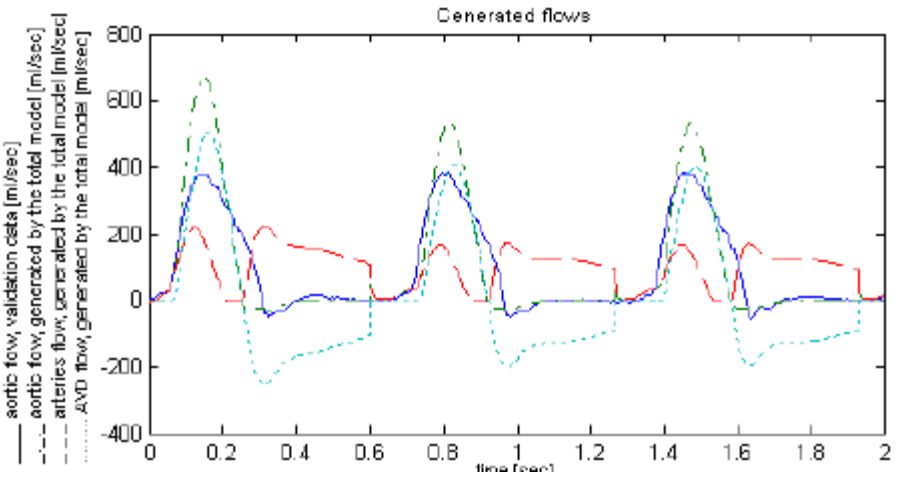


Figure 41: Generated flows with resistance at 40% of normal.

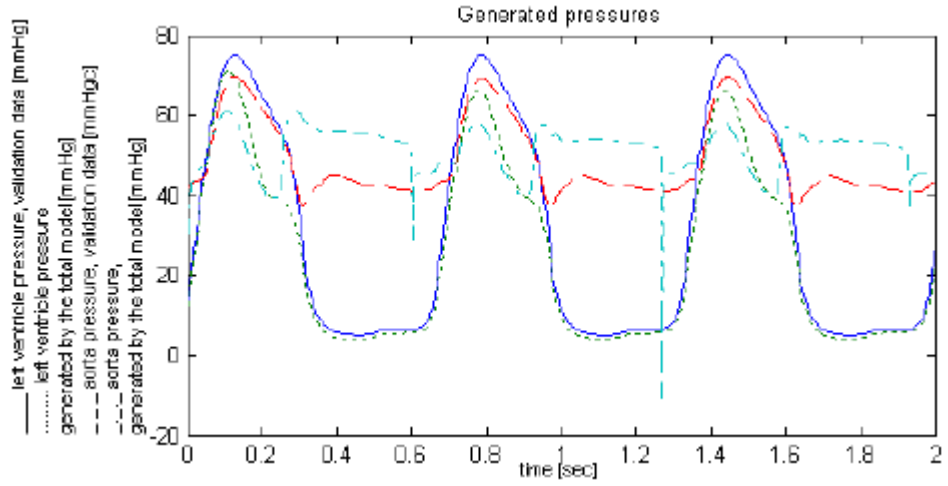


Figure 42: Generated pressures with resistance at 40% of normal.

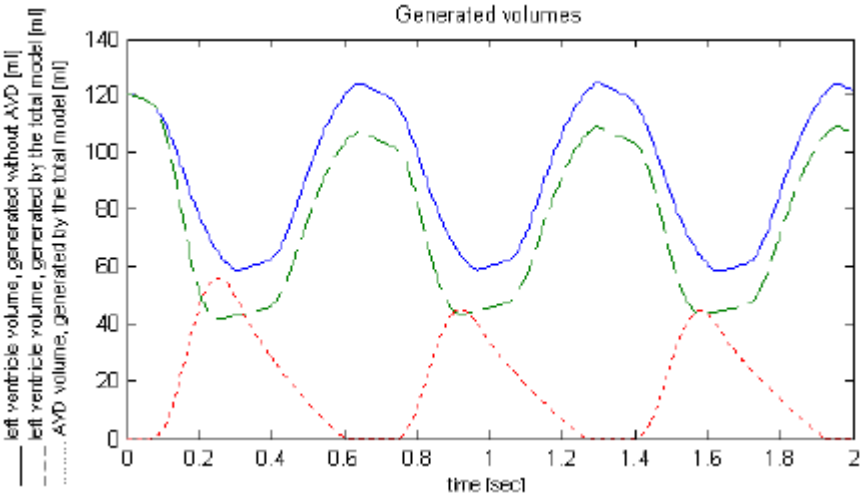


Figure 43: Generated volumes with resistance at 40% of normal.

With both simulations the lower wanted load creates a big change in the first beat but that that everything stabilizes at new values after a few beats.

When comparing the simulations using different wanted resistance it is clear that the one with 40% of normal affects the ventricle more than the 80% one. The affects include higher peak aortic flow but shorter ventricle ejection time. This means that the flow profile was changed with the use of the AVD but that the normal amount of blood gets ejected (after stabilization). The volume of the left ventricle becomes lower which in turn makes the ventricle pressure lower. That the pressure is lower means that the ventricle doesn't have to work as hard which is the point of the AVD. It is not obvious just how to measure how much easier it gets for the left ventricle to eject. Besides just looking at the pressure it is also possible to look at the integral of the pressure times the flow. This integral tells how much energy is needed to eject the blood and it is shown in figure 44. It can clearly be seen that with the lower the load the lower the energy needed.

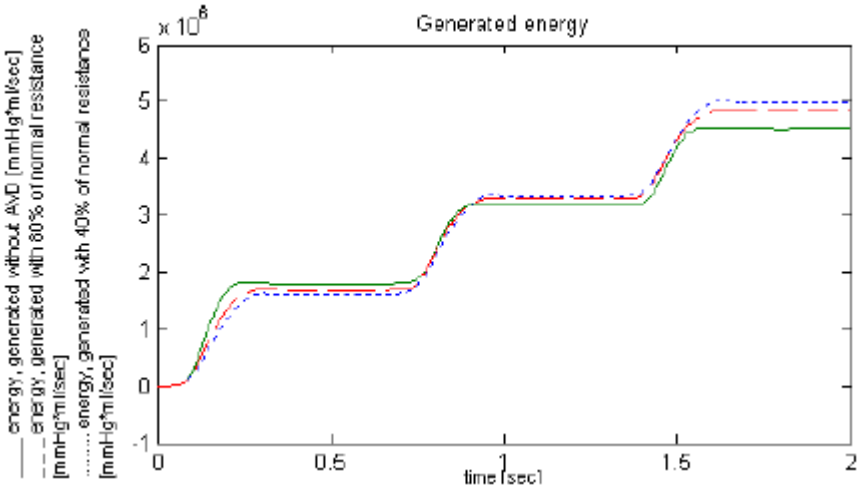


Figure 44: Generated energy to eject the blood from the left ventricle when using different wanted loads.

The AVD can not be used to make the load seen by the ventricle to be noticeably higher than it is without the AVD. To do this would require that the AVD ejected blood when the ventricle is ejecting and then take blood in when the ventricle is taking in blood which would

create a backflow. If the wanted resistance in the beginning of the aorta is set to 140% of normal with the current AVD the result is as shown in figure 45. As can be seen the flow is at first disturbed by the AVD but after the first beat the AVD isn't affecting the arterial system at all; it is pushing at an already empty container.

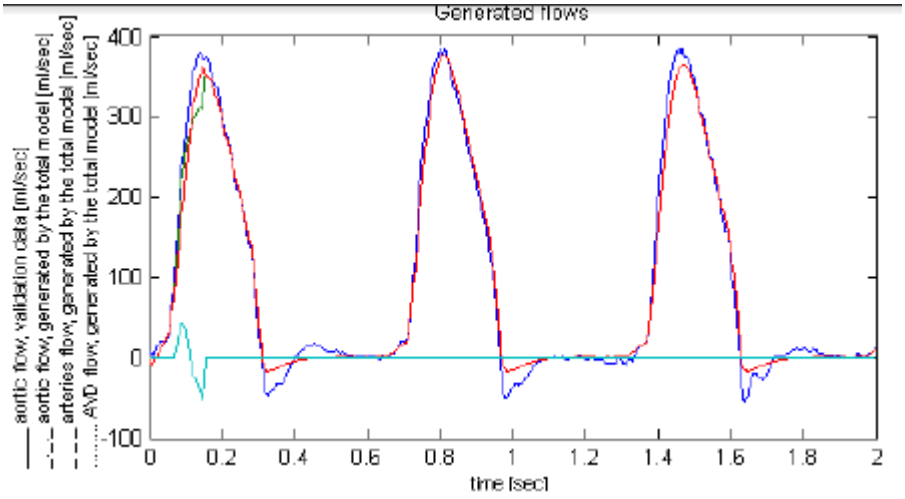


Figure 45: Generated flows with resistance at 140% of normal.

The added valve is not needed if the wanted load is reasonably high. With the resistance at the beginning of the aorta at 40% of normal the peak backflow in the arteries flow is at the same level as without the AVD. If a lower wanted load than that is desired the backflow will become bigger and bigger. With the resistance at 20% of normal the peak backflow is about 4 times higher than without the AVD which could be dangerous. The flow with the 20% resistance is shown in figure 46.

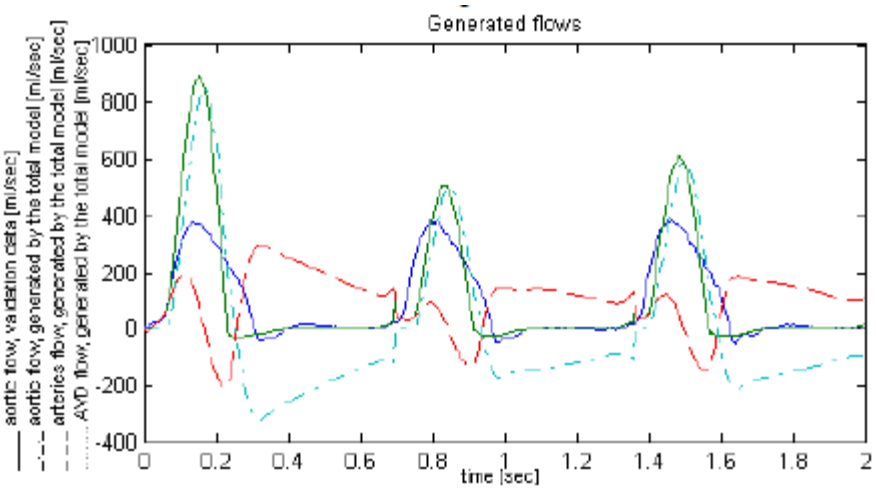


Figure 46: Generated flows with resistance at 20% of normal and no added valve.

The controller has been built without considering a maximum current in the motors electrical part. When the wanted load resistance is set to 40% of normal the current in the motor is very high as shown in figure 47. It is doubtful the ordinary type of motor used in the model could handle such a high current so something needs to be changed. A different motor that is built for high currents should be used. There is also the possibility of adding gears with will make the current lower as the motor doesn't need to push as hard but instead the motor needs to be faster.

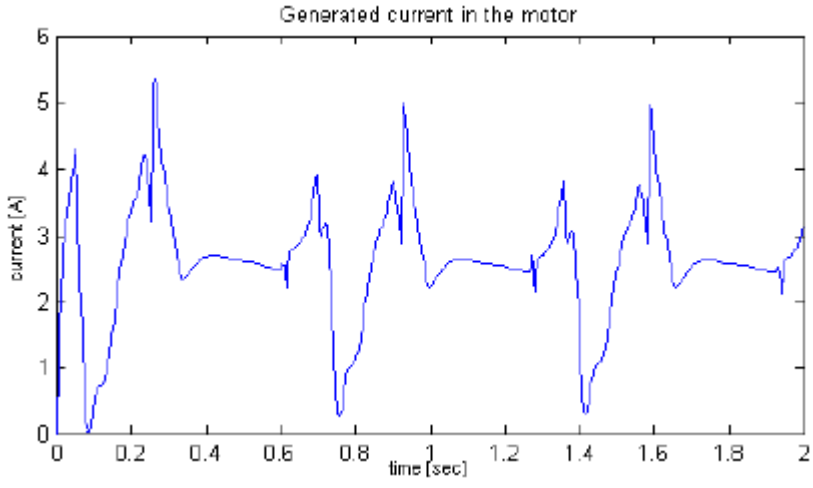


Figure 47: Current generated by the model in the motors electrical part.

5 Results

The main purpose of this thesis was to derive a model that can simulate an implanted AVD and the affect it would have on the arterial system and this main purpose has been achieved.

The AVD is a slightly different type of assist device for patients with injured or sick hearts. It is implanted in parallel with the arteries seen from the left ventricle. When the ventricle is ejecting the AVD takes in blood which makes the load the ventricle experiences lower. When the ventricle is filling up again the AVD ejects the blood it just took in, so that it is ready for the next beat.

5.1 AVD model

The AVD was modeled using standard models for the servo motors, the tubes, the container and the piston. Even though the model structures are mostly linear and ignore many probable nonlinearities they should be correct enough for these studies. The values for the different parameters were obtained from motor specifications and specifications on how the AVD would be built so there is no way of telling how accurate they are. Incorrect values would mostly affect the resistance and inertia of the AVD so if these happen to be too big the motor can just be exchanged for a stronger one. If the values used are somewhat close to the actual ones then something needs to be changed in the AVD design. The resistance and inertia that the motor feels are too great which can be seen in the motors current. The changes could include gearing, wider tubes and/or smaller container base area.

5.2 Arterial system model

The arterial system was divided into four parts for modeling; the arteries, the root of the aorta, the aortic valve and the left ventricle. The models are structured by using physical modeling and the parameter values identified from data by minimizing the prediction error. The

data comes from measurements taken from a patient of the left ventricular pressure, aortic pressure and aortic flow.

The arteries were modeled as a tube with fluid inertia and resistance followed by a container with compliance and finally a tube with resistance. The pressure at the end of the last tube is the flow through it times a constant. The equation is the same as for the four element Windkessel model but the explanation is slightly different. With this difference the pressure at the end is not zero and so it can be connected to a model of the veins that bring the blood back to the heart without using negative pressure. The model produces next to perfect predictions of the aortic flow from aorta pressure measurements.

Using a less complex model without fluid inertia gives good but inferior predictions so fluid inertia is defiantly needed to explain the load the left ventricle sees. The root of the aorta is modeled as a continuation of start of the arteries which is natural since the arteries start with the aorta. The model consists of a tube with fluid inertia and resistance that adds to the fluid inertia and resistance of the first tube in the arteries model. This model also produces next to perfect prediction when the aortic valve is opened. Validation can not be done for the time intervals when the valve is closed.

The effects of the opened aortic valve are included in the aortic root model. The closed valve is thought of as a drum and idealized as if it was a flat piston with a spring and damper behind. Only the data from the time intervals when the valve is closed are used to identify the compliance and resistance values of the valve. The predictions from this model are not as perfect as from the two previous ones. Most likely a better model structure for the valve can be achieved with more complexity and/or nonlinearity. The current model does however predict the negative aortic flow when the valve closes and even the slight positive aortic flow just before the ventricular pressure exceeds the aortic pressure. That it can predict these two phenomenon makes it much better than just using an on/off type valve.

The model of the left ventricle is a chamber with varying compliance. Using either the ventricular pressure or the aortic flow as the input would be worse since both of these should change with a change in load so the only other option is to use the compliance as input. The compliance is merely a recalculated input to the model so there is no way of validating it with the

available data. The effects of using the compliance as input does lower the ventricle volume as it should so it works in the right sort of way.

5.3 Controller

The Controller for the AVD is made up of two controllers; one the deals with the intake of blood to the AVD and one the deals with the ejection. The goal of the intake controller is to make the left ventricle see the desired load. The way to make the ventricle see the wanted load is to alter the aortic flow to what the wanted load would have generated. In other words a reference aortic flow is calculated using a wanted load and measurements of the ventricular pressure. The wanted load is a copy of the arteries model where the different parameter values can be changed to desired values. A PI controller then alters the voltage to the motor in the AVD so that the aortic flow becomes the reference aortic flow.

Important for the intake controller is that the measurements are made with a low delay which means the pre sampling filter can not have too high order. This in turn puts high requirements on the measurement devices.

The ejection part of the controller is only active during the time the aortic valve is closed. It is basically a PI controller that has a reference AVD flow that is meant to eject all the blood before the left ventricle starts ejecting again without letting the AVD flow become to high or jump to fast. The reference AVD flow is also needs to be made so that the AVD flow isn't too high just before the AVD becomes empty. A too high flow at that point results in a surge of blood from the ventricle due to the inertia of the blood. Every time the ejection controller activates it is reset so no errors can build up over several beats.

The intake part is still running during the time the ejection part is active but it measures the flow at the root of the aorta above the AVD insertion so it doesn't get affected much when the AVD ejects blood.

The constructed controller does not produce the exact load seen by the ventricle that is wanted but it should come close enough for practical purposes. The controller also doesn't assume anything about the workings of the ventricle or arteries so it isn't affected if the heart rate

changes except that the ejection of AVD blood might need to be made faster if the heart rate is faster.

5.4 Simulations

The computer model that has been constructed was used to simulate different conditions and different wanted loads. The resulting affect on the left ventricle should only be taken as a guide to what would really happen. How the ventricle would really be affected by a lower load can not be validated from the available data. The numerical values of the pressure, flow and volume might therefore be wrong, but the general behavior and trends should be correct.

The most important thing that can be seen from the simulations is that lower and lower wanted loads produces lower and lower left ventricular pressure and volume just as intended. The lower load also alters the profile of the aortic flow; the lower the load the higher the peak flow. Lower load also means shorter flow time so that the normal amount of flow is ejected from the ventricle.

Simulations without the added valve implanted reveal that the AVD functions well for slightly lower wanted loads compared to the true arteries load. If the wanted load is lower then there will be backflow from the arteries into the AVD so the added valve is needed to prevent this.

6 Concluding remarks

6.1 Conclusions

A model that can simulate the interaction between the arterial system and an implanted AVD has been derived. Simulations with this model shows that it is possible to alter the load the left ventricle experiences to whatever is wanted using an AVD. In the model the ventricle's volume and pressure decreases with lower and lower experienced load. The amount of aortic flow is the same as without the AVD but the profile changes; the peak flow is increased and the time of the flow decreased. The same average flow and a lower pressure mean that the work

done by the ventricle is lower. Making the work lower and easier for the ventricle to eject is what the AVD is meant to do. In simulations it is also shown that an extra valve has to be implanted down-flow of the AVD intake tube if quite low loads are desired.

The AVD is modeled as a servo motor that pushes a piston. The piston is inside a container with tubes in the other end. The tubes are connected with the aorta and each has a valve that force the blood to flow in one direction only; in through one and out through the other. The parameter values in the model of the AVD could not be validated with the data available but the model can easily be improved with the correct values.

The Arterial system is modeled in three parts; the left ventricle, the aortic valve and the arteries. The left ventricle is modeled as a compliance chamber where the compliance profile is constant. The aortic valve is modeled as a linear drum that appear when the aortic flow starts to be negative and disappear when the pressure in the ventricle is greater than the pressure right after the valve. This valve model is a great improvement over having an on off valve model as it reproduces the dip in flow when the valve closes and also the positive flow before the ventricle pressure is higher than the aorta pressure. The arterial model gives identical equations as the 4 element Windkessel model and it is shown that the inertia of the blood has to be included to get good a model. The model of the left ventricle could not be validated with the data available but the model can easily be improved with the correct values.

The designed controller is simple but produces adequate results and it is able to make the AVD change the load fast enough. However, with this simple controller it is shown that any controller used needs to be fast which includes a fast pre-sampling filter. The simple controller also shows that the controller can easily be divided in two; one for intake and one for ejection.

6.2 Future work and recommendations

The AVD has not even been built yet so there is much work left to been done. Eventually the product might even be commercially and getting there is a long journey of improvements, tests and certifications.

6.2.1 Improve AVD

The motor chosen for the AVD in this preliminary study can't be used in the final version of the AVD so a new and better one needs to be found. The motor used here is way too big to be implanted in a patient and in addition its characteristics are not suited for the work the motor in the AVD will do. The current motor is designed for voltages up to 200 V but in this application it only works with voltages under 10 V and the current is very high. A motor more suited to the low speed and high force would be a better choice.

The problem of the motor can be somewhat solved by using gears for the motor, this would lower the current but it might also slow down the AVD reaction time. Another way of maybe solving the problem would be to change the base area of the AVD container; this would also change the relation between the speed and force for the motor (this would probably mean that the piston and motor would have to be connected by other means than an arm). The base area in the model is however the same one as used in an existing VAD so it can't be all wrong. The area of the tube(s) leading blood into the AVD can also be changed to tune the AVD, a larger area would mean less resistance and inertia there. Further simulations could be done to research which choice of motor characteristics, gearing (arm length), container area and tube area is optimal. What is optimal depends on the size of the AVD (should be small), energy consumption (low), ability to alter the load seen by the ventricle (fast reaction time).

An idea to lower the energy consumption would be to add a mechanical source of force to the AVD piston (for example a spring) that would counter the base pressure in the aorta.

6.2.2 Improve the models

The model of the arteries is quite good with the exception of the aortic valve which could be seen as adequate. With further studies a more complex and better model for the valve might be able to be constructed. However, the model of the left ventricle is questionable.

There is no way to validate it with the current data and most likely the ventricle will react to a change in load by changing the compliance profile at least a little. The only way to investigate this is to do measurements with different loads and identify how the ventricle reacts. This investigation should be done to really be able to simulate the interaction between the ventricle and AVD. All these models concentrate on the arteries side of the cardio vasculature system. To fully understand how the interaction between the AVD and the whole cardio vasculature system measurements need to be made on other places in the heart and blood vessels. With these measurements the whole cardio vasculature system might be able to be identified and this would benefit not only the studies with the AVD but also all other studies that relate to the cardio vasculature system and especially the heart.

The model of the AVD needs to be improved. Several of the values could be incorrect and the structure of the model might not be perfect. The best way to model it would be to build it and then conduct measurements and identify parameters similar to how the arteries were identified here. With a correct model of the AVD the controller can be improved easier.

The model of the arterial system was designed to simulate the interaction with an AVD, but it could very easily be modified to simulate the interaction with a continues VAD (under the assumption that the flow is kept constant). With this model those to types of assist devices can be compared.

6.2.3 Improve the controllers

The controller used here is quite simple, a better one could surely be constructed when more is known about the cardio vasculature system and a correct model of the AVD is available. Suggestions for this would be to use the repetitiveness of the heart beats and predict when the beat will come. If it could be predicted without error when the beat would come the controller can use this and produce a much better result than the current one.

The measurement the controller uses is very important for the result. Faster sampling and less noise would of course improve this or any controller used. Also other measurements can be

used than the ones in the model. The flow into the AVD could be measured together with the flow into the arteries to improve or substitute the measurement of the aortic flow. The current in the electrical part of the motor can also be measured which would enable the use of a feed forward controller.

A feed forward controller would be useful if the resistance and inertia in the AVD (including the tube and piston) is reduced with gears or change in container area. Whatever controller is chosen in the end the stability of the whole system needs to be proved. Without knowing if it is stabile or not it isn't possible to be sure the controller will behave as it should.

6.2.4 Studies using the AVD

When the AVD is completed it can be used to research how the ventricle reacts to different loads. The instantaneous reaction can be identified and used to improve the AVD models (simulations) and the controller. The reaction over long time can be used to research how patients with injured or sick ventricles can be healed and what loads are best for different healings or assistances.

The working AVD can also be compared to VADs. Especially the advantages and disadvantages with taking the blood from the aorta instead of the left ventricle should be researched. In theory there might not be much difference from taking the blood from either place but it should be investigated. Also the advantages and disadvantages between them when they are explanted and the patient's chances for recovery should be researched. The difference in explanting is the fixing of the hole in the ventricle the

VADs needs and the extra valve the AVD needs. Whether or not to leave the extra valve in place after explanting the AVD is also a question that needs to be studied; the valve adds resistance so it increases the load seen by the ventricle but it might be good to have it in place if the patient suffers relapse and the AVD needs to be implanted again.

A study that can be done using only the identification code here is to identify models for several different patients. The parameter values can then be compared with the patients' physical health. If there are connections this might be useful to treat other patients in all sorts of ways.

Appendix

7.1 Matlab code for identification

To run the identification code have the files below in the same folder and execute ‘human_data.m’ in MatlabR 6.5 with the Identification Toolbox installed.

Identification code files;

Patient_003_001.mat

human_data.m

body_idgrey_1.m

body_idgrey_2.m

body_idgrey_W4.m

(measurement data, not included in the appendix)

body_idgrey_W4_fromheart.m

body_idgrey_valveW4

The code used to identify the parameters in the SimulinkR model is intended to be used with the given data, however the code can easily be reused for other persons' or animals' data. The only requirement is that the data includes measurements of the aorta flow, the left ventricle pressure and the root aorta pressure. To use the code with other data some smaller changes need to be made; a good start point, the number of samples per beat and good start and end points for when the aortic valve is opened and closed needs to be found from inspecting the new data. Also different start estimations might need to be tested to make sure the identification finds the right minimum.

7.1.1 Code for human_data.m

```
clear; % clearing the matlab workspace
```

```
%-------------------------------------------------------------------------

% Adjusting the data for identifying,

% Make sure the data has as many points.

%-------------------------------------------------------------------------

load Patient_003_001.mat   % loading identification data Ts=1/400; % sample rate.

% data point 166 is chosen as start data so that initial values are easier % to approximate.

start_data=166;

Q_A=AoF(start_data:length(AoF)).*(1000/60); % renaming the aorta flow

P_A=AoP(start_data:length(AoP));   % renaming the aorta pressure

P_LV=LVP(start_data:length(LVP)); % renaming the left ventricle pressure

driving_pressure=max(P_LV,P_A);   % approximate driving pressure all the way from

the LV. last_est_data=floor(263.82*11);   % using that 22 full beats on 5804 samples

% gives 263.82 samples per beat, 11 full beats is used for the estimation data.

% All the models have Aorta flow as the output.

Yest=Q_A(1:last_est_data);

Yval=Q_A(last_est_data+1:length(Q_A));









% when using a constant flow as input to the model this flow is used.
```

```
Q_average_est=zeros(length(Yest),1)+sum(Yest)/length(Yest);

Q_average_val=zeros(length(Yval),1)+sum(Yval)/length(Yval);


%---------------------------------------------------------------------
```

% IDENTIFYING MODEL 2, called model A in the thesis text % Models for heart pressure and aorta pressure to flow.

```
%---------------------------------------------------------------------
```

% Input signals; Aorta pressure and constant flow at "end" of body

```
Uest1=[P_A(1:last_est_data) Q_average_est];

Uval1=[P_A(last_est_data+1:length(P_A)) Q_average_val]; %make iddata vectors.

est_data1=iddata(Yest,Uest1,Ts);

val_data1=iddata(Yval,Uval1,Ts);

pars2= [-0.1     1          35]; % start estimates

aux2=[]; % constant parameters

model2=idgrey('body_idgrey_2',pars2,'c',aux2); % set up model

IdentModel2=pem(est_data1,model2);   % minimize prediction error

% naming the identified values

R1_2=-1/IdentModel2.c(1,1);

C1_2=-IdentModel2.b(1,2);

P1_2=IdentModel2.x0(1);

% validation  model 2

[YH2,FIT2] = compare(val_data1,IdentModel2);
```

```
FIT2

corrcoef2=corrcoef(val_data1.y,YH2{1}.y)

loss_func2=sum((val_data1.y-YH2{1}.y).^2)/length(val_data1.y)

FPE2=loss_func2*((1+3/length(val_data1.y))/(1-3/length(val_data1.y)))

%--------------------------------------------------------------------

% IDENTIFYING MODEL 1, called model B in the thesis text

% Models for aorta pressure to aorta flow and "LV" pressure to aorta flow.

%--------------------------------------------------------------------

% same input is used as in model 2

% approximated inertia for start estimate

aorta_length_guessed= 0.2; % guessed aorta length, in meters

aorta_diameter_guessed= 0.020; %guessed aorta diameter

aorta_area_guessed= pi*(aorta_diameter_guessed/2)^2;      % guessed aorta area in meters aorta_blood_mass_guessed= aorta_area_guessed*aorta_length_guessed*1000; % mass of the blood in the tube.

aorta_inertia_guessed=  aorta_blood_mass_guessed/(aorta_area_guessed^2);      % in SI units L_init=aorta_inertia_guessed/(133*10^6);   % in model units

pars1= [R1_2/L_init    1/L_init   0.01/C1_2  P1_2]; % start estimates, using values from

model 2

aux1=[Q_A(1) ]; % constant parameters

model1=idgrey('body_idgrey_1',pars1,'c',aux1); % set up model

IdentModel1=pem(est_data1,model1); %minimize prediction error
```

```
% naming the identified values

R1_1=IdentModel1.a(1,1)/IdentModel1.a(1,2); L1_1=-1/IdentModel1.a(1,2);

C1_1=1/IdentModel1.a(2,1);

P1_1=IdentModel1.x0(2);

% validation 1

[YH1,FIT1] = compare(val_data1,IdentModel1);

FIT1

corrcoef1=corrcoef(val_data1.y,YH1{1}.y)

loss_func1=sum((val_data1.y-YH1{1}.y).^2)/length(val_data1.y)
FPE1=loss_func1*((1+length(pars1)/length(val_data1.y))/(1-

length(pars1)/length(val_data1.y)))

%------------------------------------------------------------------------

% IDENTIFYING MODEL W4, called model C in the thesis text % Models for aorta pressure to aorta flow.

% Know as a 4-element windkessel model.

%------------------------------------------------------------------------

% input signals

P_end_W=0;

P_end_est=zeros(length(Yest),1)+P_end_W;

P_end_val=zeros(length(Yval),1)+P_end_W;

UestW=[P_A(1:last_est_data) P_end_est];

UvalW=[P_A(last_est_data+1:length(P_A)) P_end_val];
```

```
%make iddata vectors.
est_dataW4=iddata(Yest,UestW,Ts);
val_dataW4=iddata(Yval,UvalW,Ts);

parsW4= [R1_1/L1_1 1/L1_1 1/C1_1 1/(R1_1*C1_1) P1_1 0]; % start estimates,
using values from model 1
auxW4=[Q_A(1) ]; % constant parameters
modelW4=idgrey('body_idgrey_W4',parsW4,'c',auxW4); % set up model
IdentModelW4=pem(est_dataW4,modelW4); %minimize prediction error
% naming the identified values
R1_W4=IdentModelW4.a(1,1)/IdentModelW4.a(1,2); L1_W4=-1/IdentModelW4.a(1,2);
C1_W4=1/IdentModelW4.a(2,1);
R2_W4=IdentModelW4.a(2,1)/(-IdentModelW4.a(2,2)); Pcap=IdentModelW4.b(2,2)/(-IdentModelW4.a(2,2)); P1_W4=IdentModelW4.x0(2);
Q_A_W4=IdentModelW4.x0(1);
% validation W4
[YHW4,FITW4] = compare(val_dataW4,IdentModelW4); FITW4
corrcoefW4=corrcoef(val_dataW4.y,YHW4{1}.y)
loss_funcW4=sum((val_dataW4.y-YHW4{1}.y).^2)/length(val_dataW4.y)
FPEW4=loss_funcW4*((1+length(parsW4)/length(val_dataW4.y))/(1-
length(parsW4)/length(val_dataW4.y)))
```

```
% calculating the length of a part of an aorta that has the identified % inertia ((((((control so they are correct))))))

aorta_diameter_guessed= 0.023; %guessed aorta diameter

aorta_area_guessed= pi*(aorta_diameter_guessed/2)^2;      % guessed aorta area in meters

Inertia=L1_W4*133*10^6;

aorta_length=Inertia*aorta_area_guessed/1000          % in meters

%-----------------------------------------------------------------------

% IDENTIFYING MODEL W4_from_heart, called root of the aorta model in teh % thesis text

% Models for left ventricle pressure to aorta flow.

%-----------------------------------------------------------------------

% Using parameters from model W4

% input signals

P_end_estFromVentricle=zeros(length(Yest),1)+Pcap;

P_end_valFromVentricle=zeros(length(Yval),1)+Pcap;

UestW4FromVentricle=[driving_pressure(1:last_est_data)        P_end_estFromVentricle];
UvalW4FromVentricle=[driving_pressure(last_est_data+1:length(P_A))

P_end_valFromVentricle];

% make iddata vectors

est_dataW4FromVentricle=iddata(Yest,UestW4FromVentricle,Ts);

val_dataW4FromVentricle=iddata(Yval,UvalW4FromVentricle,Ts);
```

```
parsW4FromVentricle= [R1_W4/L1_W4 1/L1_W4]; % start estimates, using values

from model W4

auxW4FromVentricle=[Q_A(1) P1_W4 1/C1_W4 1/(R2_W4*C1_W4)]; % constant

parameters

modelW4FromVentricle=idgrey('body_idgrey_W4_from_heart',parsW4FromVentricle,'c',auxW4FromVentricle); % set up model

IdentModelW4FromVentricle=pem(est_dataW4FromVentricle,modelW4FromVentricle);

% naming the identified values

R_HV_o_W4=(IdentModelW4FromVentricle.a(1,1)/IdentModelW4FromVentricle.a(1,2))-R1_W4;

L_HV_W4=(-1/IdentModelW4FromVentricle.a(1,2))-L1_W4;

% validation W4 from heart

[YHW4FromVentricle,FITW4FromVentricle] =

compare(val_dataW4FromVentricle,IdentModelW4FromVentricle); FITW4FromVentricle

corrcoefW4FromVentricle=corrcoef(val_dataW4FromVentricle.y,YHW4FromVentricle{

1}.y)

loss_funcW4FromVentricle=sum((val_dataW4FromVentricle.y-

YHW4FromVentricle{1}.y).^2)/length(val_dataW4FromVentricle.y)

FPEW4FromVentricle=loss_funcW4FromVentricle*((1+3/length(val_dataW4FromVentricle.y))/(1-3/length(val_dataW4FromVentricle.y)))
```

```
% calculating the length of a part of an aorta that has the identified % inertia ((((((control so they are correct))))))

aorta_diameter_guessed= 0.023; %guessed aorta diameter

aorta_area_guessed= pi*(aorta_diameter_guessed/2)^2;      % guessed aorta area in meters

root_aorta_Inertia=L_HV_W4*133*10^6;

root_aorta_length=root_aorta_Inertia*aorta_area_guessed/1000      % in meters

%-----------------------------------------------------------------------

% IDENTIFYING MODEL valveW4, called aortic valve model in the thesis text. % Models for left ventricle pressure to aorta flow when the valve is closed.

%-----------------------------------------------------------------------

% Using parameters from model W4

Uest_valveW=[P_LV(1:last_est_data) P_end_estFromVentricle];

Uval_valveW=[P_LV(last_est_data+1:length(P_LV))          P_end_valFromVentricle]; est_data_valveW_pre=iddata(Yest,Uest_valveW,Ts);

val_data_valveW_pre=iddata(Yval,Uval_valveW,Ts);


est_data_valveW=merge(est_data_valveW_pre(171:331),est_data_valveW_pre(435:595),

est_data_valveW_pre(699:859),est_data_valveW_pre(963:1123),est_data_valveW_pre(1

228:1388),est_data_valveW_pre(1491:1651),est_data_valveW_pre(1755:1915),est_data_

valveW_pre(2018:2178),est_data_valveW_pre(2283:2443),est_data_valveW_pre(2547:2

707));
```

```
pars_valveW4= [(R_HV_o_W4+R1_W4)/L_HV_W4          10/C1_W4      45];

% parameters are total aorta resistance divided by aorta inertia now % includes the valve resistance, vavle compliance.

average_start_flow=(Q_A(171)+Q_A(435)+Q_A(699)+Q_A(963)+Q_A(1228)+Q_A(1491)+Q_A(1755)+Q_A(2018)+Q_A(2283)+Q_A(2547))/10;

aux_valveW4=[average_start_flow          0          1/(L_HV_W4+L1_W4)          1/C1_W4          1/(C1_W4*R2_W4)   45]; % constant parameters

model_valveW4=idgrey('body_idgrey_valveW4',pars_valveW4,'c',aux_valveW4);  %  set up model;

IdentModelValveW4=pem(est_data_valveW,model_valveW4); % minimize prediction

error

% naming the identified values

R_HV_c=(-IdentModelValveW4.a(1,1)*(L_HV_W4+L1_W4))-

(R_HV_o_W4+R1_W4);

C_HV_c=-1/IdentModelValveW4.a(3,1);



% validation   valve, model W4

% signals for validation

val_data_valveW1=val_data_valveW_pre(173:331);

val_data_valveW2=val_data_valveW_pre(436:597);

val_data_valveW3=val_data_valveW_pre(700:860);

val_data_valveW4=val_data_valveW_pre(964:1124);
```

```
val_data_valveW5=val_data_valveW_pre(1227:1388);

val_data_valveW6=val_data_valveW_pre(1492:1651);

val_data_valveW7=val_data_valveW_pre(1757:1915);

val_data_valveW8=val_data_valveW_pre(2020:2180);

val_data_valveW9=val_data_valveW_pre(2284:2442);

val_data_valveW10=val_data_valveW_pre(2547:2707);

[YH_valveW1,FIT_valveW1] = compare(val_data_valveW1,IdentModelValveW4);

[YH_valveW2,FIT_valveW2] = compare(val_data_valveW2,IdentModelValveW4);

[YH_valveW3,FIT_valveW3] = compare(val_data_valveW3,IdentModelValveW4);

[YH_valveW4,FIT_valveW4] = compare(val_data_valveW4,IdentModelValveW4);

[YH_valveW5,FIT_valveW5] = compare(val_data_valveW5,IdentModelValveW4);

[YH_valveW6,FIT_valveW6] = compare(val_data_valveW6,IdentModelValveW4);

[YH_valveW7,FIT_valveW7] = compare(val_data_valveW7,IdentModelValveW4);

[YH_valveW8,FIT_valveW8] = compare(val_data_valveW8,IdentModelValveW4);

[YH_valveW9,FIT_valveW9] = compare(val_data_valveW9,IdentModelValveW4);

[YH_valveW10,FIT_valveW10] = compare(val_data_valveW10,IdentModelValveW4);

% loss function

e1=sum(((val_data_valveW1.y)-(YH_valveW1{1}.y)).^2);
l1=length(val_data_valveW1.y);

e2=sum(((val_data_valveW2.y)-(YH_valveW2{1}.y)).^2);
l2=length(val_data_valveW2.y);
```

```
e3=sum(((val_data_valveW3.y)-(YH_valveW3{1}.y)).^2);
l3=length(val_data_valveW3.y);

e4=sum(((val_data_valveW4.y)-(YH_valveW4{1}.y)).^2);
l4=length(val_data_valveW4.y);

e5=sum(((val_data_valveW5.y)-(YH_valveW5{1}.y)).^2);
l5=length(val_data_valveW5.y);

e6=sum(((val_data_valveW6.y)-(YH_valveW6{1}.y)).^2);

l6=length(val_data_valveW6.y);

e7=sum(((val_data_valveW7.y)-(YH_valveW7{1}.y)).^2);
l7=length(val_data_valveW7.y);

e8=sum(((val_data_valveW8.y)-(YH_valveW8{1}.y)).^2);
l8=length(val_data_valveW8.y);

e9=sum(((val_data_valveW9.y)-(YH_valveW9{1}.y)).^2);
l9=length(val_data_valveW9.y);

e10=sum(((val_data_valveW10.y)-(YH_valveW10{1}.y)).^2);
l10=length(val_data_valveW10.y);

loss_func_valveW=(e1+e2+e3+e4+e5+e6+e7+e8+e9+e10)/(l1+l2+l3+l4+l5+l6+l7+l8+l9
+l10)

loss_func_stiff_valveW4=sum(((val_data_valveW1.y).^2)/length(val_data_valveW1.y))
```

```
% approx calculation of the movement of a valve.
int_valve=min(cumsum(YH_valveW1{1}.y))/400;                % in ml
int_valve_m3=int_valve*10^-6;   % in m^3
valve_stretch=int_valve_m3/aorta_area_guessed;
% Average valve stretch in the validation data.
int1=min(cumsum(val_data_valveW1.y))/400;   % in ml
int2=min(cumsum(val_data_valveW2.y))/400;
int3=min(cumsum(val_data_valveW3.y))/400;
int4=min(cumsum(val_data_valveW4.y))/400;
int5=min(cumsum(val_data_valveW5.y))/400;
int6=min(cumsum(val_data_valveW6.y))/400;
int7=min(cumsum(val_data_valveW7.y))/400;
int8=min(cumsum(val_data_valveW8.y))/400;
int9=min(cumsum(val_data_valveW9.y))/400;
int10=min(cumsum(val_data_valveW10.y))/400;
int_average=(int1+int2+int3+int4+int5+int6+int7+int8+int9+int10)/10;
int_valve_m3_val=int_average*10^-6;   % in m^3
valve_stretch_val=int_valve_m3_val/aorta_area_guessed;


%-----------------------------------------------
% displaying the identified values;
%-----------------------------------------------
```

Pcap

Q_A_W4

P1_W4

R1_W4

L1_W4

C1_W4

R2_W4

R_HV_o_W4

L_HV_W4

R_HV_c

C_HV_c

7.1.2 Code for body_idgrey_1.m

```
function [A,B,C,D,K,x0] = body_idgrey_1(pars,Ts,aux)
% parameters to be identified
R1_d_I1=pars(1);
one_d_I1=pars(2);
one_d_C1=pars(3);
% state space matrixes
A=[-R1_d_I1 -one_d_I1;
one_d_C1        0          ];
```

```
B=[one_d_I1 0;

0 -one_d_C1];

C = [1 0];

D = [0 0];

K = [0; 0];

% state initial values

x0 = [aux(1) ; pars(4)]; % first is constant and the second is identified
```

7.1.3 Code for body_idgrey_2.m

```
function [A,B,C,D,K,x0] = body_idgrey_2(pars,Ts,aux)

% parameters to be identified

one_d_C1=pars(1);

one_d_R1=pars(2);

% state space matrixes

A=[-one_d_R1*one_d_C1];

B=[one_d_R1*one_d_C1 -one_d_C1];

C = [-one_d_R1];

D = [one_d_R1 0];

K = [0];

% state initial values

x0 = [pars(3)]; % to be identified
```

7.1.4 Code for body_idgrey_W4.m

```
function [A,B,C,D,K,x0] = body_idgrey_W4(pars,T,aux)

% parameters to be identified

R1_d_I1=pars(1);

one_d_I1=pars(2);

one_d_C1=pars(3);

one_d_C1R2=pars(4);

Pcap_d_C1R2=0; % for model with an end pressure that equals the flow times a constant.
% Pcap_d_C1R2=pars(6); % for model with a constant end pressure

% state space matrixes

A=[-R1_d_I1 -one_d_I1;

one_d_C1       -one_d_C1R2];

B=[one_d_I1 0;

0 Pcap_d_C1R2];

C = [1 0];

D = [0 0];

K = [0; 0];

% state initial values

x0 = [aux(1); pars(5)]; % first is constant and the second is identified
```

7.1.5 Code for body_idgrey_W4_from_heart.m

```
function [A,B,C,D,K,x0] = body_idgrey_W4_from_heart(pars,T,aux)
```

```
% parameters to be identified

R1_d_I1=pars(1);

one_d_I1=pars(2);

% constant parameters

one_d_C1=aux(3);

one_d_C1R2=aux(4);

% state space matrixes

A=[-R1_d_I1 -one_d_I1;

one_d_C1        -one_d_C1R2];

B=[one_d_I1 0;

0 one_d_C1R2];

C = [1 0];

D = [0 0];

K = [0; 0];

% state initial values

x0 = [aux(1); aux(2)]; % first is constant and the second is identified
```

7.1.6 Code for body_idgrey_valveW4.m

```
function [A,B,C,D,K,x0] = body_idgrey_valveW4(pars,T,aux)

% parameters to be identified
```

```
R1_d_I1=pars(1);

one_d_Cv=pars(2);

% constant parameters

one_d_I1=aux(3);

one_d_C1=aux(4);

one_d_C1R2=aux(5);

% state space matrixes

A=[-R1_d_I1 -one_d_I1      one_d_I1;

one_d_C1 -one_d_C1R2        0;

-one_d_Cv      0          0];

B=[one_d_I1 0;

0 one_d_C1R2;

0          0];

C = [1 0 0];

D = [0 0];

K = [0; 0; 0];

% state initial values

x0 = [aux(1) ; aux(6); aux(2)]; % constant
```

7.2 Matlab and Simulink code for simulations

The SimulinkR model needs parameter values to run and these are created by running the identification code as described previously. The values then need to be copied to the file 'Final_model_CODE.m' and the file executed. The MatlabR workspace now contains the parameter values and the SimulinkR model can be executed in any way the user seems fit.

The model can be run with or without the AVD "implanted" by clicking on the manual switch 'no added valve/added valve' inside the 'Body and added valve' block.

The model can be run by using a measured compliance profile of the left ventricle over several beats or by using the compliance measurement of one beat repeated. The several beats option is more accurate since it is true measurements but it only works for a limited number of beats. If a longer simulation is wanted the repeating option has to be used. The change is done by clicking the manual switches 'same beat loop/several true beats' inside the 'Body and added valve' block.

7.2.1 Code for Final_model_CODE.m

```
%-----------------------------------------------------------------

% Getting compliance and left ventricular inflow from data. %-------------------------------
--------------------------------

load Patient_003_001.mat   % load the data

start_heart_volume=120; % volume in heart before ejection. educated guess.

% making simulink data, one beat repeated.

%-----------------------------------------

% 22 full beats on 5810-6 samples gives 263.82 samples per beat % chosing to start beat
at sample 166.

start_data=166;
```

```
end_data=5756;

Q_LV_out_one_beat=(AoF(166:(166+264-1)))*1000/60;

Q_LV_out_shift_one_beat=[Q_LV_out_one_beat(65:264);Q_LV_out_one_beat(1:64)];

Q_LV_in_one_beat=[Q_LV_out_shift_one_beat(113:264);

Q_LV_out_shift_one_beat(1:112)];

Q_LV_in_shift_one_beat=Q_LV_in_one_beat;   % use in simulink

in_out_flow_one_beat=(Q_LV_in_shift_one_beat-Q_LV_out_shift_one_beat);

volume_data_one_beat=start_heart_volume+cumsum(in_out_flow_one_beat/400);

P_LV_beat_one_beat=LVP(166:(166+264-1));

P_LV_shift_one_beat=[P_LV_beat_one_beat(65:264);P_LV_beat_one_beat(1:64)];

LV_compliance_one_beat=volume_data_one_beat./P_LV_shift_one_beat;      % use in simulink time_one_beat=[1:length(Q_LV_in_shift_one_beat)]'/400; % used together with the % compliance and inflow in simulink

% making simulink data, several beats for a true sequence of data %----------------------------------------last_est_data=floor(263.82*11)+166;   % using that 22 full beats on 5804 samples % gives 263.82 samples per beat, 11 full beats is used for the estimation data. Q_out_seq_val=AoF((last_est_data+50):(end_data+50))*1000/60;;

Q_fill_seq_val=AoF((last_est_data+50-132):(end_data+50-132))*1000/60;

in_out_flow_seq_val=Q_fill_seq_val-Q_out_seq_val;

volume_seq_val=start_heart_volume+cumsum(in_out_flow_seq_val/400);
P_seq_val=LVP((last_est_data+50):end_data+50);

LV_compliance_seq_val=volume_seq_val./P_seq_val;

one_div_LV_compliance_data_val=([1:length(LV_compliance_seq_val)]'/400)
```

```
1./LV_compliance_seq_val]; % use in simulink

Q_fill_data_val=[([1:length(Q_fill_seq_val)]'/400) Q_fill_seq_val]; % use in simulink

% simulink comparison data.

P_LV_data_val=[([1:length(P_seq_val)]'/400)  P_seq_val];

P_A_val=AoP((last_est_data+50):end_data+50);

P_A_data_val=[([1:length(P_A_val)]'/400)   P_A_val];

Q_A_data_val=[([1:length(Q_out_seq_val)]'/400)                    Q_out_seq_val];
Q_average_tot=sum(Q_A_data_val)/length(Q_A_data_val);

%-----------------------------------------------------------------

% Get values from identification experiments.

%-----------------------------------------------------------------

P_cap=0;   % Pcap

% initial state values.

initial_flow1=2; %  Q_A_W4

initial_pressure1=43.5526;     % P1_W4

% Resistances,

R_1=0.0679;   % R1_W4

R_2=0.4888;   % R2_W4

R_HV_o=0.0136;       %  R_HV_o_W4

R_HV_c=0.5153;   % R_HV_c

R_AV_o=R_HV_o/2;   % extra friction from the implanted valve. Assumed to be same % half of the resistance of the aortic valve plus the root aorta
```

```
% resistance.
% estimated from heart valve identification and artificial valve data.
R_AV_c=R_HV_c;   %estimated from heart valve identification and artificial valve data.
% Compliances, the driving left ventrical compliance is given above.
C_HV_c=0.0611;       % C_HV_c
C_AV_c=C_HV_c;    %
C_1=11.5393; % C1_W4
% inertias,
I_1=0.0012;    % I1_W4
I_HV=0.00039073;    % I_HV_W4
% Start stored flows. volumes in ml
v_LV=start_heart_volume;
v_1=initial_pressure1*C_1;
%start stored efforts.
e_1=initial_flow1*I_1;
%-------------------------------------------------------------
% AVD characteristics
%-------------------------------------------------------------
v_Pump=0;   % initail value
max_pump_volume=150;   %might not use
% GY_const = 43.6*60/(1000); % Voltage constant V/f
```

```
GY_const = 43.6*60/1000/(2*pi); % Voltage constant V/w

% Gearing=0.2;   % gears are added 0.2

Gearing=1;

arm_length=0.02; % m

% TF_const = 1/(arm_length*2*pi*Gearing);        % if f is used TF_const = 1/(arm_length*Gearing);   % if w is used

Pump_diameter = 0.096; %in meters

Pump_area = pi*(Pump_diameter/2)^2;

tube_diameter = 0.015;          % m, approximate guess.

tube_area = pi*(tube_diameter/2)^2; % 2 tubes in the origional heartmate. tube_length = 0.15; % m, educated guess

% Windings data

motor_inductance = 9*10^-3;   %Henry

motor_resistance = 2.4; %Ohm

% Motor data

motor_inertial_mass = 3.6*10^-5;   % in kgm^2

motor_friction = 0.034*60/1000/(2*pi);    %using 0.03 Nm/krpm, taken from a different but % similar motor.

motor_static_friction= 0.04;   % Friction torque  Nm

motor_dynamic_friction_dcpart=0.04;          % assumed value, same as the static friction

% to avoid discontinuities

% Piston data
```

piston_mass = 0.100; % 100 gram

piston_mass_transformed = piston_mass/(TF_const^2);

piston_friction = 1; % guessed value, gives about the same friction as the

% motor which should be about right

piston_friction_transformed = piston_friction/(TF_const^2);

piston_static_friction =1; % gives about half the static friction as the motor, % just a guess to make them in the same size range. piston_static_friction_transformed = piston_static_friction/(TF_const^2); % transformed so it can be added to the motor dynamics

% Tube data blood_mass= tube_area*tube_length*1000; % mass of the blood in the tube.

tube_inertia= blood_mass/(tube_area^2); % larger then the total inertia of the arteries, % which at first sounds very wrong, but when considering that the aorta

% branches out very early and that therefore the area becomes larger and

% larger it might just be correct.

body_inertia=I_1*10^6*133; % for comparison

tube_inertia_transformed=tube_inertia*(Pump_area^2)/(TF_const^2);

tube_friction = 1*10^-2*10^6*133; % 133*10^6 is to transform it into SI units.

% 10^-3 is approx the same resistance as between the heart and the tube, % but that includes a valve which brings the resistance up. So 10 times % that value for the blood friction in the tube should be ok.

tube_friction_transformed = tube_friction*Pump_area^2/(TF_const^2); %the blood is assumed not to have a static friction. % transformed so it can be added to the motor dynamics

% Inertia and friction for the tube, piston and motor together.

```
total_inertial_mass = motor_inertial_mass + piston_mass_transformed + tube_inertia_transformed; total_motor_friction = motor_friction + piston_friction_transformed + tube_friction_transformed ; static_friction = motor_static_friction + piston_static_friction_transformed; dynamic_friction_dcpart = motor_dynamic_friction_dcpart +

piston_static_friction_transformed;

%-----------------------------------------------------------

% Noise characteristics and sample rate for the A/D converter, % not used in the thesis

%-----------------------------------------------------------

% Noise variance, guessed

Flow_noise_LperMin=5;

Preassure_noise_mmHg=2;

Motor_position_noise=0.0001; %in m

volume_noise_m3=Motor_position_noise*Pump_area;


%constants to be able to change the noise easy. F_noise_factor=4; %4

P_noise_factor=5; %5

V_noise_factor=5; %5

% make flow noise for ml per second.

Flow_noise=Flow_noise_LperMin*1000/60*F_noise_factor;

Preassure_noise=Preassure_noise_mmHg*P_noise_factor;

Volume_noise = volume_noise_m3*V_noise_factor*10^6;
```

```
% sample time for measurements
sample_time=1/400;


%--------------------------------------------------------------------------
% Controller
%--------------------------------------------------------------------------
% wanted parameters, used to calculate the reference for Q_heart, R_1_w=0.8 * (R_HV_o+R_1+R_AV_o);
C_1_w=1 * C_1;
I_1_w=1 * (I_HV+I_1);
R_2_w=1 * R_2;
Q_average_tot_w=Q_average_tot;   % do not change. init_pressure_w=40;
P_cap_w=P_cap;
P_arteries_w=40;
K_P=0.1;   % no gears
% K_P=0.3; % unstabile
K_I=1;
K_P_E=0.01;
K_I_E=0.01;
% K_P=0.03;   %gears=0.2
% K_I=0.2;
```

```
% K_P_E=0.02;

% K_I_E=0.02;

%-----------------------

% Presampling filters

[B_bessel_8_60,A_bessel_8_60]=besself(8,60*2*pi);

[B_bessel_3_60,A_bessel_3_60]=besself(3,60*2*pi);

[B_bessel_1_60,A_bessel_1_60]=besself(1,60*2*pi); % the one used.
[B_bessel_2_60,A_bessel_2_60]=besself(2,60*2*pi);
```

7.2.2 Simulink schematics for Final_model.mdl

Figures 48 through 64 shows the SimulinkR schematics for the model used to simulate the AVD and arterial system.

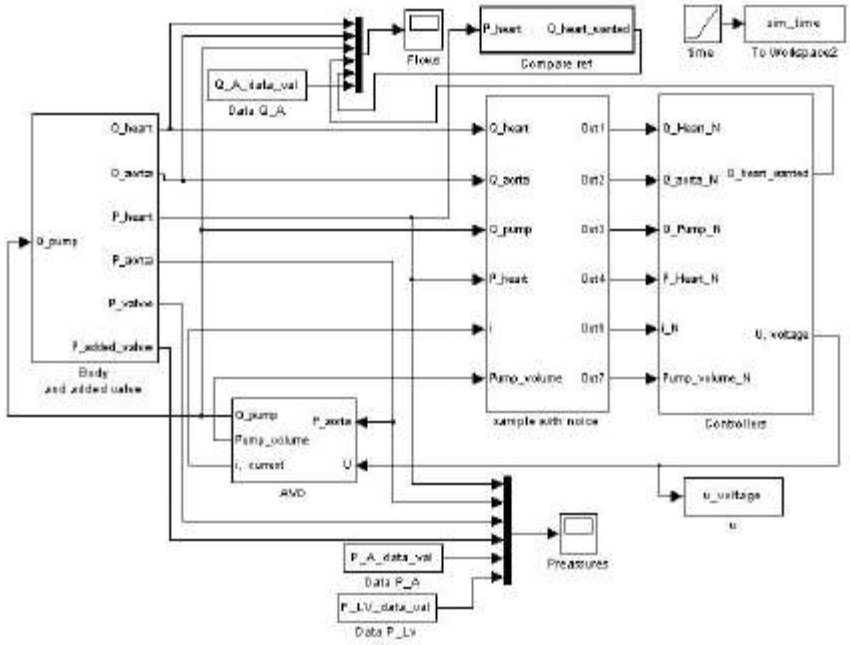


Figure 48: Simulink schematic of top of final_model.mdl.

Figure 48: Simulink schematic of top of final_model.mdl.

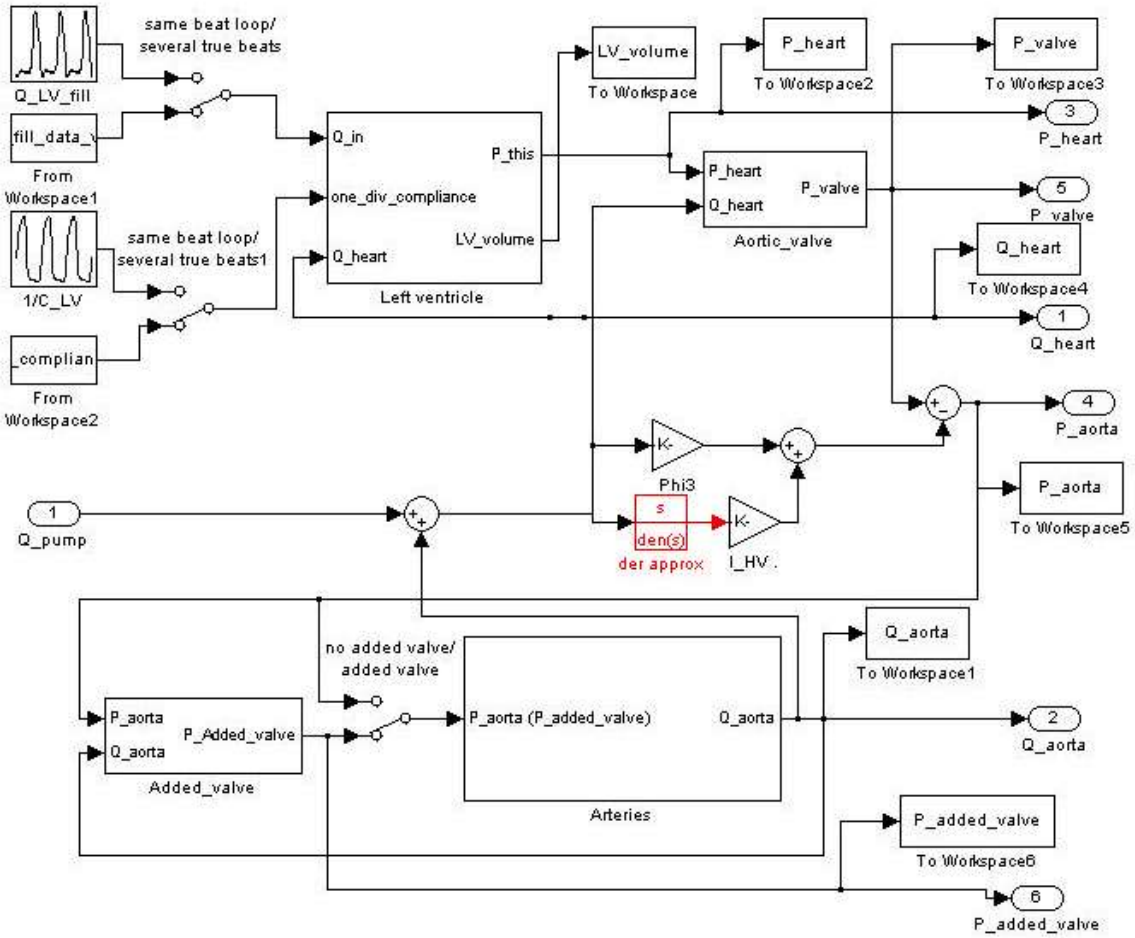


Figure 49: Simulink schematic of 'Body and added valve' in final_model.mdl.

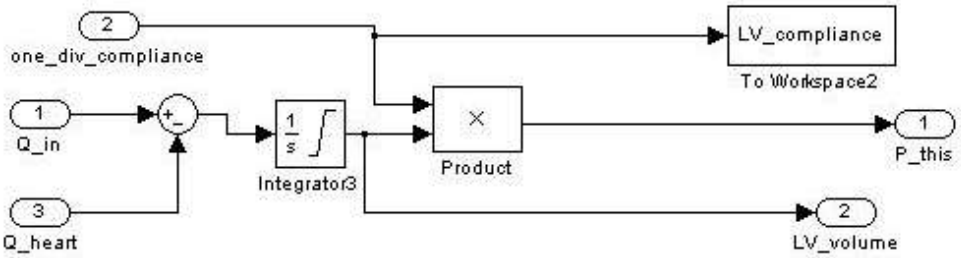


Figure 50: Simulink schematic of 'Left ventricle' in final_model.mdl.

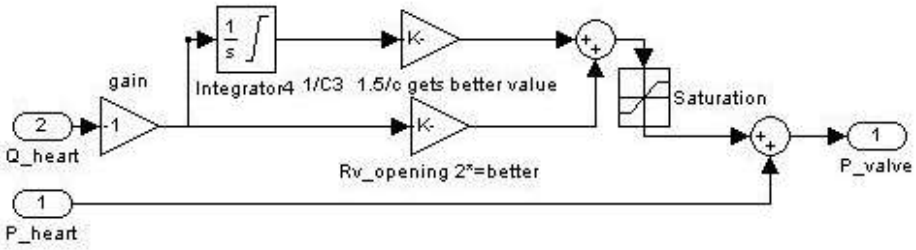


Figure 51: Simulink schematic of 'Aortic valve' in final_model.mdl.

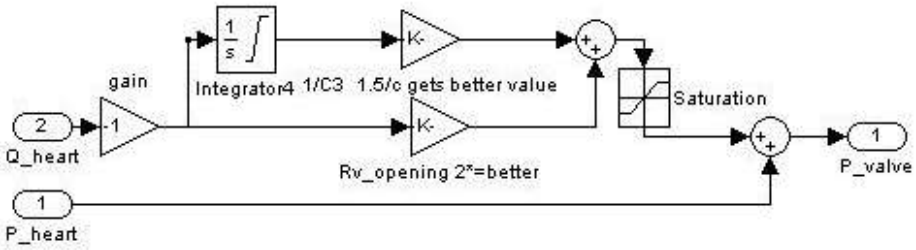


Figure 52: Simulink schematic of 'Added valve' in final_model.mdl.

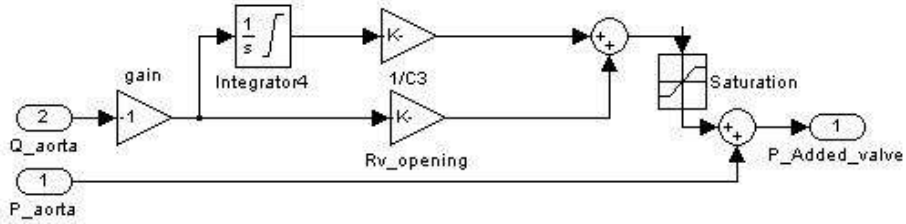


Figure 53: Simulink schematic of 'Arteries' in final_model.mdl.

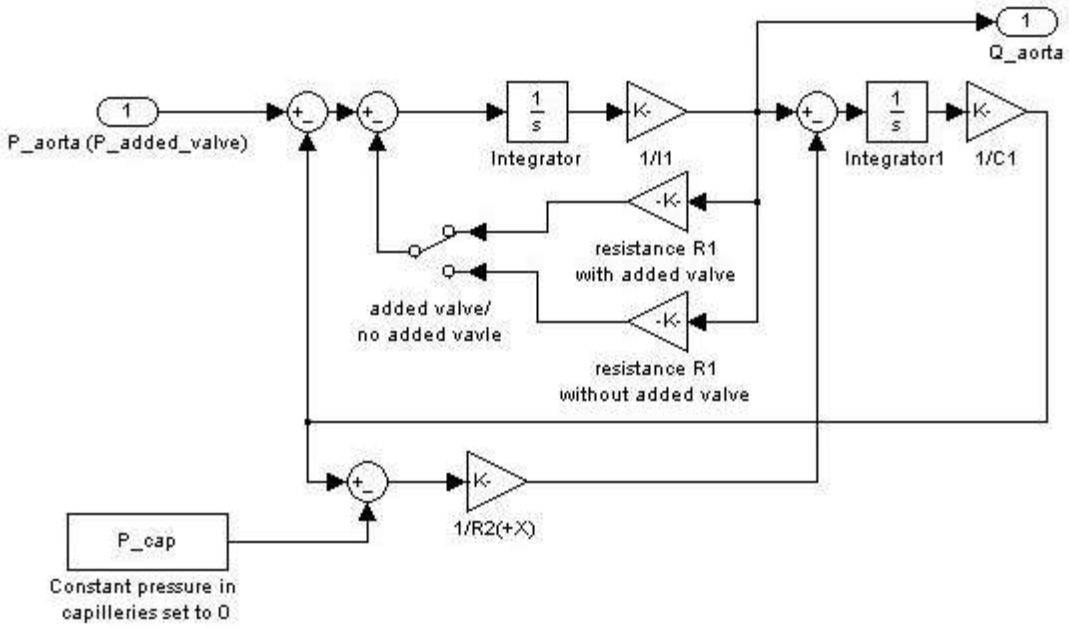

1
Q_aorta
1
P_aorta (P_added_valve)
Integrator
1/I1
Integrator1
1/C1
-K-
resistance R1
with added valve
added valve/
no added vavle
-K-
resistance R1
without added valve
1/R2(+X)
P_cap
Constant pressure in
capilleries set to 0

Figure 54: Si

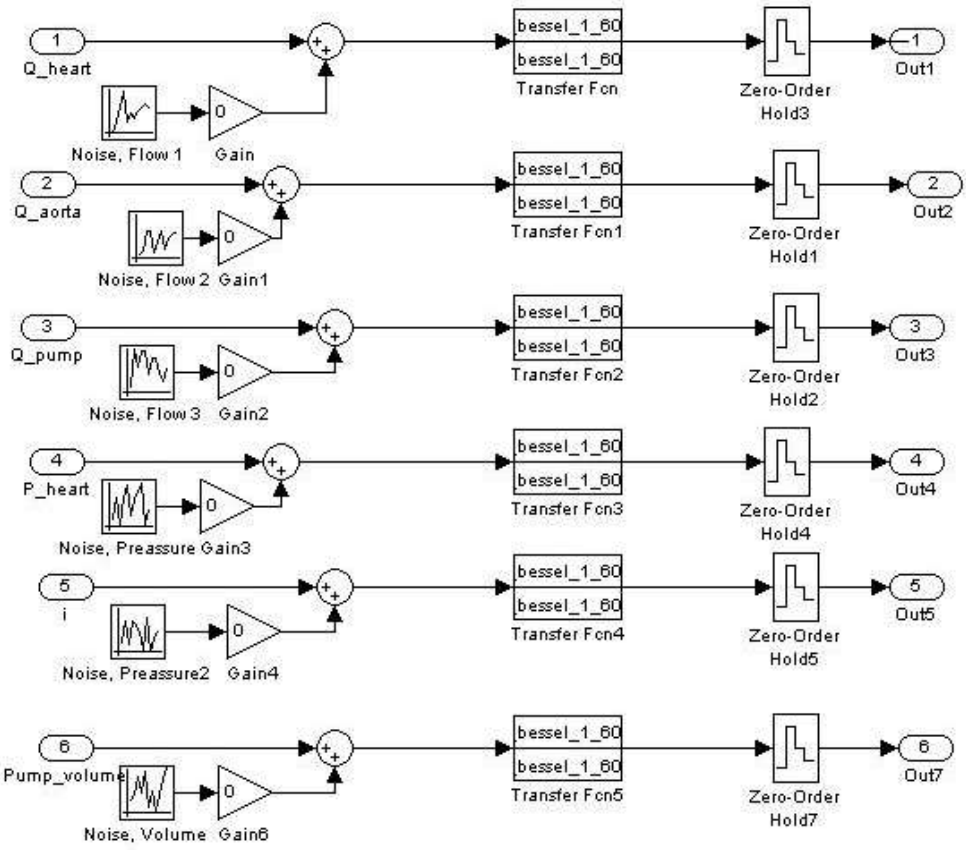


Figure 55: Simulink schematic of ‘Controllers’ in final_model.mdl.

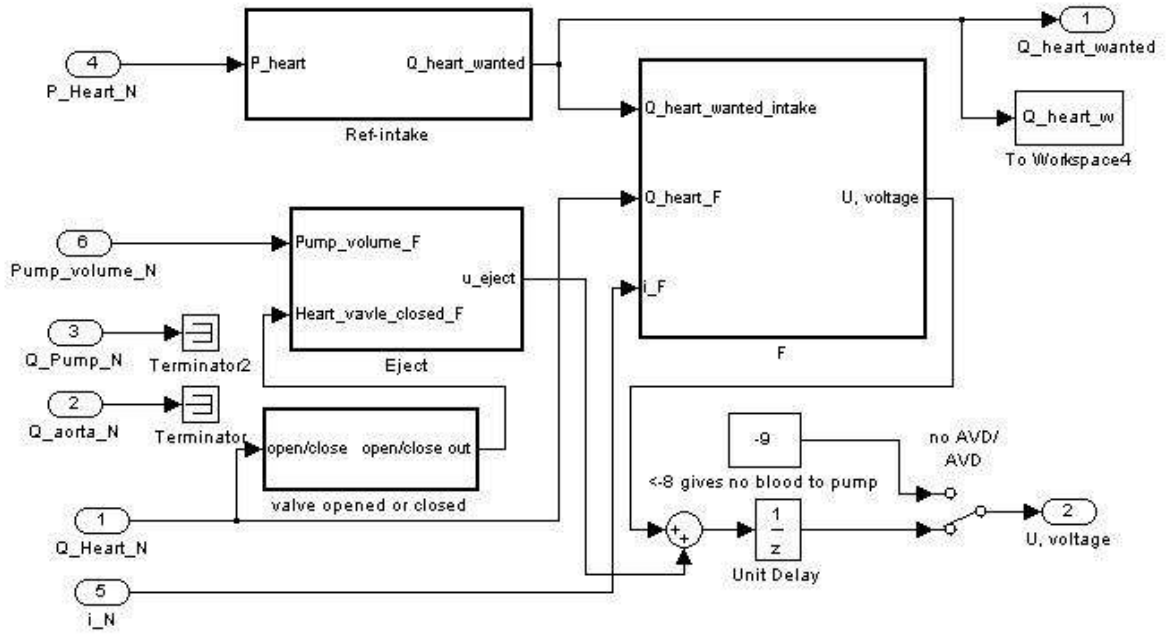


Figure 56: Simulink schematic of 'Ref-intake' in final_model.mdl.

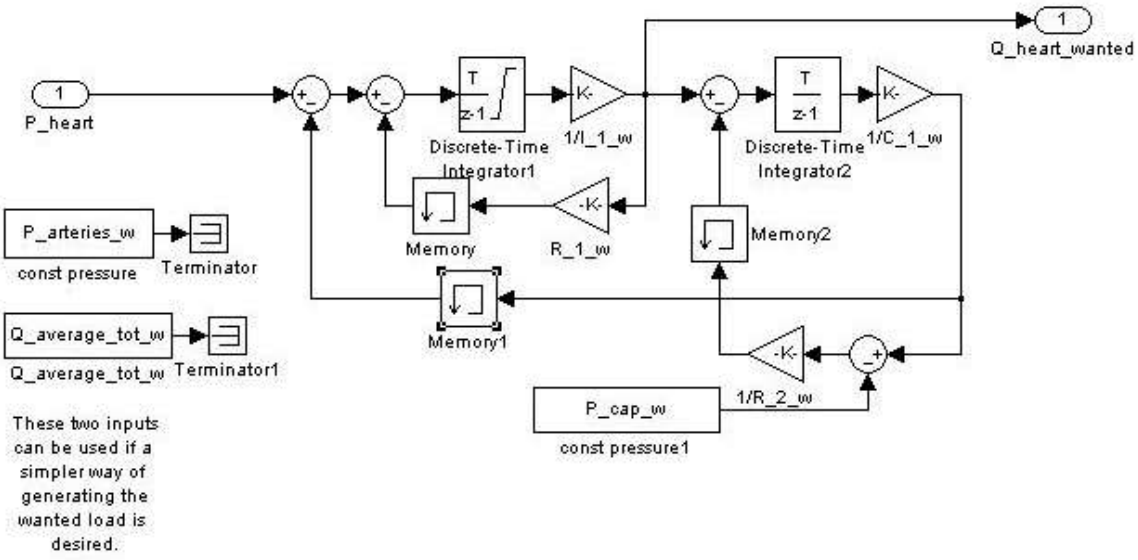


Figure 57: Simulink schematic of 'Eject' in final_model.mdl.

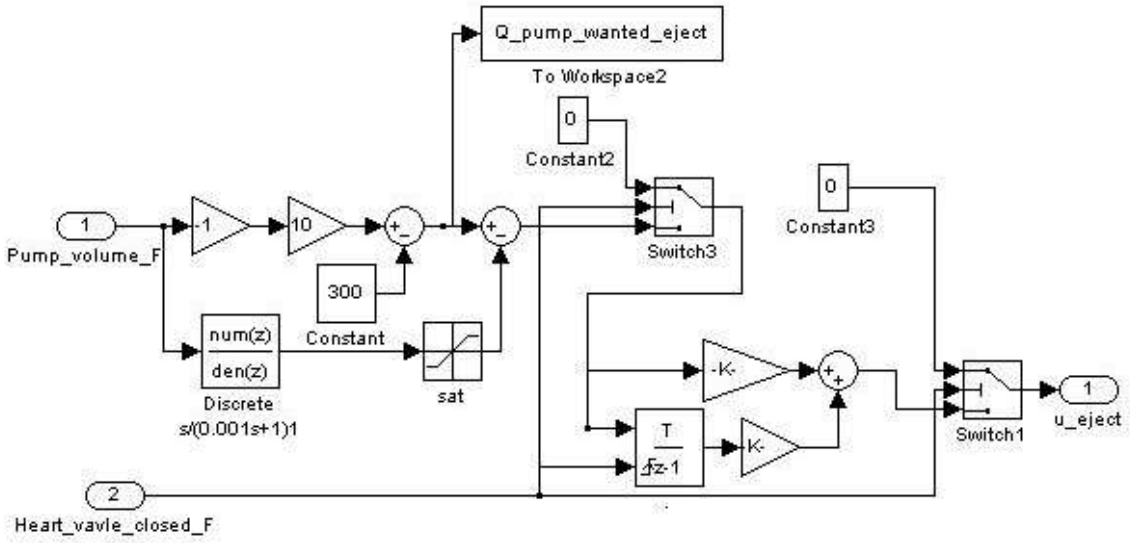


Figure 58: Simulink schematic of 'Valve opened or closed' in final_model.mdl.

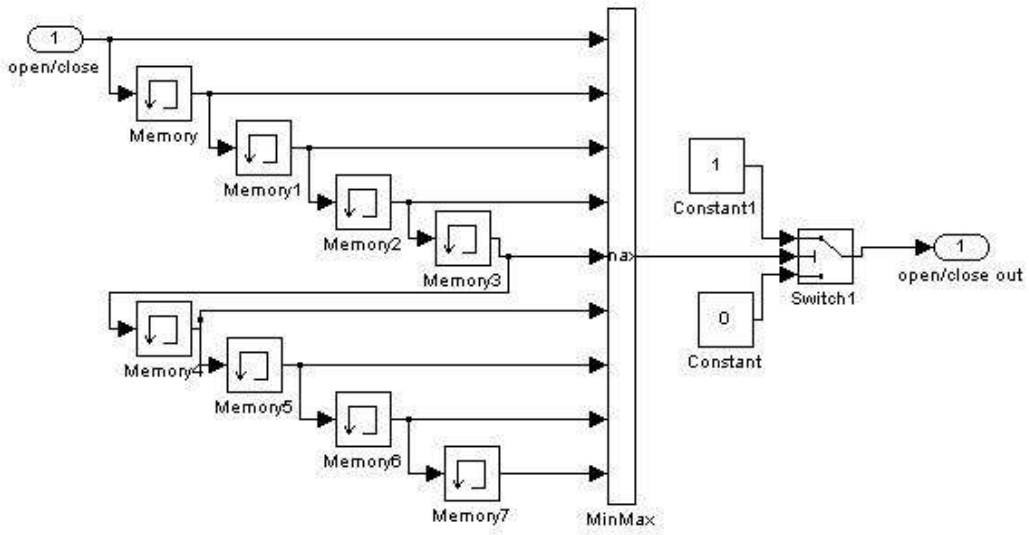


Figure 59: Simulink schematic of 'F' in final_model.mdl.

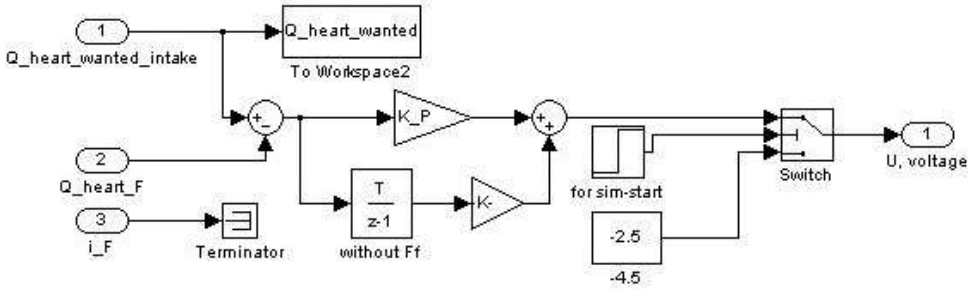


Figure 60: Simulink schematic of 'Compare ref' in final_model.mdl.

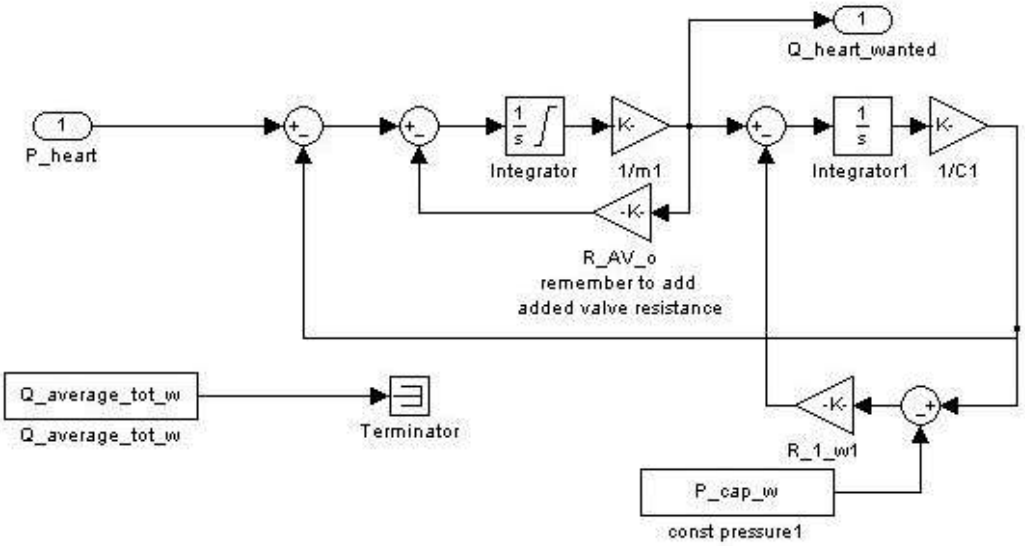


Figure 61: Simulink schematic of 'AVD' in final_model.mdl.

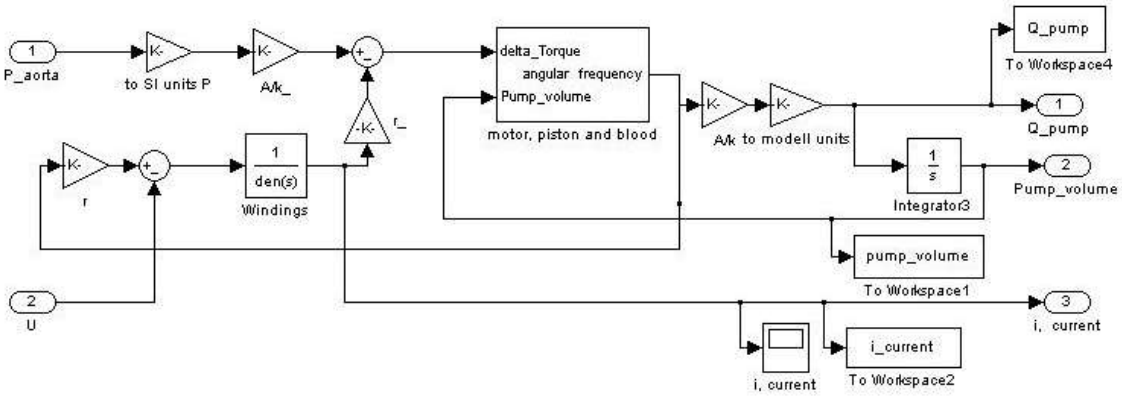


Figure 62: Simulink schematic of ‘Motor, piston and blood’ in final_model.mdl.

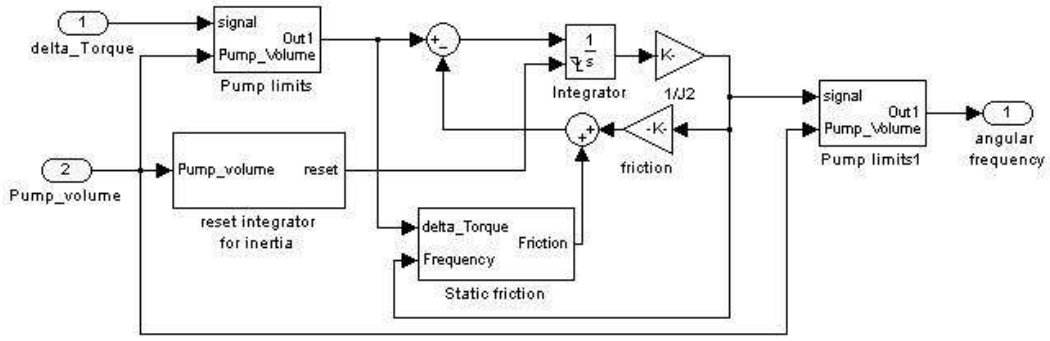


Figure 63: Simulink schematic of ‘Pump limits’ and ‘Pump limits1’ in final_model.mdl.

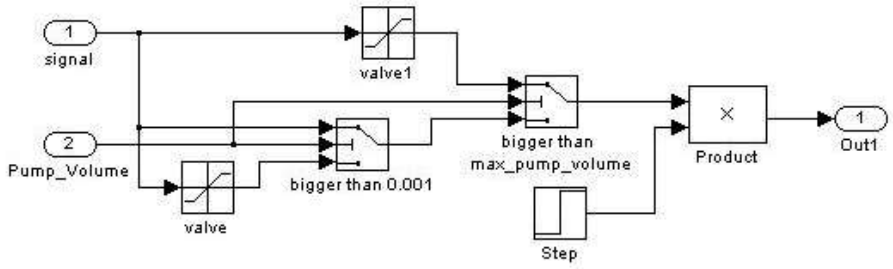


Figure 64: Simulink schematic of ‘Reset integrator for inertia’ in final_model.mdl.

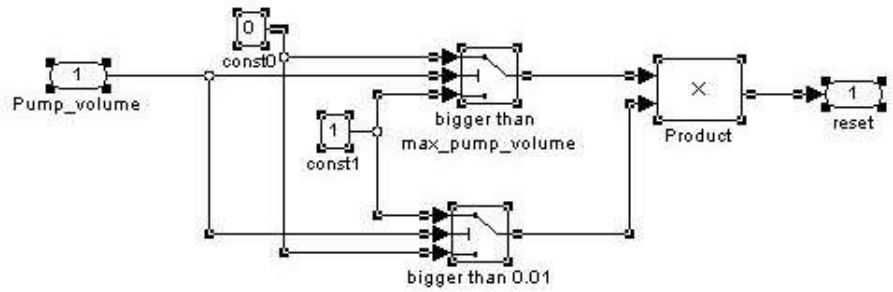


Figure 65: Simulink schematic of ‘Static friction’ in final_model.mdl.

Conclusion

This device is in the proper functioning of the heart muscle to work, because due to edit a decrease in blood pressure left ventricular with the aorta is a device that is connected between. It is heart enough pressure to create this device is not required, it creates pressure. Ideal pressure patients are connected to the permanent ECG device to the heart of the instantaneous value of the measuring device, the working speed of the sensor count. In a similar manner to that seen about this process are portable. Here, the cd sleeve, made by the reservoir of the heart left ventricular taken by the model. As the heart muscle work remains untreated. this section is whether this device makes the task of that u see. Here is pressed, the fresh blood to the body, stopping contaminated blood he heart is in the right ventricularina came from there to the lungs is gradually cleansed of the blood back to the left ventriculara returns to this circulation modelling here you can see. Here you are seeing is that the movement of the leg is the old model of the artificial leg's motor. With the Wheel that I replacement up to the this we are arranging the what amount of blood that pump in it. Eccentiric exel turns with making horizontal force by this way piston goes and comes back. The piston attached to the horizontal rod piston that moves the shaft.

Part of wood is a support which is attached to under the exel effects block of the spasm and bending.The case which is tied up to the machine agree with to make it stable besides that it helps to the air inside the machine to go out with this way. With this way I am controlling the blood flow with check walves. The pipe which comes on to the cd case this is the way how the clean blood comes in.The other pipe which comes from down it Works for the clean blood's entrance way.

While I am preparing this Project I had lots of problem about finding parts, technological disadvantages of our country, financial problems, not enough time, I was alone on this Project so that it was so hard to complete it in time with perfect materials. On this Project I tried to add more technological devices to improve this material. As you see on this prototype I wanted to add blood pump control which is Works for arranges the blood pump with digital screen. With

the alarm system which Works according to the blood pump increase and decrease after a while if the device can not work properly my system takes the control to balances the blood pump. And I wanted to add sms and gprs system on this device, by this way device can send sms or the doctor could reach the own patient when the patient has problem quickly. If the battery has problem or the charge value dicrease this control system that I designed can give alarm to the patient. So that patient can refill or replace the battery before it finish.

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483-586.

Nicholas A. Peppas - Chaired Professor in Engineering, University of Texas at Austin,

pioneer in drug delivery, biomaterials, hydrogels and nanobiotechnology.

Otto Schmitt (deceased) - biophysicist with significant contributions to BME, working with biomimetics

Ascher Shapiro (deceased) - Institute Professor at MIT, contributed to the development of the BME field, medical devices (e.g. intra-aortic balloons)

John G. Webster - Professor Emeritus at the University of Wisconsin–Madison, a pioneer in the field of instrumentation amplifiers for the recording of electrophysiological signals

Robert Plonsey - Professor Emeritus at Duke University, pioneer of electrophysiology.

U. A. Whitaker (deceased) - provider of The Whitaker Foundation, which supported research and education in BME by providing over $700 million to various universities, helping to create 30 BME programs and helping finance the construction of 13 buildings.

Frederick Thurstone (deceased) - Professor Emeritus at Duke University, pioneer of diagnostic ultrasound.

Kenneth R. Diller - Chaired and Endowed Professor in Engineering, University of Texas at Austin. Founded the BME department at UT Austin. Pioneer in bioheat transfer, mass transfer, and biotransport

Alfred E. Mann - Physicist, entrepreneur and philanthropist. A pioneer in the field of Biomedical Engineering.

Forrest Bird - aviator and pioneer in the invention of mechanical ventilators

Willem Johan Kolff (deceased) - pioneer of hemodialysis as well as in the field of artificial organs

John James Rickard Macleod(deceased) - one of the co-discoverers of insulin at Case Western Reserve University.