

Model based heart sound analysis for the detection of diagnostically relevant symptoms

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Abstract

Heart auscultation is a fundamental tool in the diagnosis of heart disease. It is particularly important in primary health care, due to its effectiveness in detecting a wide range of heart abnormalities, and to the low cost of the equipment involved. However, forming a diagnosis based on heart sounds is a skill that can take years to acquire and refine. Particularly in remote areas and in developing countries, physicians with the necessary training may not be widely available. In this paper, we develop a simple model for the production of heart sounds, and demonstrate its utility in identifying features useful in diagnosis.

1. Introduction

Heart auscultation (the monitoring of sounds produced by the heart) is a fundamental tool in the diagnosis of heart disease. While somewhat eclipsed in the research literature due to the advent of electrocardiographic (ECG) methods, there are heart defects that are difficult to detect using ECG (e.g., structural abnormalities in natural or implanted heart valves, and defects characterized by heart murmurs). Furthermore, auscultation remains the primary tool for screening and diagnosis in primary health care, due in part to the higher cost and relatively limited availability of ECG equipment, and to the special skills necessary to administer and interpret the results of ECG. In some circumstances, particularly in remote areas or developing countries, auscultation may be the only means available.

However, forming a diagnosis based on sounds heard through either a conventional acoustic or an electronic stethoscope is itself a very special skill, one that can take years to acquire. Despite its obvious utility, because this skill is also very difficult to teach in a structured way, the majority of internal medicine and cardiology programs offer no such instruction. It would be very useful if the benefits of auscultation could be obtained with a reduced learning curve, using equipment that is low-cost, robust, and easy to use.

Recent technological developments have made very powerful digital signal processing (DSP) techniques both widely accessible and practical. These methods have been applied to study the fundamental mechanisms underlying the production of sound by the heart, and the correlation between these sounds and various heart defects (e.g., [1]- [10]). For a survey and discussion of work in this area see [11].

Despite the clear success of DSP based techniques, many heart sounds associated with defects remain subtle, and difficult to detect and discriminate from similar sounds with no underlying pathology.

Some of the same technological advances that have supported DSP oriented methods have also facilitated the development of increasingly sophisticated models of the heart, from relatively simple models focusing on sound formation such as [12], to full 3-D structural models.

The goal of this work is to combine DSP based methods of analysis with the additional information provided via modeling, to provide improved detection and interpretation of sounds corresponding to symptoms important for diagnosis. It is hoped that the results of this analysis may prove valuable in themselves as a diagnostic aid, and as input to more sophisticated machine diagnosis systems, for example [13].

2. The Structure of a Heart Sound

Heart sounds are complex and highly nonstationary signals. The “beats” associated with these sounds are reflected in the signal by periods of relatively high activity, alternating with comparatively long intervals of low activity.

Most commonly there are two major components to each heart sound cycle, referred to as S1 and S2. A normal heart sound signal is shown in Figure 1, with S1 and S2 extracted and shown on expanded time axes at the bottom of the figure. While the physiological origins of all the contributions to S1 and S2 are not agreed upon, it is clear that the closure of the mitral and tricuspid valves are major contributors to S1. Similarly, the closure of the aortic and pulmonary valves are primary contributors to S2 [14].

In addition to S1 and S2, which are always present, third and fourth heart sounds (S3 and S4) may also be heard. When present, S3 occurs shortly after S2, and is associated with early diastolic filling of the ventricle. When S4 is audible, it occurs shortly before S1, and is associated with late diastolic filling. An audible S3 is considered normal in the young (less than 35 years of age), while an audible S4 is always abnormal.

Finally, there is a large class of sounds, referred to as “murmurs,” that occur at different points in (and sometimes throughout) the overall heart cycle. It is generally agreed that these sounds are caused by turbulent blood flow. While usually a sign of abnormality, murmurs with no underlying pathology (“innocent murmurs”) may be detected in young children.

3. A Simple Heart Sound Model

The heart-thorax acoustic system, like the heart sounds themselves, is extremely complex. An approach to modeling complex systems that has proven useful in a number of domains is to approximate them as linear systems, and to use the tools developed for the study of such systems. Usually, this approach includes the assumption that the system of interest is time-invariant. Clearly, this is not the case here.

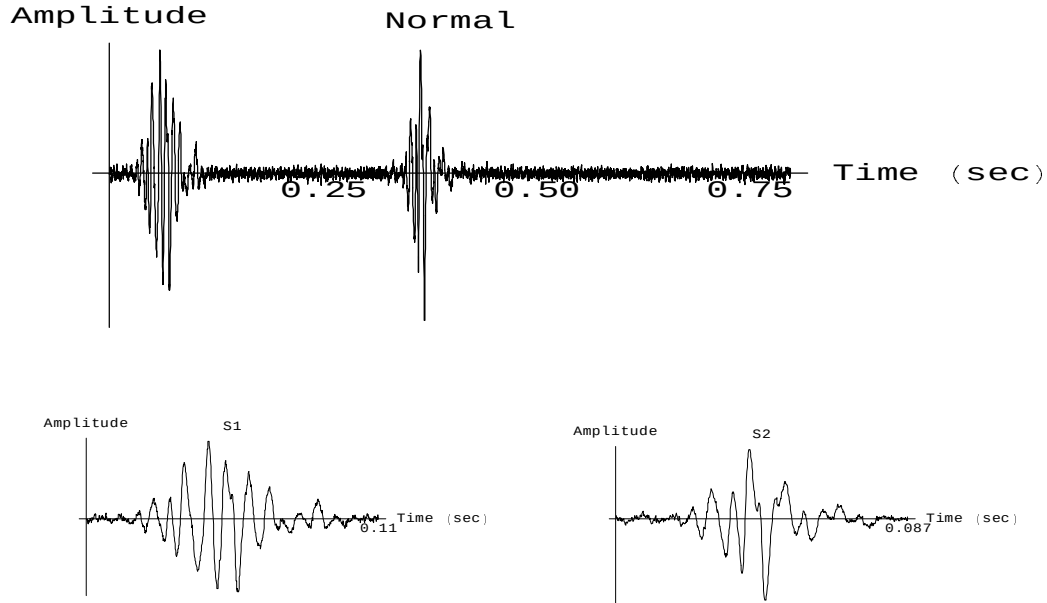


Figure 1. A normal heartbeat (top), with S1 and S2 (bottom).

Durand and Pibarot [11] have proposed a linear model with both time-varying and time-invariant components. The system includes (in series) a subsystem which varies quickly with time (to represent the myocardial system), one which varies slowly with time (to model the respiratory system), and one which is time invariant (to capture the behavior of the chest wall system). The model also includes impulse-like, stochastic, and periodic inputs to represent components of the heart sound due to events such as valve closure, stenotic and regurgitant murmurs, and musical murmurs, respectively.

The architecture of the model in [11] reflects the physiological structure of the heart-thorax system in a particularly elegant way. It provides an intuitive framework for understanding the dynamics of the system, and in situations where the implantation of intracardiac transducers is feasible, provides a very good approach to system characterization.

The goal of this work, however, is to capture a diagnostically useful system description non-invasively. While the general approach described above is still valuable for this task, some approximations are required due to the lack of internal data. First of all, we will assume that the system is time-invariant during each of the resolvable heart sounds. This restriction is due to the fact that the responses to the events associated with each sound (e.g., the responses to the mitral and tricuspid valves closing in S1) are generally not distinguishable at the chest wall. For the current discussion, we will also restrict our model to the S1 and S2 heart sounds, assume that S1 and S2 are due only to the respective valve closings, and consider only cases which do not involve murmurs (although these restrictions may be removed in future work).

The resulting simplified system is shown in Figure 2. The input to the system, the

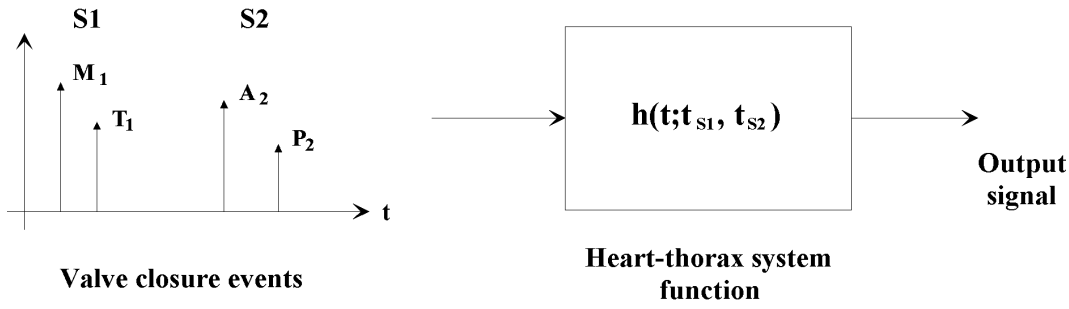


Figure 2. A simple heart sound production model.

valve closure events, are represented as impulses at the time of each event. The heart-thorax system is represented by its system function (impulse response). The notation $h(t; t_{s1}, t_{s2})$ indicates that the impulse response is time-varying, with different (time-invariant) responses over the periods of time corresponding to S1 and S2.

The characterization of the system, then, requires two steps. The first is the estimation of the relative amplitudes and times of the impulses representing the valve closures (a nontrivial task, given only the output signal). The second is the estimation of the S1 and S2 system functions. As is customary in linear systems, this second task will be done in the frequency domain, yielding the S1 and S2 transfer functions.

4. Determination of the Inputs and Transfer Functions

To determine the relative locations and amplitudes of the input impulses, we first observe that for stable, damped linear systems, the response to an impulse is maximum in magnitude at the time corresponding to the application of the impulse. When the input to the system is a sequence of impulses, then there will be peaks in energy in the output (which may or may not coexist with amplitude extrema in the output) for each impulse.

Locating such peaks is an ideal application for time-frequency or time-scale (wavelet) analysis. In these experiments, we have chosen to use a Coifman 4th order wavelet basis, decomposing the heart sounds into 7 levels (with the L th level corresponding to a basis function of extent 2^L). The results of applying this decomposition to the normal heart sound from Figure 1 (4096 samples in length, sampled at 8012 samples/second) are shown in Figure 3. The A7 coefficients are the residual values after the last level of decomposition. The remaining coefficients belong to basis functions of the given length (e.g., the coefficients for decomposition level D5 are for basis functions $2^5 = 32$ samples long).

It is clear that the information content at the different levels varies widely. Making a compromise between signal to noise ratio and temporal resolution, we have chosen to use the D5 coefficients to estimate the impulse locations. The result is a time resolution of 4 msec. This could be improved if a higher input signal to noise ratio could be obtained.

The magnitudes of the D5 coefficients for the normal heart sound are shown in Figure 4, with the coefficients for the S1 and S2 sounds extracted and displayed on

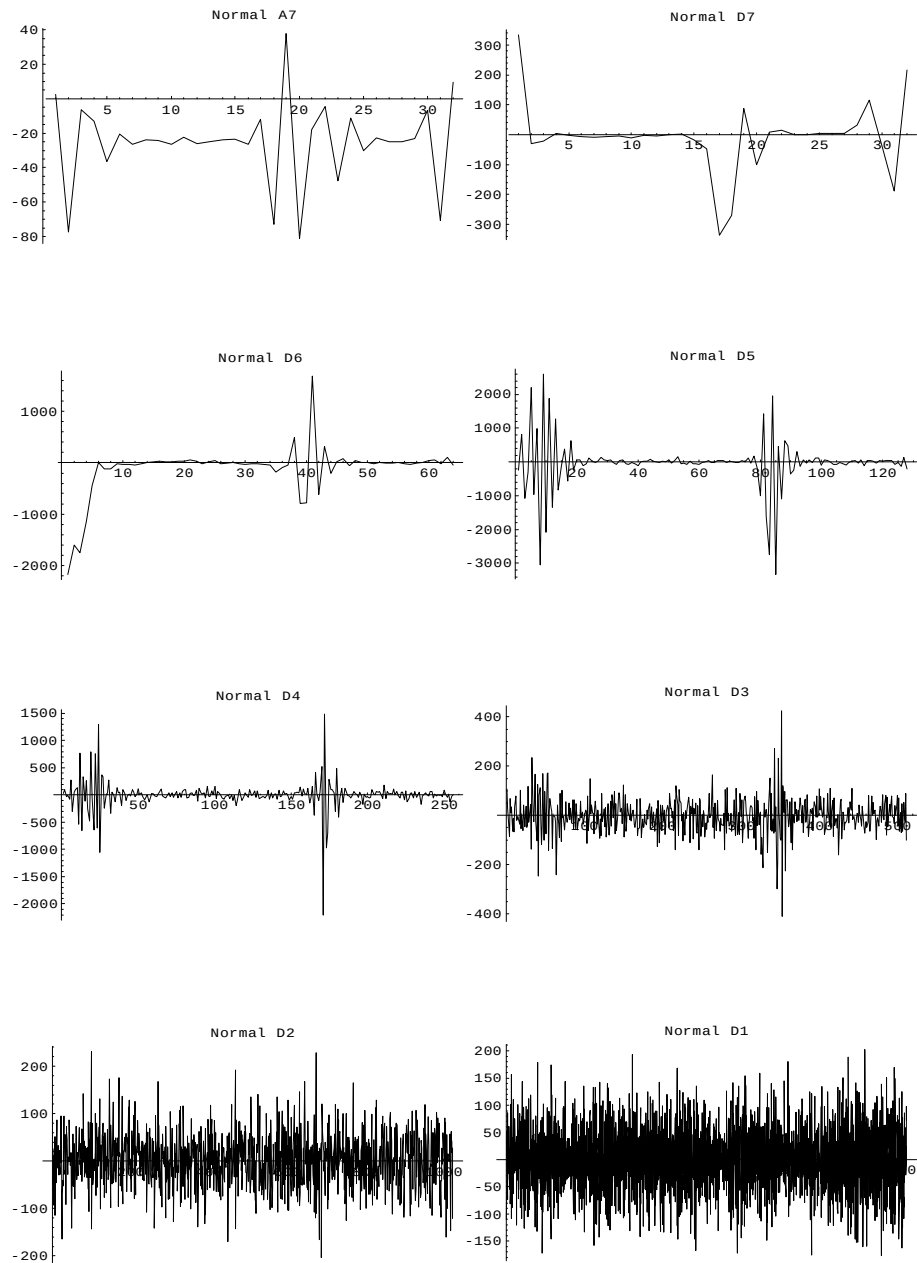


Figure 3. The Coifman 4th order wavelet coefficients for a normal heartbeat.

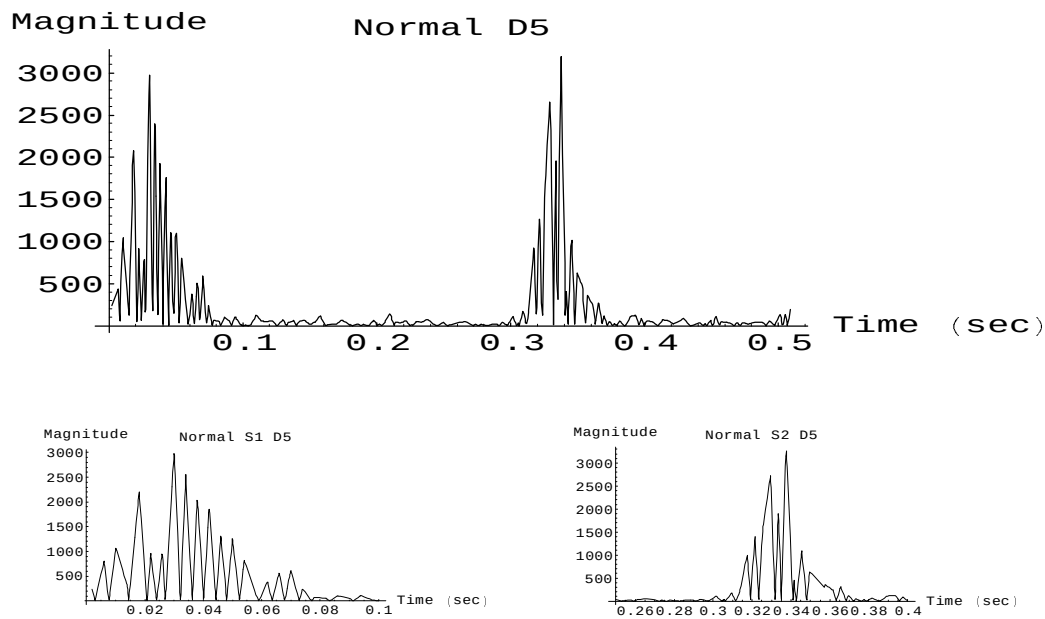


Figure 4. The magnitude of the D5 wavelet coefficients for a normal heart-beat (top), and S1 and S2 (bottom).

an expanded time scale at the bottom of the figure. From these results, it is estimated that the input impulses occur at 18 msec and 30 msec in S1 (with relative amplitudes .665 and .917) and at 330 msec and 338 msec in S2 (with relative amplitudes .824 and 1.00). In normal subjects, the mitral and tricuspid, and aortic and pulmonary valves have generally been found to close within 10 - 30 msec of each other. The estimated time between closures for S1 is therefore within the expected range, while that for S2 is somewhat shorter.

With the input to the system established, the S1 and S2 transfer functions can be found by dividing the discrete Fourier transforms of the S1 and S2 heart sounds by the transforms of the impulse pairs generating the sounds. Note that this requires that the transforms of the impulse pairs not include nulls, which is the case here. The resulting magnitude responses for the S1 and S2 transfer functions are shown in Figure 5. Their utility in discriminating between normal and abnormal heart sounds will be demonstrated in the next section.

5. The Characterization of Abnormal Heart Sounds

We will consider two types of abnormal heart sounds in this section: those resulting from coarctation of the aorta, and those exhibiting so-called “splits.”

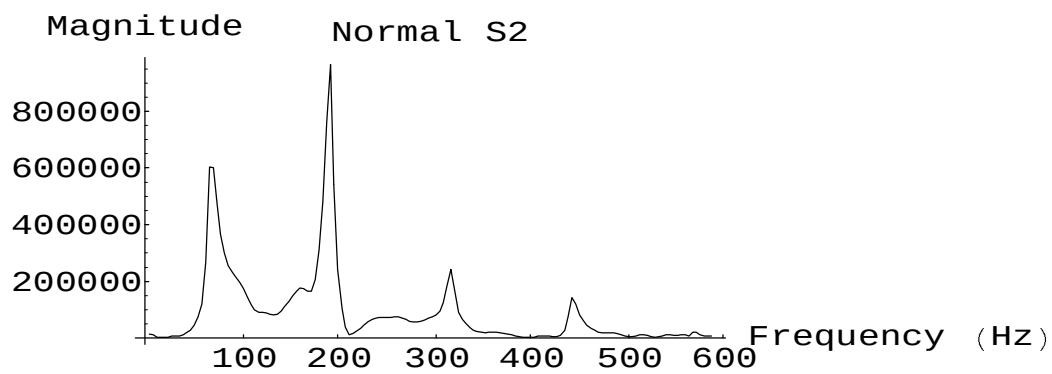
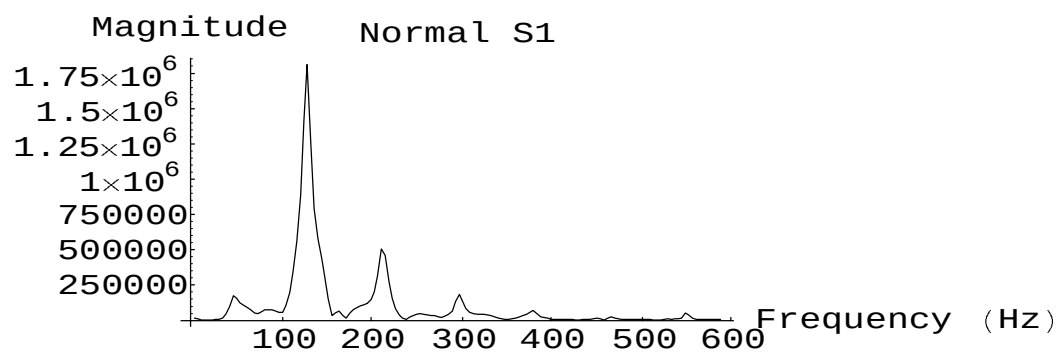


Figure 5. The magnitude responses of the normal S1 and S2 transfer functions.

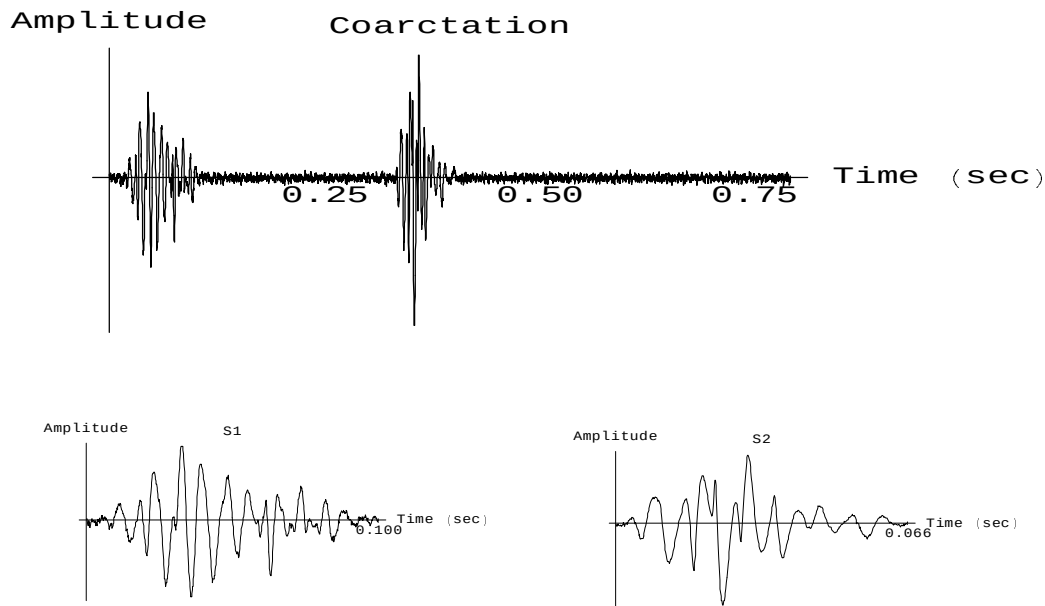


Figure 6. A heartbeat resulting from coarctation of the aorta.

5.1. Coarctation of the Aorta

As an example demonstrating the value of the S1 and S2 transfer functions for diagnosis, we will consider the case of coarctation of the aorta. A heartbeat from a patient exhibiting this abnormality is shown in Figure 6. While clearly different than the normal heartbeat in Figure 1 the differences are difficult to quantify.

Coarctation of the aorta is a constriction of the descending aorta, restricting the flow of oxygenated blood from the heart to the body. The result is elevated blood pressure in the left ventricle and the vessels before the coarctation, and reduced blood pressure in the circulatory system after the coarctation. Left ventricular hypertrophy (thickening of the walls of the left ventricle) results due to the increased pressure [15]. If left untreated, premature coronary artery disease is common, and ultimately heart failure.

Following the procedure described above, the D5 level of the wavelet decomposition of the heartbeat was examined, and the times and amplitudes of the input impulses due to valve closure were estimated. The resulting impulses were at 14 msec and 26 msec for S1 and 326 msec and 342 msec for S2. Their relative amplitudes were .552, 1.0, .545 and .888, respectively. The S1 and S2 transfer functions were then computed, the magnitude plots of which are shown in Figure 7.

Because this defect is associated with the aorta and left ventricle, it would be reasonable to expect its primary effects to be seen in S2. Comparing the normal S1 transfer function in Figure 5 with the S1 transfer function of the case with coarctation in Figure 7, we see that they are in fact very similar.

Comparing the normal and abnormal S2 transfer functions, however, there are clear

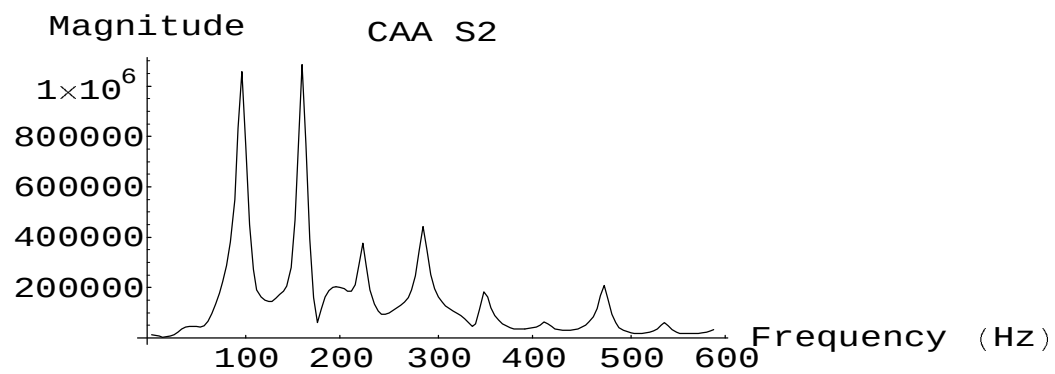
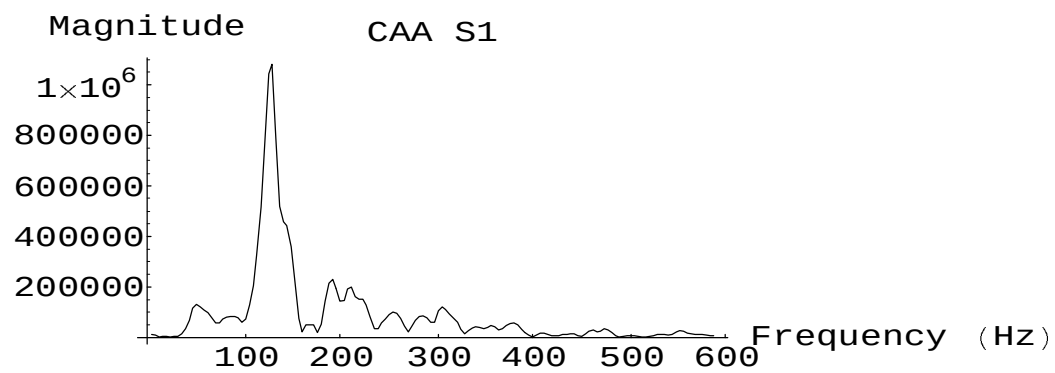


Figure 7. The magnitude responses of the S1 and S2 transfer functions with coarctation of the aorta.

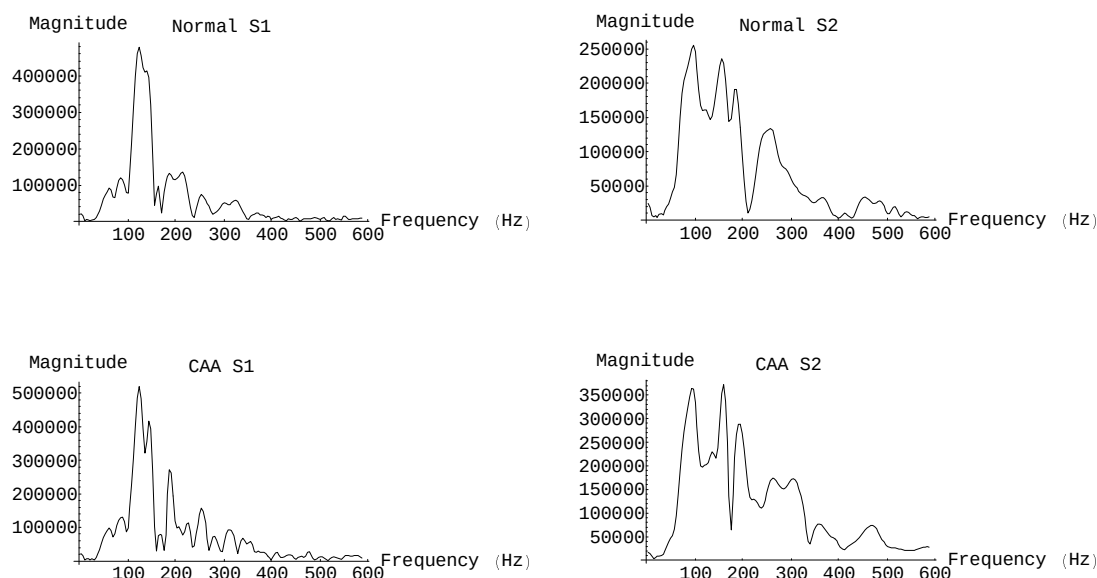


Figure 8. The magnitudes of the Fourier transforms of the normal S1 and S2 sounds and those with coarctation of the aorta.

differences. There are two pronounced additional peaks in the case with coarctation, together with shifts in the locations and relative amplitudes of the peaks observed in the normal case. A correlation between these effects and the underlying physiology will require substantial additional investigation (complicated by the fact that coarctation of the aorta often coexists with other defects, such as a bicuspid aortic valve). However, it is clear that sufficient information exists to discriminate between the two cases.

It would be natural at this point, given that the above discussions are in the frequency domain, to ask whether one could bypass the calculation of the S1 and S2 transfer functions and simply examine the S1 and S2 power spectra (avoiding the estimation of the valve closure times and amplitudes). The magnitudes of the Fourier transforms of S1 and S2 for these two cases are shown in Figure 8. While sufficient study reveals that the basic features shown in the transfer functions exist in the Fourier transforms of the signals themselves (as they of course must), the distinguishing features between the cases are much more difficult to identify. Normalization by the Fourier transforms of the input impulse sequences, as done in the calculation of the transfer functions, serves to sharpen and enhance the features of interest.

5.2. Split Sounds

Because the mitral and tricuspid, and aortic and pulmonary valves typically close within less than 30 msec, the S1 and S2 heart sounds are generally perceived by physicians as unified, single sounds. That is, the components of the sounds due to the individual valve closures are not individually audible. There are a number of circum-

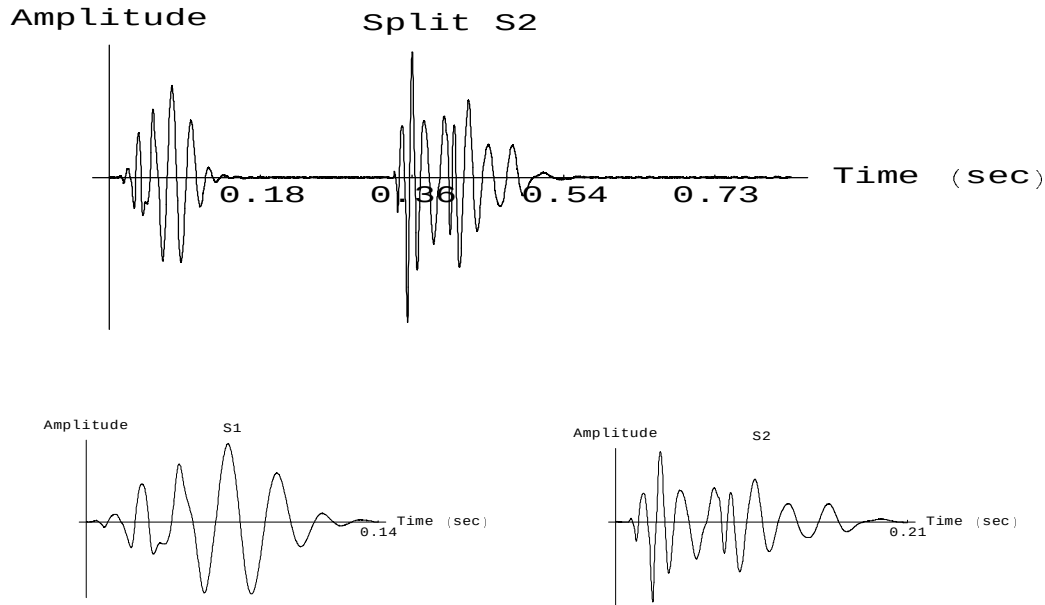


Figure 9. A heartbeat with split S2.

stances, however, that can lead to an increased interval between valve closures. Some of these circumstances involve an underlying abnormality, while others do not. In either case, the resulting heart sounds become resolvable into two components, and are referred to as “split.”

The time intervals between valve closures are therefore important diagnostic cues. As demonstrated above in the process of finding the S1 and S2 transfer functions, these time intervals can be estimated using local frequency/scale analysis.

A heart sound with a split S2 is shown in Figure 9. Examining the D5 wavelet coefficient magnitudes for this sound (Figure 10), magnitude peaks can be seen at 14, 26, 338, and 382 milliseconds. Based on these peaks, the interval between the closure of the mitral and tricuspid valves (in S1) is estimated to be 12 msec, while the time between the closure of the aortic and pulmonary valves (in S2) is estimated to be 44 msec. The interval between the closures in S1 is therefore within the normal range, while the interval for S2 is indicative of a (wide) split.

6. Conclusions and Future Work

In this paper, we have introduced a simple model for the generation of heart sounds, and demonstrated its usefulness as a source of relevant features for cardiac diagnosis. Establishing a correlation between different pathologies and specific features in the transfer functions, and the evaluation of the utility of this approach as part of a complete computer-based diagnostic aid (e.g., in conjunction with the system described in [13]) are areas of future work.

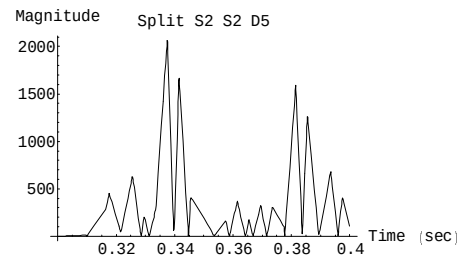
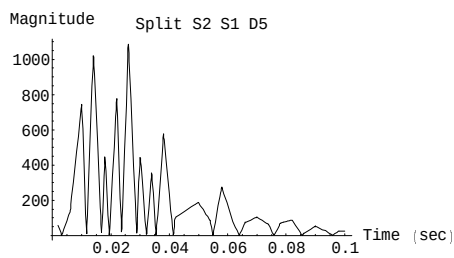
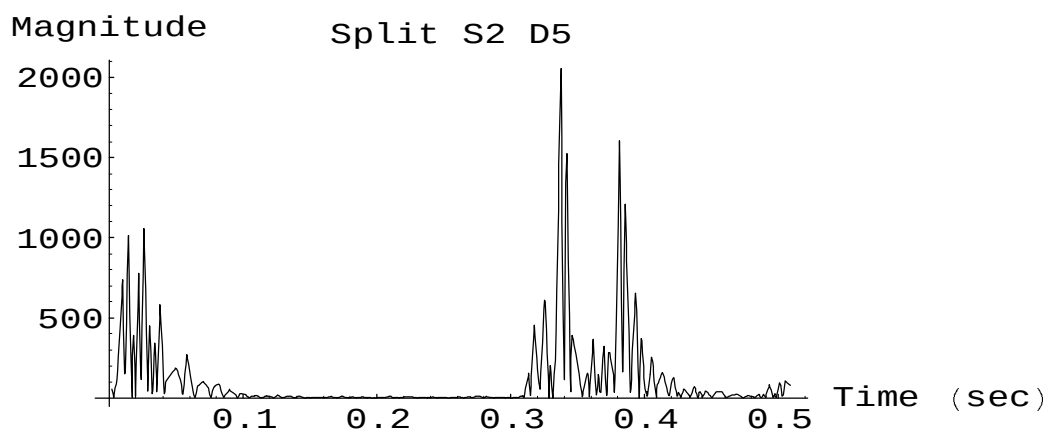


Figure 10. The magnitude of the D5 wavelet coefficients for a heartbeat with split S2 (top), and those for S1 and S2 (bottom).

Acknowledgments

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