



Analysis of Concurrent, Coupled, and Correlated Signal Processes

Lecture #5

Analysis of Concurrent, Coupled, and Correlated Signal Processes

Introduction

The human body is a complex integration of a number of biological systems with several ongoing physiological, functional, and possibly pathological processes. Most biological processes within a body are not independent of one another; rather, they are mutually correlated and bound together by physical or physiological control and communication phenomena. Analyzing any single process without due attention to others that are concurrent, coupled, or correlated with the process may provide only partial information and pose difficulties in the comprehension of the process. The problem, then, is how do we recognize the existence of concurrent, coupled, and correlated phenomena? How do we obtain the corresponding signals and identify the correlated features? Unfortunately, there is no simple or universal rule to apply to this problem.

Ideally, an investigator should explore the system or process of interest from all possible angles and use multidisciplinary approaches to identify several potential sources of information. The signals so obtained may be electrical, mechanical, biochemical, or physical, among the many possibilities, and may exhibit interrelationships confounded by peculiarities of transduction, time delays, multipath transmission or reflection, waveform distortions, and filtering effects that may need to be accounted for in their simultaneous analysis. Events or waves in signals of interest may be nonspecific and difficult to identify and analyze. How could we exploit the concurrency, coupling, and correlation present between processes or related signals to better understand a system?

PROBLEM STATEMENT

“Determine the correspondences, correlation, and inter-relationships present between concurrent signals related to a common underlying physiological system or process, and identify their potential applications.”

The statement above represents, of necessity at this stage of the discussion, a rather vague and generic problem. The case-studies and applications presented in the



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following sections provide a few illustrative examples dealing with specific systems and problems. Signal processing techniques for the various tasks identified in the case-studies will be developed in chapters that follow. Note that the examples cover a diverse range of systems, processes, and signals. The specific problem of your interest will very likely not be directly related to any of the case-studies presented here. It is expected that a study of the examples provided will expand the scope of your analytical skills and lead to improved solution of your specific case.

ILLUSTRATION OF THE PROBLEM WITH CASE-STUDIES

The electrocardiogram and the phonocardiogram

A clinical ECG record typically includes 12 channels of sequentially or simultaneously recorded signals, and can be used on its own to diagnose many cardiac diseases. This is mainly due to the simple and readily identifiable waveforms in the ECG, and the innumerable studies that have firmly established clinical ECG as a standard procedure, albeit as an empirical one. The PCG, on the other hand, is a more complex signal. PCG waveforms cannot be visually analyzed except for the identification of gross features such as the presence of murmurs, time delays as in a split S2, and envelopes of murmurs. An advantage with the PCG is that it may be listened to; auscultation of heart sounds is more commonly performed than visual analysis of the PCG signal. However, objective analysis of the PCG requires the identification of components, such as S1 and S2, and subsequent analysis tailored to the nature of the components.

Given a run of a PCG signal over several cardiac cycles, visual identification of S1 and S2 is possible if there are no murmurs between the sounds, and if the heart rate is low such that the S2 - S1 (of the next beat) interval is longer than the S1 - S2 interval (as expected in normal situations). At high heart rates and with the presence of murmurs or premature beats, identification of S1 and S2 could be difficult.

Problem: Identify the beginning of S1 in a PCG signal and extract the heart sound signal over one cardiac cycle.

Solution: The ECG and PCG are concurrent phenomena, with the noticeable difference that the former is electrical while the latter is mechanical (sound or vibration). It is customary to record the ECG with the PCG; see Figures 1 and 2 for examples.



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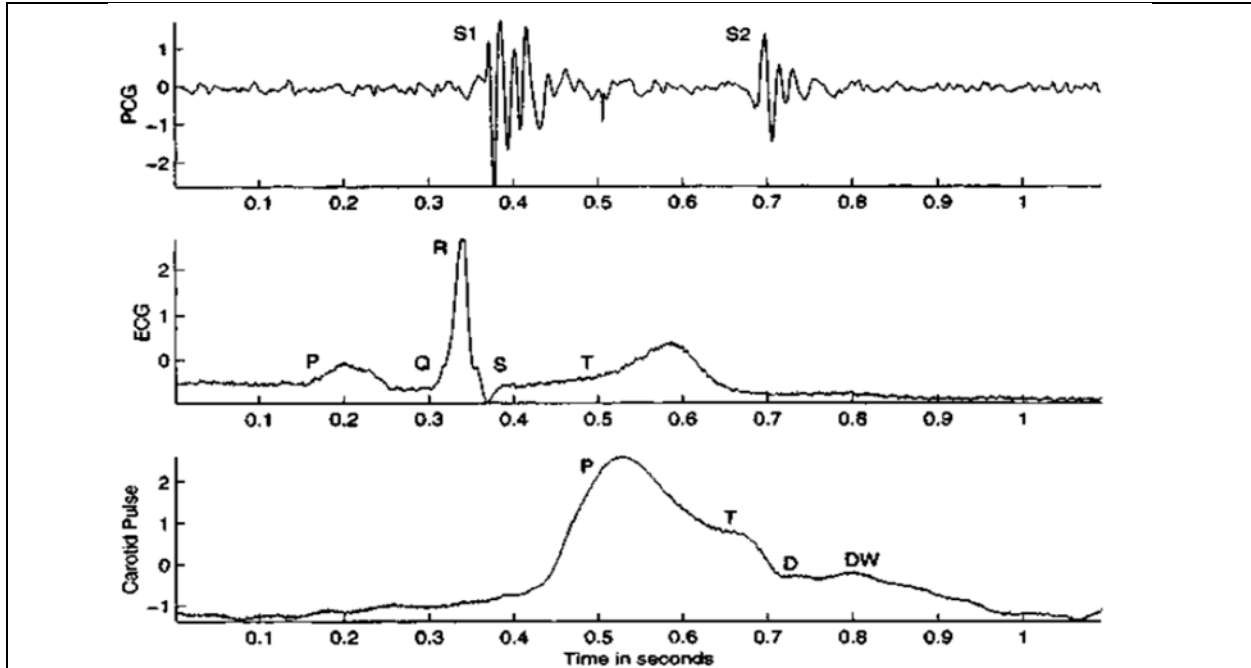


Figure 1: Three-channel simultaneous record of the PCG, ECG, and carotid pulse signals of a normal male adult.

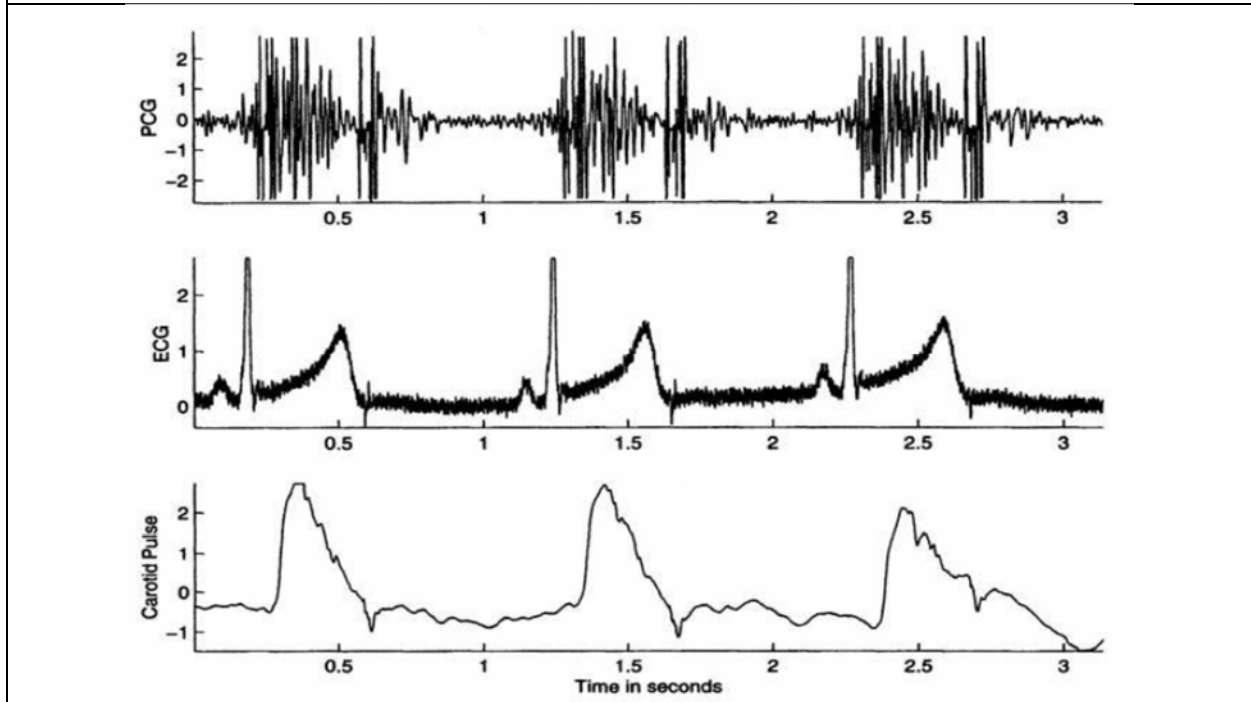


Figure 2: Three-channel simultaneous record of the PCG, ECG, and carotid pulse signals of a patient (female, 11 years) with aortic stenosis. Note the presence of the typical diamond shaped systolic murmur and the split nature of S2 in the PCG.



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The QRS wave in the ECG is directly related to ventricular contraction, as the summation of the action potentials of ventricular muscle cells.

As the ventricles contract, the tension in the chordae tendineae and the pressure of retrograde flow of blood toward the atria seal the AV valves shut, thereby causing the initial vibrations of S1. Thus S1 begins immediately after the QRS complex. Given the nonspecific nature of vibration signals and the various possibilities in the transmission of the heart sounds to the recording site on the chest, detection of S1 on its own is a difficult problem.

Detection of the QRS is fairly easy, given that the QRS is the sharpest wave in the ECG over a cardiac cycle; in fact, the P and T waves may be almost negligible in many ECG records. Thus the QRS complex in the ECG is a reliable indicator of the beginning of S1, and may be used to segment a PCG record into individual cardiac cycles: from the beginning of one QRS (and thereby S1) to the beginning of the next QRS and S1. This method may be applied visually or via signal processing techniques

The phonocardiogram and the carotid pulse

Identification of the diastolic segment of the PCG may be required in some applications in cardiovascular diagnosis. Ventricular systole ends with the closure of the aortic and pulmonary valves, indicated by the aortic (A2) and pulmonary (P2) components of the second heart sound S2. The end of contraction is also indicated by the T wave in the ECG, and S2 appears slightly after the end of the T wave (see Figure 1). S2 may be taken to be the end of systole and the beginning of ventricular relaxation or diastole. Shaver et al. and Reddy et al. have included S2 in the part of their article on systolic sounds.) However, as in the case of S1, S2 is also a nonspecific vibrational wave that cannot be readily identified (even visually), especially when murmurs are present.

Given the temporal relationship between the T wave and S2, it may appear that the former may be used to identify the latter. This, however, may not always be possible in practice, as the T wave is often a low-amplitude and smooth wave and is sometimes not recorded at all (see Figure 3). ST segment elevation or depression may make even visual identification of the end of the T wave difficult. Thus the T wave is not a reliable indicator to use for identification of S2.



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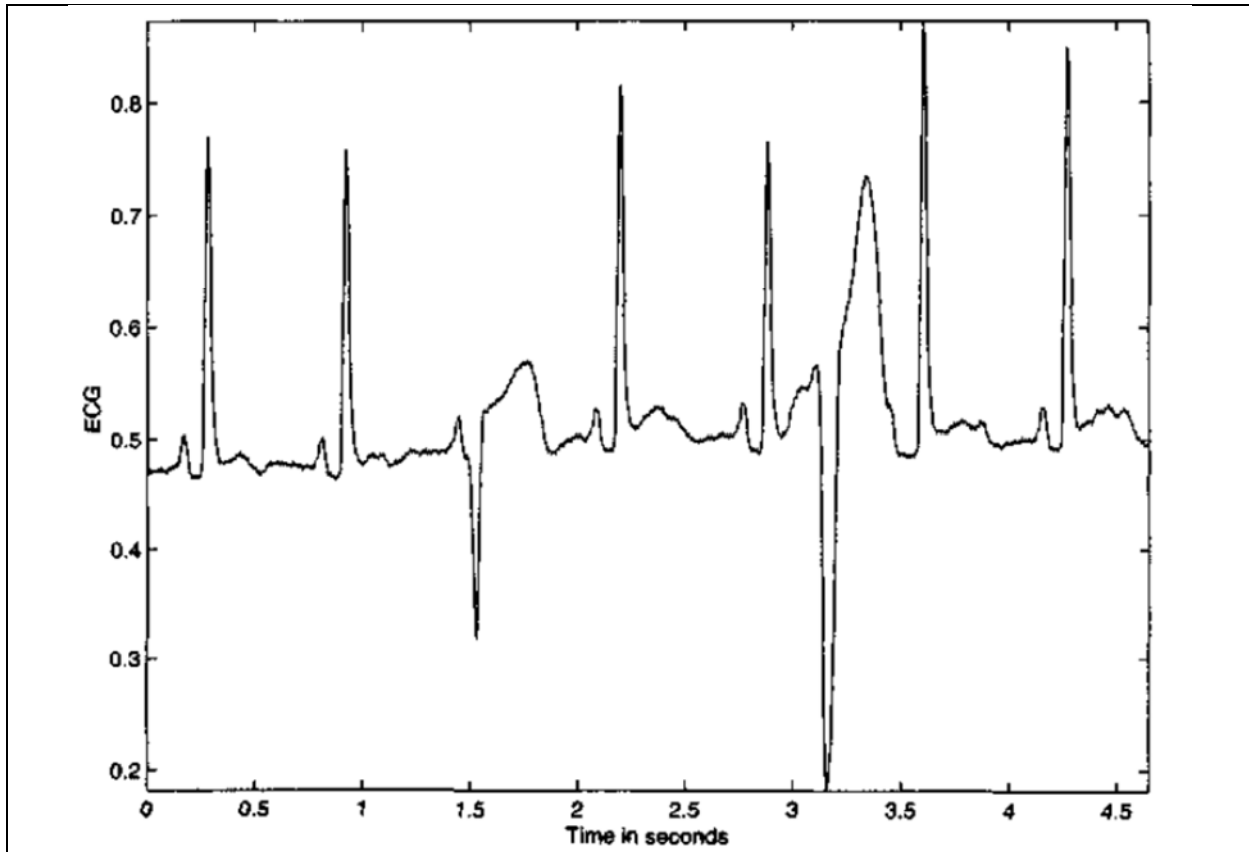


Figure 3: ECG signal with PVCs. The third and sixth beats are PVCs. The first PVC has blocked the normal beat that would have appeared at about the same time instant, but the second PVC has not blocked any normal beat triggered by the SA node.

Problem: Identify the beginning of S2 in a PCG signal.

Solution: Given the inadequacy of the T wave as an indicator of diastole, we need to explore other possible sources of information. Closure of the aortic valve is accompanied by deceleration and reversal of blood flow in the aorta. This causes a sudden drop in the blood pressure within the aorta, which is already on a downward slope due to the end of systolic activity. The sudden change in pressure causes an notch in the aortic pressure wave (see Figures 4 and 5). The aortic pressure signal may be obtained using catheter-tip sensors, but the procedure would be invasive. Fortunately, the notch is transmitted through the arterial system, and may be observed in the carotid pulse recorded at the neck.



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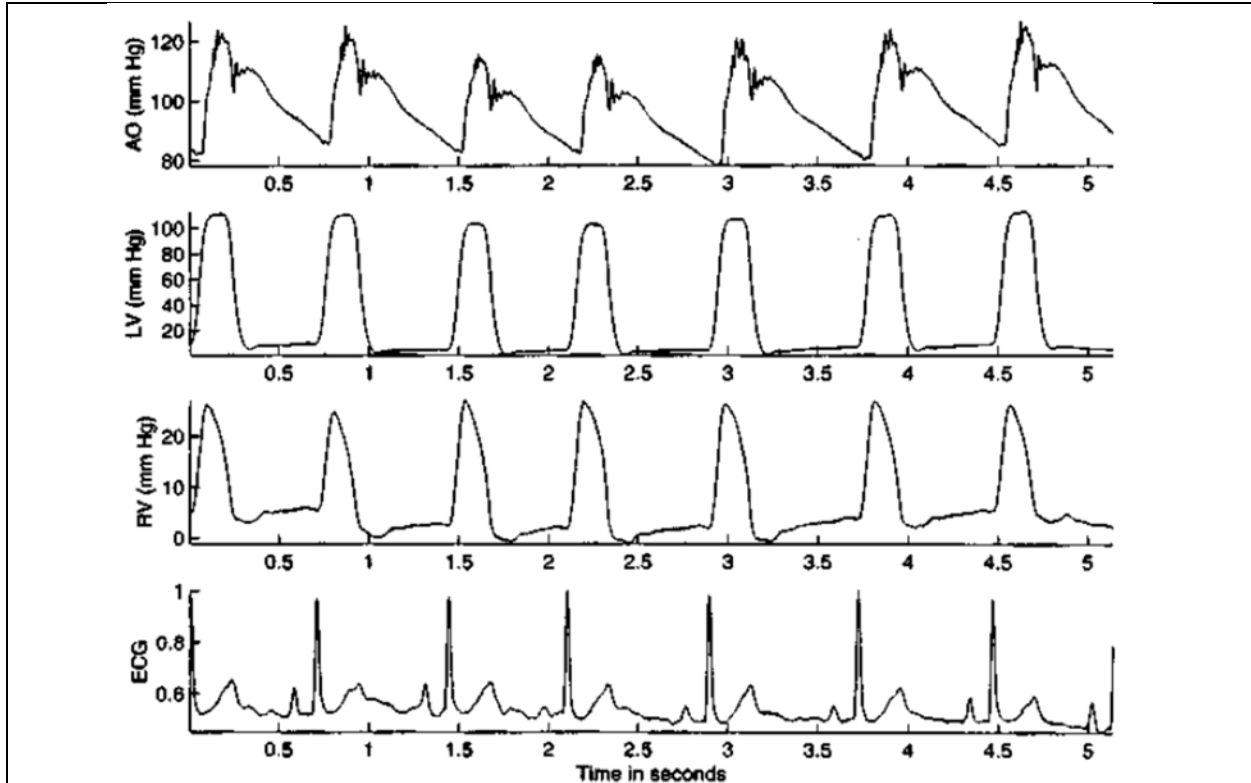


Figure 4: Normal ECG and intracardiac pressure signals from a dog. A0 represents aortic pressure near the aortic valve.

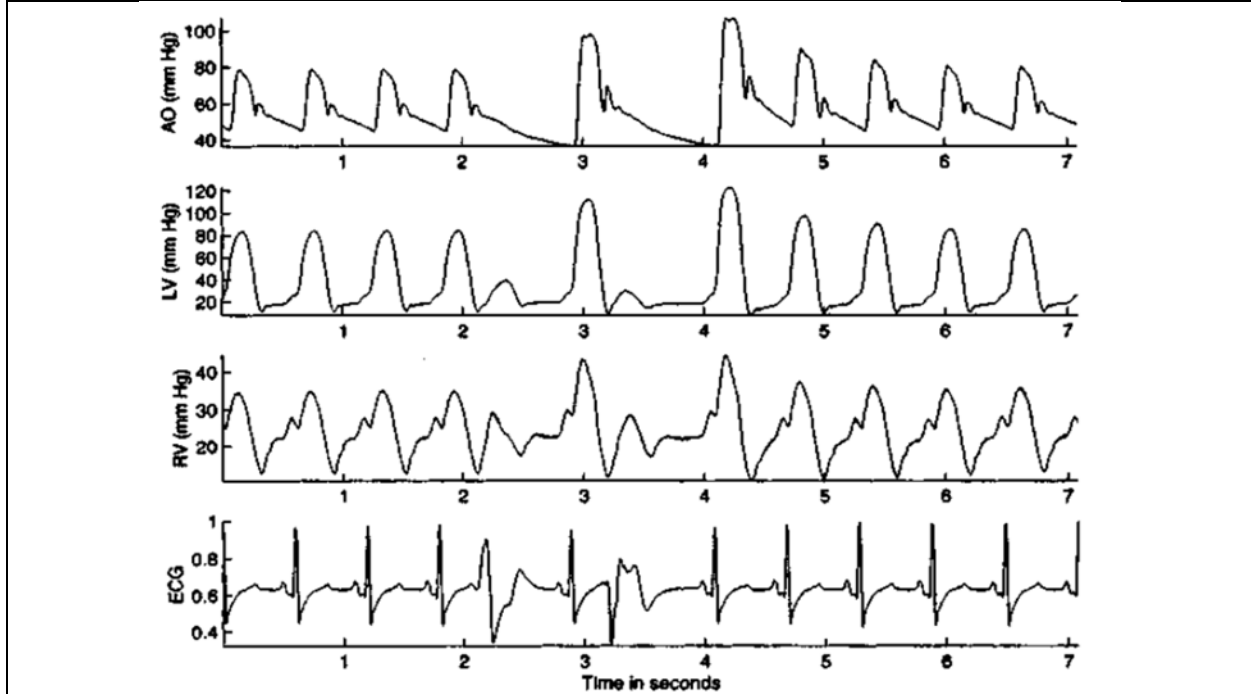


Figure 5: ECG and intracardiac pressure signals from a dog with PVCs.



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The dicotic notch D in the carotid pulse signal will bear a delay with respect to the corresponding notch in the aortic pressure signal, but has the advantage of being accessible in a noninvasive manner. (Similar events occur in the pulmonary artery, but provide no externally observable effects.) See Figures 1 and 2 for examples of three-channel PCG - ECG - carotid pulse recordings that illustrate the D - S₂ - T relationships. The dicotic notch may thus be used as a reliable indicator of the end of systole or beginning of diastole that may be obtained in a noninvasive manner. The average S₂ - D delay has been found to be 42.6 rns with a standard deviation of 5 ms, which should be subtracted from the dicotic notch position to obtain the beginning of S₂.

The ECG and the atrial electrogram

Most studies on the ECG and the PCG pay more attention to ventricular activity than to atrial activity, and even then, more to left ventricular activity than to the right. Rhythm analysis is commonly performed using QRS complexes to obtain inter-beat intervals known as RR intervals. Such analysis neglects atrial activity.

Recollect that the AV node introduces a delay between atrial contraction initiated by the SA node impulse and the consequent ventricular contraction. This delay plays a major role in the coordinated contraction of the atria and the ventricles. Certain pathological conditions may disrupt this coordination, and even cause AV dissociation. It then becomes necessary to study atrial activity independent of ventricular activity and establish their association, or lack thereof. Thus the interval between the P wave and the QRS (termed the PR interval) would be a valuable adjunct to the RR interval in rhythm analysis. Unfortunately, the atria, being relatively small chambers with weak contractile activity, cause a small and smooth P wave in the external ECG. Quite often the P wave may not be recorded or seen in the external ECG; see, for example, leads I and V₃ - V₆ in Figure 6.

Problem: Obtain an indicator of atrial contraction to measure the PR interval.

Solution: One of the reasons for the lack of specificity of the P wave is the effect of transmission from the atria to the external recording sites. An obvious solution would be to insert electrodes into one of the atria via a catheter and record the signal at the source. This would, of course, constitute an invasive procedure.



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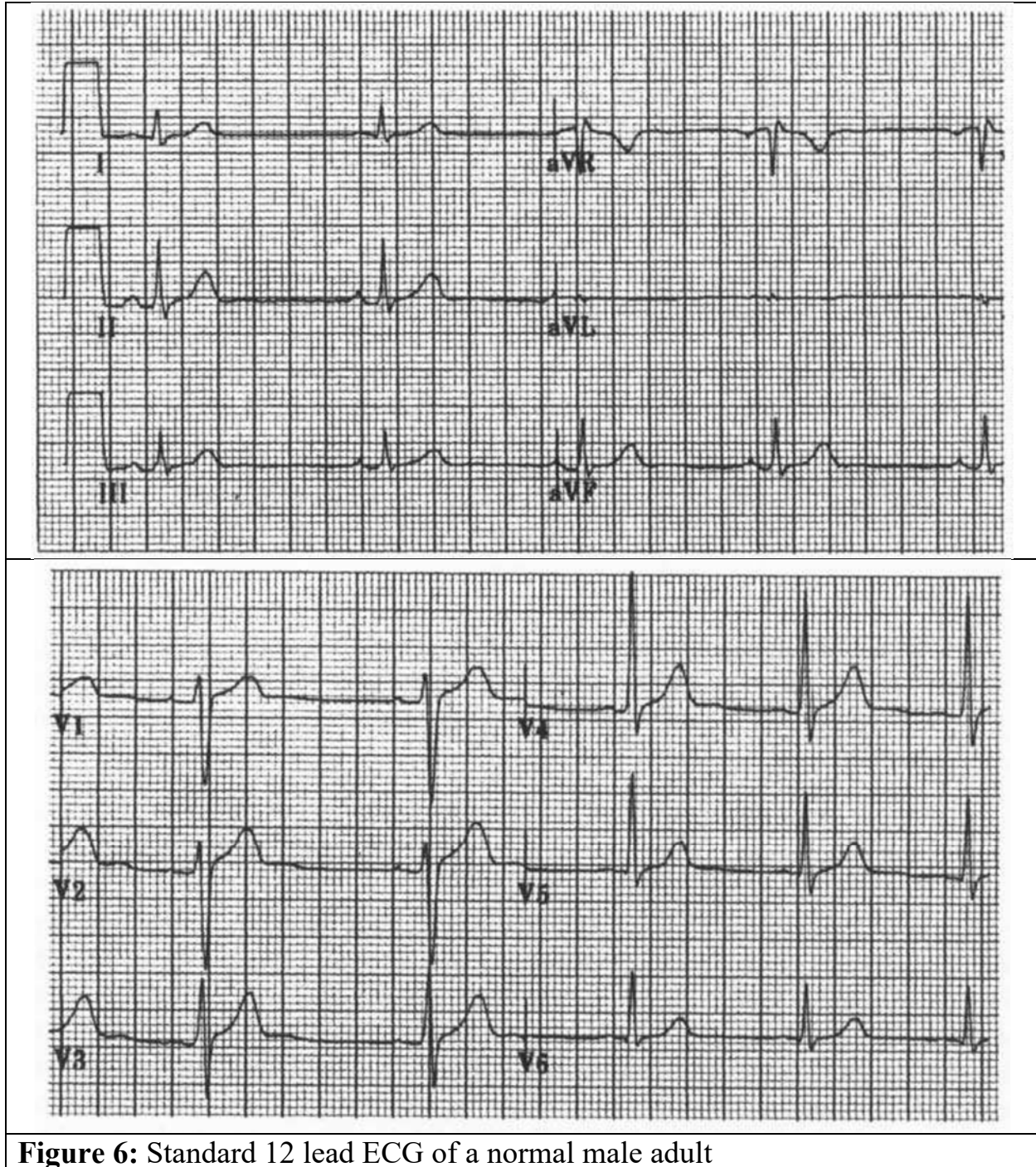


Figure 6: Standard 12 lead ECG of a normal male adult

Jenkins et al. [67, 68, 29, 30] proposed a unique and very interesting procedure to obtain a strong and clear signal of atrial activity: they developed a pill electrode that could be swallowed and lowered through the esophagus to a position close to



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the left atrium (the bipolar electrode pill being held suspended by wires about 35 cm from the lips). The procedure may or may not be termed invasive, although an object is inserted into the body (and removed after the procedure), as the action required is that of normal swallowing of a tablet-like object. The gain required to obtain a good atrial signal was 2 - 5 times that used in ECG amplifiers. With a 5 - 100 Hz bandpass filter, Jenkins et al. obtained an SNR of 10.

Figure 7 shows recordings from a normal subject of the atrial electrogram from the pill electrode and an external ECG lead. Atrial contraction is clearly indicated by a sharp spike in the atrial electrogram. Measurement of the PR interval (or the AR interval, as called by Jenkins et al.) now becomes an easy task, with identification of the spike in the atrial electrogram (the “A” wave, as labeled by Jenkins et al.) being easier than identification of the QRS in the ECG.

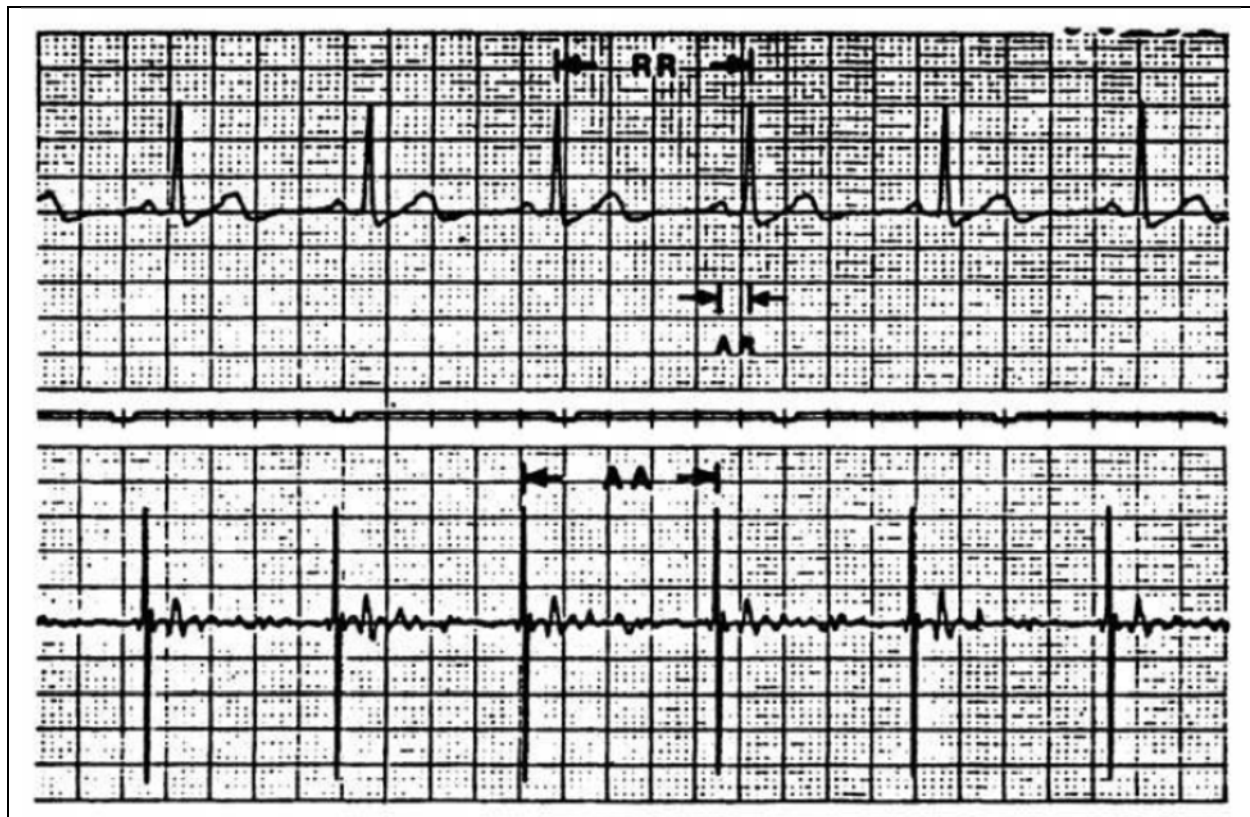


Figure 7: Pill-electrode recording of the atrial electrogram (lower tracing) and the external ECG (upper tracing) of a normal subject. The pulse train between the two signals indicates intervals of 1 s.



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Figure 2.2 shows the atrial electrogram and external ECG of a subject with ectopic beats. The PVCs have no immediately preceding atrial activity. The first PVC has blocked the conduction of the atrial activity occurring immediately after, resulting in a compensatory pause before the following normal beat. The second PVC has not blocked the subsequent atrial wave, but has caused a longer-than-normal AV delay and an aberrant conduction path, which explains the different waveshape of the consequent beat. The third PVC has not affected the timing of the following SA node-initiated pulse, but has caused a change in waveshape in the resulting QRS-T by altering the conduction path.

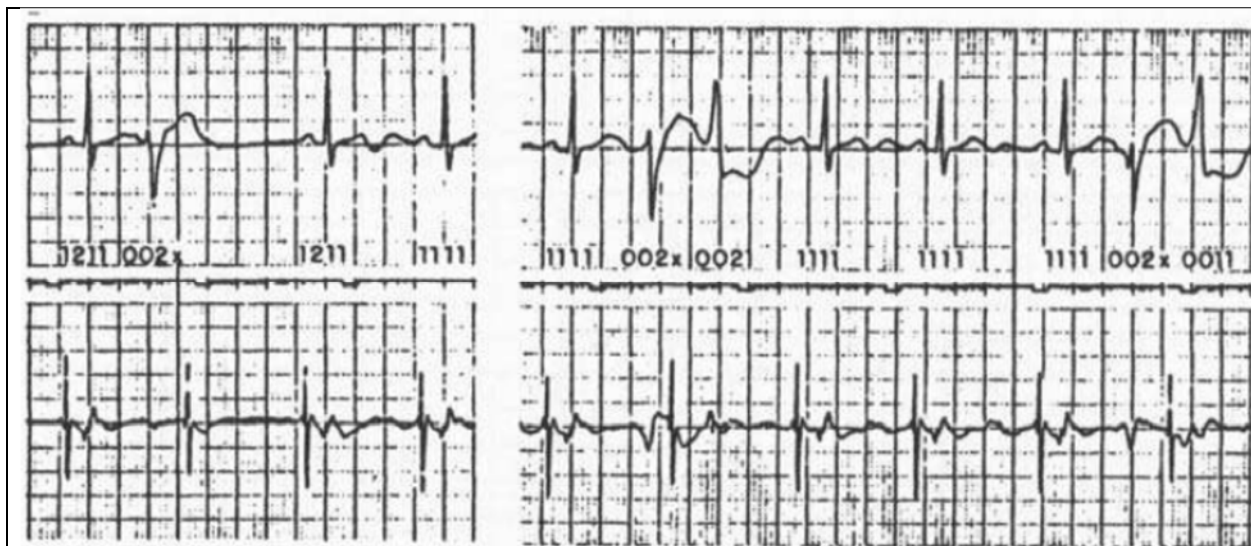


Figure 8: Atrial electrogram (lower tracing) and the external ECG (upper tracing) of a subject with ectopic beats. The pulse train between the two signals indicates intervals of 1 s.

Jenkins et al. developed a four-digit code for each beat, as illustrated in Figure 8. The first digit was coded as

0: abnormal waveshape, or

1: normal waveshape,

as determined by a correlation coefficient computed between the beat being processed and a normal template. The remaining three digits encoded the nature of the RR, AR, and AA intervals, respectively, as

0: short,



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1: normal, or

2: long.

The absence of a preceding A wave related to the beat being analyzed was indicated by the code z in the fourth digit (in which case the AR interval is longer than the RR interval). Figure 8 shows the code for each beat. Based upon the code for each beat, Jenkins et al. were able to develop a computerized method to detect a wide variety of arrhythmia.

Cardio-respiratory interaction

The heart rate is affected by normal breathing due to the coupling and interaction existing between the cardiac and respiratory systems. Breathing also affects the transmission of the heart sounds from the cardiac chambers to the chest surface. Durand et al. recorded intracardiac and chest-surface FTG signals and derived the dynamic transfer function of the heart - thorax acoustic system in dogs. Analysis of the synchronization and coupling within the cardio-respiratory system could require sophisticated analysis of several signals acquired simultaneously from the cardiac and respiratory systems.

The electromyogram and the vibromyogram

The EMG signal has been studied extensively and the relationship between EMG signal parameters and muscle contraction level has been established. It is known that the EMG root mean-squared (RMS) and mean frequency values increase with increasing muscle contraction until fatigue sets in, at which point both values begin to decrease. In this situation, while the muscle output measured is mechanical contraction (using force or strain transducers), the signal analyzed is electrical in character. A direct mechanical signal related to basic muscle-fiber or motor unit phenomena may be desired in some situations.

Problem: Obtain a mechanical signal that is a direct indicator of muscle-fiber or motor unit activity to study muscle contraction and force development.

Solution: The VMG, as introduced in Section 1.2.12, is a vibration signal measured from a contracting muscle. The signal is a direct manifestation of the contraction of muscle fibers, and as such represents mechanical activity at the muscle-fiber or motor-unit level. The VMG signal is the mechanical counterpart and contemporary



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of the EMG signal. Although no direct relationship has been established between the force outputs of individual motor units and the net force output of the muscle, it has been shown that the RMS and mean frequency parameters of the VMG signal increase with muscle force output, in patterns that parallel those of the EMG. Thus the VMG may be used to quantify muscular contraction.

Given the simplicity and noninvasive nature of EMG and VMG measurement, simultaneous analysis of the two signals is an attractive and viable application. Such techniques may find use in biofeedback and rehabilitation. Figure 9 shows simultaneous EMG - VMG recordings at two levels of contraction of the rectus femoris muscle. Both signals are interference patterns of several active motor units even at low levels of muscle effort, and cannot be analyzed visually. However, a general increase in the power levels of the signals from the lower effort to the higher effort case may be observed.

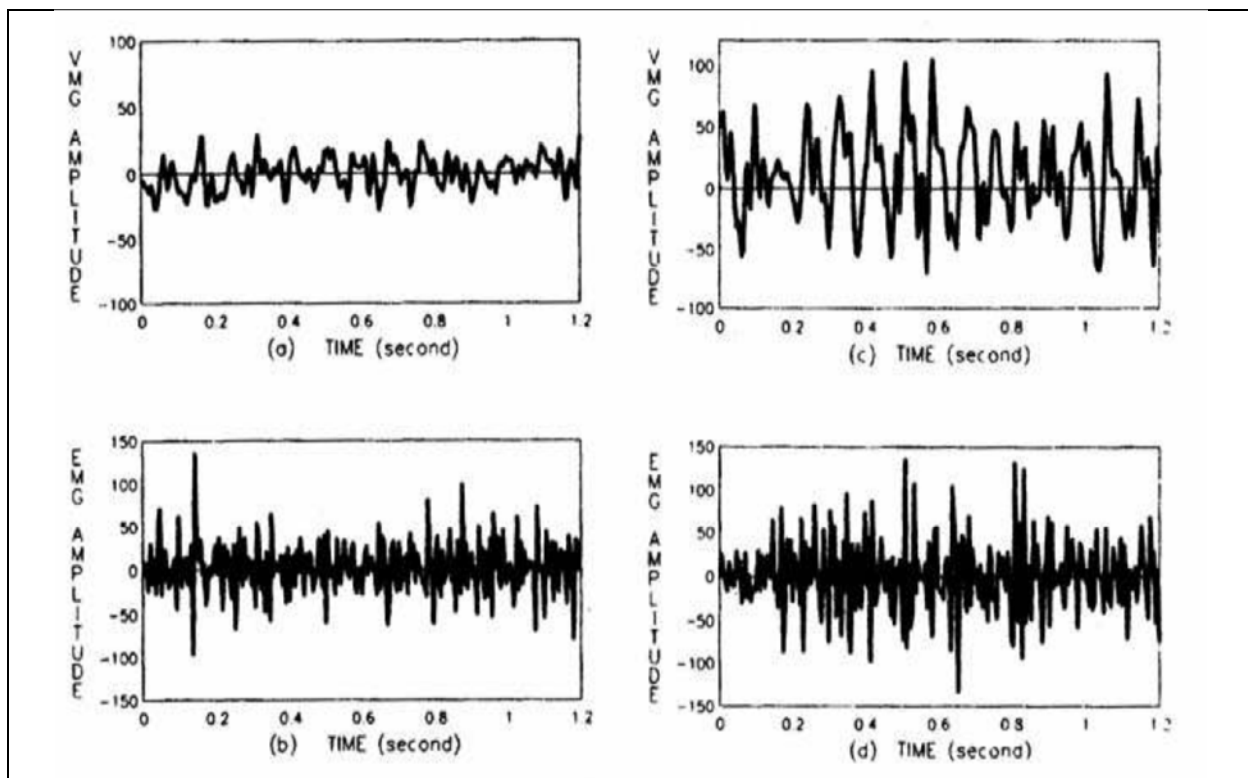


Figure 9: Simultaneous EMG - VMG records at two levels of contraction of the rectus femoris muscle. (a) VMG at 40% of the maximal voluntary contraction (MVC) level. (b) EMG at 40% MVC. (c) VMG at 60% MVC. (d) EMG at 60% MVC.



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APPLICATION: SEGMENTATION OF THE PCG INTO SYSTOLIC AND DIASTOLIC PARTS

Problem: Show how the ECG and carotid pulse signals may be used to break a PCG signal into its systolic and diastolic parts.

Solution: A cardiac cycle may be divided into two important parts based upon ventricular activity: systole and diastole. The systolic part starts with S1 and ends at the beginning of S2; it includes any systolic murmur that may be present in the signal. The diastolic part starts with S2, and ends just before the beginning of the S1 of the next cardiac cycle. (The aortic and pulmonary valves close slightly before the A2 and P2 components of S2. Therefore systole may be considered to have ended just before S2. Although Shaver et al. and Reddy et al. have included S2 in the part of their article on systolic sounds, we shall include S2 in the diastolic part of the PCG.) The diastolic part includes any diastolic murmur that may be present in the signal; it might also include S3 and S4, if present, as well as AV valve-opening snaps, if any.

We saw previously that the QRS complex in the ECG may be used as a reliable marker of the beginning of S1. We also saw, that the dicrotic notch in the carotid pulse may be used to locate the beginning of S2. Thus, if we have both the ECG and carotid pulse signals along with the PCG, it becomes possible to break the PCG into its systolic and diastolic parts.

Figure 10 shows three-channel PCG - ECG - carotid pulse signals of a subject with systolic murmur due to aortic stenosis (the same as in Figure 2), with the systolic and diastolic parts of the PCG marked in relation to the QRS and D events. The demarcation was performed by visual inspection of the signals in this example.



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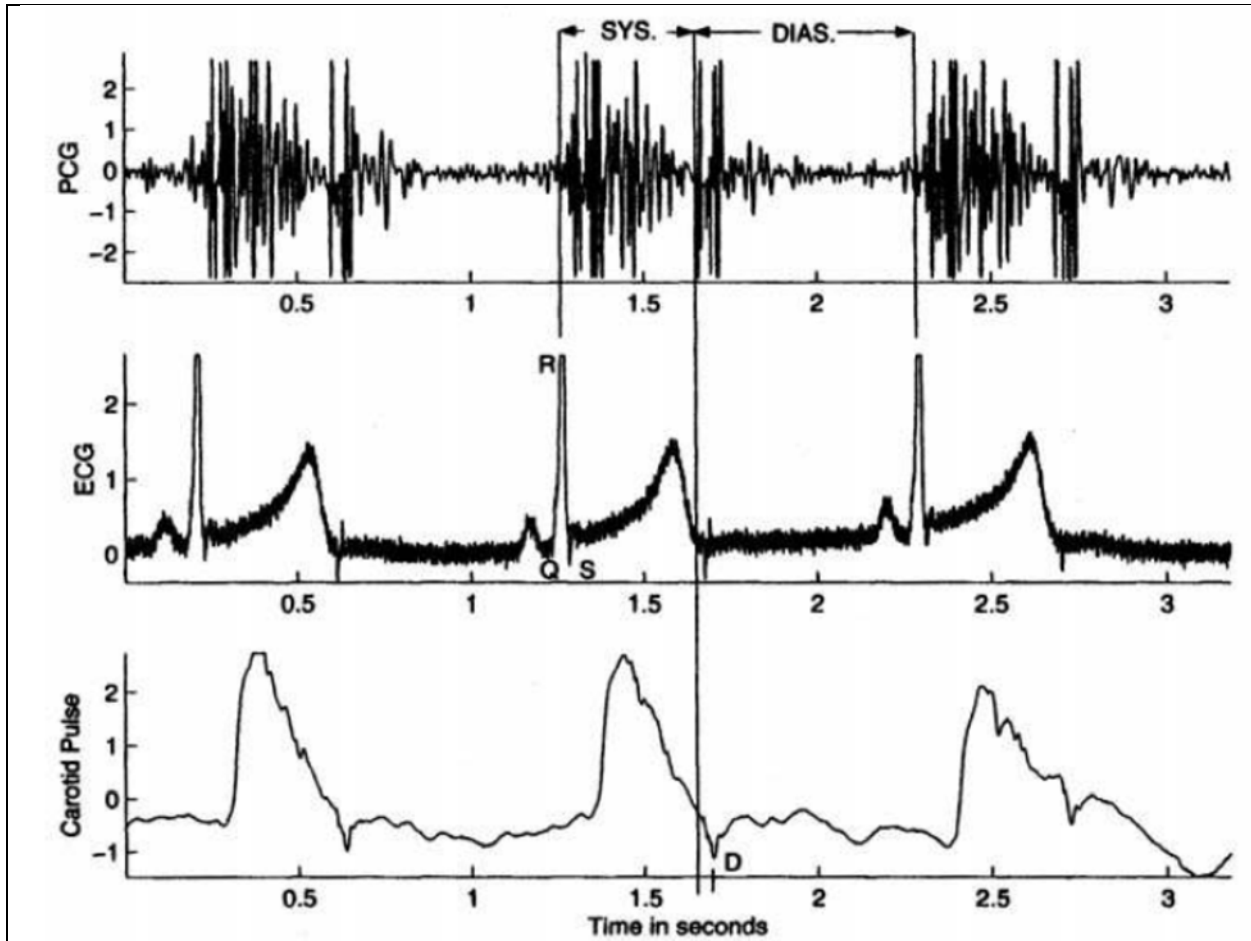


Figure 10: Demarcation of the systolic (SYS.) and diastolic (DIAS.) parts of the PCG signal in Figure 2 by using the ECG and carotid pulse as reference signals. The QRS complex and the dicotic notch D are marked on the ECG and carotid pulse signals, respectively.