Physiological measurement

Noninvasive acoustical detection of coronary artery disease using the adaptive line enhancer method

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Abstract—*Previous studies have indicated that heart sounds may contain information which is useful in the detection of occluded coronary arteries. Specifically, previous work based on analysing heart sounds recorded during the diastolic portion of the cardiac cycle, when blood flow through the coronary arteries is maximum, has shown that additional frequency components are present in patients with coronary artery disease. To further explore the application of advanced signal processing techniques to the noninvasive detection of coronary artery disease, a new signal-processing approach is presented using adaptive line enhancing (ALE) and spectral estimation of diastolic heart sounds taken from recordings made at the patient's bedside. This approach comprises two cascaded processes. In the first the ALE method is used to enhance the diastolic heart sounds and eliminate background noise. In the second process, either autoregressive (AR) or autoregressive moving average (ARMA) spectral methods are used to estimate the model parameters. Model parameters (the power spectral density (PSD)functions and the poles of the AR or ARMA method) were used to diagnose patients as diseased or normal. Results showed that normal and abnormal recordings were correctly identified in 39 of 43 cases using the new method. These results also confirm that high-frequency energy above 400 Hz is associated with coronary stenosis.*

Keywords—*Acoustic, Autoregressive spectral methods, Autoregressive moving average spectral methods, Coronary artery disease, Heart sounds, Noninvasive methods, Signal processing*

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1 Introduction

ONE-THIRD of all deaths in the world are due to coronary artery disease (KRUPP, 1982). For this reason, early detection of coronary artery disease is one of the most important areas of medical research. Although direct assessment of coronary occlusions is conclusive only using catheter angiography, this method is expensive, and has an element of risk (mortality rates range from 0·2 to 7 per cent) (KRUPP, 1982). A reliable noninvasive method is required for the early detection of coronary artery disease and for repeatedly monitoring the state of coronary occlusions before and after angioplasty.

Previous studies showed that coronary stenoses produce sounds due to the turbulent blood flow in partially occluded arteries (SEMMLOW *et al.*, 1983; AKAY *et al.*, 1988*a*; *b*; 1989; SEMMLOW *et al.*, 1990). During diastole, coronary blood flow is maximum and the sounds associated with turbulent blood flow through partially occluded coronary arteries are loudest (SEMMLOW *et al.*, 1983; AKAY *et al.*, 1990*a*; *b*). If these signals could be reliably detected, they would provide a simple, noninvasive approach to the detection of coronary artery disease.

The principal objective of this study is to improve signal-processing techniques used to identify the additional signal components found in heart sounds of patients with coronary artery disease. These added components form the basis of our approach to noninvasive detection of coronary artery disease. In earlier studies, the average power spectrum of diastolic heart sounds was estimated using traditional FFT methods (SEMMLOW *et al.*, 1983). Although some success was achieved in distinguishing normal from abnormal patients, this signal-processing technique was not pursued because it was sensitive to the effects of noise, which masks detection of the desired diastolic signal (SEMMLOW *et al.*, 1983).

Because the application of parametric modelling methods to signal identification problems results in a better estimation of spectral features, particularly for low signal-to-noise ratios (SNR), such model-based methods were employed to analyse recordings of diastolic heart sounds and to detect features associated with coronary stenosis (AKAY *et al.*, 1988*a*; *b*; 1990*a*; *b*; SEMMLOW *et al.*,

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1990). From the many model-based methods, the adaptive AR method was chosen to represent the diastolic signal source because it does not require prior knowledge of the signal characteristics and it can track changes in signal characteristics (ORFANIDIS, 1988; HAYKIN, 1986). The results showed encouraging differences between normal and diseased patients, indicating a resonance process in diseased patients (AKAY *et al.*, 1988*a*; 1990*a*; AKAY, 1990; SEMMLOW *et al.*, 1990). To support this finding, a theoretical model has been developed which shows that coronary stenoses produce well defined acoustic resonances (WANG *et al.*, 1990; TIE, 1990).

Subsequently an angioplasty study was initiated to investigate the fundamental assumptions of the acoustical approach in the detection of coronary artery disease. In a blind study, assessment based on spectral features as well as the poles and reflection coefficients of the AR and ARMA methods were correct in 21 out of 23 patients. The changes in decision parameters obtained from the angioplasty patients before and after angioplasty proved the basic acoustic concept, that coronary stenoses have an auditory correlate.

A parallel study was begun to evaluate the diagnostic capability of the acoustic approach in detecting coronary artery disease. As in the angioplasty study, all estimations were performed in a blind fashion to guard against bias. One hundred heart sound recordings obtained from 57 patients were analysed using the AR and ARMA methods (only 20 of these recordings were analysed using the ARMA method). Using the same decision parameters developed in the angioplasty study resulted in 80 out of the 100 recordings being correctly diagnosed (AKAY, 1990; AKAY *et al.*,1991; WELKOWITZ *et al.*, 1990).

Although the results of our previous studies were promising, all recordings were taken in a soundproof room. Improvements were necessary for the recording to be made at the patient's bedside (noisy room) as the AR/ARMA model of the diastolic heart sounds was found to be unstable for some patients due to excessive noise. In those cases, the poles and zeros of the ARMA method were outside the unit circle, producing errors in the spectral estimate of the diastolic heart sounds. A similar problem was reported in ARMA modelling of the electroencephalogram (EEG) (NARASIMHAN, 1989). The spectral estimates obtained after filtering the EEG were found to be far superior to the direct estimation of the ARMA spectrum (NARASIMHAN, 1989). The sensitivity of the ARMA and particularly of AR methods to observation noise has been well documented (MAKHOUL, 1975; KAY and MARPLE, 1981; TUFTS and KUMARESAN, 1982; PARK and GERHARDT, 1989; KAY, 1979; SAKAI, 1979). Thus, techniques to reduce excessive noise can be expected to improve the effectiveness of our diagnostic system, because the effect of excessive observation noise reduces the dynamic range of the estimation of the AR/ARMA power spectral density function. As some noise sources are within the body they cannot be measured and analysed independently from the desired signal.

For these reasons, we used the adaptive line enhancer (ALE) method to reduce background noise on the input signal. The modified Yule-Walker (MYW) AR and ARMA methods were then applied to the filtered signal. The MYW AR and ARMA methods were chosen because they show better performance (KAY and MARPLE, 1981; KAVEH and BRUZZONE, 1981) when the poles of the AR and ARMA models are very close to the unit circle. The analysis was carried out on 43 recordings (33 abnormal and 10 normal) taken from 34 normal and diseased patients.

2 Method

2.1 *Adaptive line enhancer (ALE) method*

The adaptive line enhancer (ALE) (WIDROW *et al.*, 1975; TREICHLER, 1979; FERRARA and WIDROW, 1981), which is a modified version of adaptive noise cancelling methods, has been widely used in many fields including biomedical signal processing (CHEN *et al.*, 1989; KENTIE *et al.*, 1981; AL-NASHASH *et al.*, 1988). A particular advantage of this method is that it does not require a reference (noise) signal. The input signal $x(n)$ provides its own reference signal, which is a delayed replica of the input. In this method the only difference between the primary and reference signals is the delay d. Assuming that the input to the ALE consists of a correlated signal $y(n)$ and an uncorrelated noise signal $v(n)$, if the delay is properly chosen, the noise in the reference signal $r(n)$ becomes uncorrelated with the primary noise signal $v(n)$. Then, the output of the adaptive line enhancer estimates the desired signal $y(n)$ after the convergence of the adaptive algorithm (FERRARA and WIDROW, 1981; ORFANIDIS, 1988).

Motivation behind choosing the delay can be explained as follow (ORFANIDIS, 1988):

$$x(n) = y(n) + v(n) \tag{1}$$

$$r(n) = y(n - d) + v(n - d) \tag{2}$$

$$E[y(n)y(n - d)] \neq 0 \tag{3}$$

$$E[v(n)v(n - d)] = 0 \tag{4}$$

where $x(n)$ represents the primary signal; $y(n)$ represents the desired signal; $r(n)$ represents the reference signal; $v(n)$ represents the primary noise signal; E represents the expectation operator; d represents the delay.

In this study, we used the adaptive lattice implementation of the line enhancement method (ORFANIDIS, 1988; AKAY *et al.*, 1990*a*). To initialise the ALE, the forward and backward estimation errors were set to 0·01. In addition, the forgetting factor λ was taken either as 1, or between 0·98 and 1·0 (AKAY *et al.*, 1990*a*; AKAY, 1990). Based on our previous findings, the diastolic heart sound signal is highly correlated and does not decay to zero even after 50 autocorrelation lags (see Fig. 3 in AKAY *et al.*, 1990*a*). Hence, we selected the delay $d = 7$ for ALE (see Fig. 3 in AKAY *et al.*, 1990*a*) because this value is large enough so that $v(n)$ in the primary signal input will be uncorrelated with $v(n - d)$ in the reference signal. Based on some initial findings (see Fig. 1 in AKAY *et al.*, 1990*a*; AKAY, 1990), the filter order $m = 10$ for ALE, which should be greater than the delay d (WIDROW *et al.*, 1975; ORFANIDIS, 1988), was chosen.

2.2 *Autoregressive (AR) method*

The AR model is the most widely used modelling method to estimate the power spectral density (PSD) function associated with some biological signals. The AR model is called the all-pole method (MAKHOUL, 1975; KAY and MARPLE, 1981). Each sample of a signal can be expressed as a linear combination of previous samples and an error signal $e(n)$. The error signal can be assumed to be independent of the previous samples (MAKHOUL, 1981).

$$y(n) = -\sum_{p=1}^{m} a_p y(n - p) + e(n) \tag{5}$$

where $y(n)$ represents the signal to be modelled; a_p represents the AR coefficients of the AR process at the pth stage; $e(n)$ represents the estimated error signal; m represents the AR model order.

The transfer function of the AR method can be calculated as follows (KAY and MARPLE, 1981):

$$H(z) = \frac{1}{A(z)} \tag{6}$$

where $A(z)$ represents filter function.

The PSD of the AR method $S_{AR}(\omega)$ can be calculated as follows (KAY and MARPLE, 1981):

$$S_{AR}(\omega) = \frac{\sigma_e^2}{\left[1 + \sum_{p=1}^{m} a_p \exp(-j\omega_p \Delta t)\right]^2} \tag{7}$$

where σ_e^2 is the noise power and is assumed to be constant; ω is the frequency; Δt is the sampling interval.

As σ_e^2 is a constant, the only values that are needed for calculating the shape of the PSD function are the so-called prediction coefficients a_p (ORFANIDIS, 1988; HAYKIN, 1986; KAY and MARPLE, 1981).

The estimation of the prediction coefficients a_p can be carried out either by using block processing methods such as autocorrelation, covariance and Burg (maximum entropy) methods, or adaptive processing methods such as least mean square (LMS), conventional recursive least square (RLS) and gradient adaptive lattice methods (GAL) (KAY and MARPLE, 1981; ORFANIDIS, 1988; HAYKIN, 1986).

2.3 *Autoregressive moving average (ARMA) method*

Some discrete-time random processes can be modelled using the ARMA method when the signal is corrupted by heavy observation noise. The method described is called the 'pole-zeros' method. The output sequence $y(n)$ can be modelled by assuming an input driving sequence $v(n)$ as follows (KAY and MARPLE, 1981):

$$y(n) = -\sum_{p=1}^{m} a_p y(n-p) + \sum_{p=0}^{q} b_p v(n-p) \tag{8}$$

where a_p represents the AR coefficients of the AR process at the pth stage; b_p represents the MA coefficients of the MA process at the pth stage; m represents the AR model order; q represents the MA model order.

The key point here is to separate the driving force $v(n)$ from any observation noise. The transfer function of the ARMA process $H(z)$ can be given in terms of the transfer functions of the AR and MA processes as follows:

$$H(z) = \frac{B(z)}{A(z)} \tag{9}$$

where $B(z)$ represents the transfer function of the MA portion of $H(z)$; $A(z)$ represents the transfer function of the AR portion of $H(z)$.

The coefficients of the AR and MA models can be calculated accurately and efficiently using the MYW method (KAVEH and BRUZZONE, 1981; BRUZZONE and KAVEH, 1984; FRIEDLANDER and PORAT, 1984; IZRAELEVITZ and LIM, 1983). Although the ARMA process can be modelled by maximum likelihood techniques that minimise a non-linear function, it has been shown that this is not the best method in practical applications (KAY and MARPLE, 1981). As an alternative to the maximum likelihood realisation of the ARMA technique, we chose the easily implementable MYW ARMA method (FRIEDLANDER and PORAT, 1984). The overdetermined YW ARMA method also shows better performance than the maximum likelihood realisation of the ARMA process when the poles of $A(z)$ are sufficiently close to the unit circle (KAVEH, 1979; BRUZZONE and KAVEH, 1984; FRIEDLANDER and PORAT, 1984).

The power spectral density (PSD) term $S_{ARMA}(\omega)$ obtained from the ARMA method can be calculated as follows:

$$S_{ARMA}(\omega) = \sigma_e^2\left[\frac{B(\omega)}{A(\omega)}\right] \tag{10}$$

where σ_e^2 represents the noise variance from the ARMA method.

For the initial estimation, one chooses $a(0) = b(0) = 1$.

Details of the MYW AR and ARMA methods have been described elsewhere (KAY and MARPLE, 1981; AKAY, 1990; WELKOWITZ *et al.*, 1990). A study using the AR model has shown that, for filter order m greater than 10, the predicted error power as a function of filter order was relatively stable (see Fig. 1*a*, in AKAY *et al.*, 1990*a*). Based on the data of Figs. 1*a* and 1*b* in AKAY *et al.*, (1990*a*) and our initial empirical findings, filter orders between 10 and 15 were judged sufficient to represent the signal recordings. Furthermore, our results were insensitive to the filter orders within this range and the reduction in normalised error with increased order is very small (see Fig. 1*b* in AKAY *et al.*, 1990*a*). Filter orders of $m = 10$ (the AR order) (see Fig. 1 in AKAY *et al.*, 1990*a*), $q = 3$ (the MA order), and $L = 15$ (the autocorrelation function order) were chosen as adequate to produce an estimate of the white noise process after filtering $y(n)$ with the estimated inverse transfer function of the ARMA method (BOX and PIERCE, 1970; AKAY *et al.*, 1991).

2.4 *Patient analysis*

Patients were selected from those undergoing catheterisation and/or angioplasty at the Cardiodynamics Laboratory of Robert Wood Johnson University Hospital. Diastolic heart sounds were recorded from the fourth intercostal space on the chest of patient using a specially designed high-sensitivity accelerometer (PADMANABHAN *et al.*, 1989). These sounds were recorded while the patients held their breath and were supine.

The objective of this study was to investigate the diagnostic ability of diastolic heart sounds to detect coronary artery disease noninvasively in a relatively noisy environment; hence, recordings were made at the patient's bedside. For each patient, ten cardiac cycles were digitised (sampling frequency = 4 kHz) and an average spectrum was constructed to obtain representative frequency information, as spectra obtained from individual diastolic cycles showed some slight variation. The spectra were obtained using the techniques described above. Before the analysis, the DC component from each recording was eliminated (period by period). As detailed elsewhere (SEMMLOW *et al.*, 1983), the diastolic heart sounds were passed through an antialiasing analogue filter with a cutoff frequency of 1200 Hz. A high-pass digital filter with a cutoff frequency of 350 Hz was used to reduce the large amount of low-frequency energy.

3 Results and discussion

For this study, 43 (33 abnormal and 10 normal recordings) heart sound recordings were obtained from 34 patients. For each recording, ten diastolic heart periods were isolated and analysed using the AR and ARMA methods. For the purpose of this study, patients with occlusions of less than 30 per cent were considered to be normal. All of these patients, except for four pseudonormals (symptomless subjects assumed to be normal, but not cath-proven) were cath-proven as normal or diseased.

Table 1 shows a description of the stenoses for the patients used in this study. Table 2 shows the absolute power between 400 and 800 Hz (*ap*), and the second complex conjugate pole pairs obtained with the AR and ARMA methods (*z*2) for the normal/abnormal patients.

Table 1 Abnormal patients' database

Patient ID	Condition, per cent	Number of occlusions
10202	LAD 40, 40, RCA 90, CFX 45	4
10702	LAD 90, 90, RCA 70	3
11002	LAD 40, RCA 40, CFX 40	3
11502	LAD 45, 35, RCA 45, 68, CFX 48	5
12702	LAD 95, RCA 100	2
13702	LAD 25, CFX 35, 80	3
14602	LAD 80, CFX 80	2
10204	LAD 40, 40, RCA 90, CFX 45	4
10902	LAD 90, RCA 85	2
12902	LAD 95, 50	2
13602	LAD 50, 60, 80	3
10502	LAD 75, 90, 60, RCA 100	4
12602	LAD 70, 90	2
12802	RCA 30, 50	2
13302	LAD 99, 70, RCA 100	3
13902	LAD 70	1
10502	LAD 75, 90, 60, RCA 100	4
14002	CFX 90	1
14302	LAD 95, RCA 40, CFX 65, 65	4
13502	LAD 50, CFX 90	2
11102	LAD 90	1
11602	LAD 65, RCA 100	2
13802	LAD 60, 60, RCA 100	3
10602	LAD 25, 30, RCA 15, 30	4
13604	LAD 50, 30, 80	3
14202	LAD 90, 100, RCA 80	3
10302	LAD 75, RCA 68	2
13102	LAD 75, RCA 40, 65	3
14502	CFX 70, 90	2
13004	LAD 25, 50, RCA 50	3
13504	LAD 50	1
11104	LAD 10	1
13304	LAD 100, RCA 100	2
13104	RCA 60, 45, CFX 30	3
12604	LAD 20, 20	2
11202	LAD 10	—

LAD is the left anterior decending artery
RCA is the right coronary artery
CFX is the circumflex artery

Table 2 Normal/abnormal patients' parameters

Patient ID	*ap*(AR)	*ap*(ARMA)	*z*2(AR)	*z*2(ARMA)
10202	1220·3	1156·2	0·89/∓0·29	0·88/∓0·23
10702	1326·5	—	0·89/∓0·27	—
11002	1274·9	4819·0	0·89/∓0·43	1·00/∓0·33
11502	1051·0	629·2	0·87/∓0·28	0·84/∓0·27
12702	1011·7	329·9	0·86/∓0·29	0·83/∓0·28
13702	711·2	1856·0	0·86/∓0·30	0·98/∓0·34
14602	710·6	673·8	0·86/∓0·32	0·84/∓0·30
10204	899·0	1221·0	0·85/∓0·31	0·86/∓0·32
10902	496·3	754·8	0·85/∓0·35	0·84/∓0·34
12902	696·2	662·0	0·85/∓0·31	0·84/∓0·30
13602	776·9	607·3	0·85/∓0·33	0·82/∓0·33
10504	612·0	1766·6	0·84/∓0·34	0·88/∓0·35
12602	512·4	598·6	0·84/∓0·32	0·83/∓0·33
12802	444·0	1520·2	0·84/∓0·34	0·91/∓0·35
13302	343·2	491·0	0·83/∓0·33	0·83/∓0·33
13902	432·8	361·7	0·83/∓0·40	0·80/∓0·41
10502	552·8	—	0·82/∓0·37	—
14002	342·5	331·2	0·82/∓0·41	0·81/∓0·42
14302	276·4	312·7	0·82/∓0·28	0·80/∓0·27
13502	356·0	455·8	0·82/∓0·26	0·80/∓0·26
11102	411·8	705·8	0·81/∓0·36	0·85/∓0·37
11602	237·1	216·2	0·80/∓0·25	0·78/∓0·27
13802	312·6	267·2	0·80/∓0·31	0·80/∓0·32
10602	410·0	1023·3	0·80/∓0·30	0·86/∓0·30
12502*†	423·2	231·0	0·80/∓0·37	0·76/∓0·37
13604	299·2	189·0	0·78/∓0·30	0·70/∓0·29
14202	197·2	212·8	0·78/∓0·32	0·77/∓0·32
10302	256·9	338·5	0·77/∓0·31	0·79/∓0·33
13102	173·3	200·1	0·76/∓0·33	0·75/∓0·33
14502	192·5	188·2	0·76/∓0·34	0·73/∓0·32
13004	136·3	89·3	0·73/∓0·27	0·62/∓0·30
13504	127·0	412·3	0·72/∓0·33	0·76/∓0·27
10402*	175·3	183·2	0·72/∓0·36	0·71/∓0·32
11104*	124·3	122·0	0·71/∓0·36	0·68/∓0·32
14702*	154·0	112·9	0·71/∓0·34	0·70/∓0·33
13304	75·0	113·0	0·69/∓0·31	0·70/∓0·30
14004*	64·2	318·5	0·69/∓0·32	0·76/∓0·34
12302*†	90·8	88·8	0·68/∓0·35	0·62/∓0·37
13104	82·0	100·2	0·67/∓0·23	0·72/∓0·27
12102*†	47·3	412·8	0·62/∓0·35	0·74/∓0·42
12604*	23·3	31·1	0·58/∓0·32	0·56/∓0·32
12002*	38·7	41·0	0·50/∓0·22	0·55/∓0·23
11202*	22·6	—	0·48/∓0·31	—

ap is the absolute area between 400 and 800 Hz (units of pressure, Hz)
*z*2 is the second complex pair of poles from the ARMA method
* normal patients
† pseudonormal patients

Fig. 1 shows a typical time recording of isolated diastolic heart sounds taken from a normal patient (14004) before (upper curve) and after (low curve) ALE filtering. The PSD functions obtained from the AR and ARMA models applied to the isolated heart sounds of a normal patient (12604) as well as an abnormal patient (12602) are shown in Figs. 2 and 3, respectively. These spectra were obtained using ALE filtering and a clear difference is seen in the energy content between 400 and 800 Hz.

The effect of ALE filtering is demonstrated in Figs. 4–7. Figs. 4 and 5 show the PSD functions obtained from the AR model applied to the diastolic heart sounds of an abnormal patient (14002) and normal patient (14004), with and without adaptive filtering. Figs. 6 and 7 show the PSD function obtained from the ARMA model for the same patients. Figs. 4–7 show that the level of noise between 400 and 800 Hz was substantially reduced after adaptive filtering. Yet, despite the reduction in noise at these frequencies, the filtered spectra of diseased patients clearly display a resonance peak around 600 Hz, as shown in Figs. 4 and 6. Note that this peak is not seen in the filtered data of normal subjects, as shown in Figs. 5 and 7.

An alternative decision criterion used an estimation of the poles obtained with the AR and ARMA method for the normal/abnormal patient groups (AKAY, 1990). In all patients, large frequency peaks were found at either end of the effective spectra shaped by a combination of microphone characteristics, analogue filtering and low-frequency energy found in both diseased and normal subjects. The source of the low-frequency energy has yet to be determined. At the high-frequency end of the spectra, the peak is created by transducer resonance. This specially designed transducer has a relatively low resonant frequency to maximise sensitivity over the region of interest (200–800 Hz) (PADMANABHAN *et al.*, 1989). These two frequency peaks gave rise to two complex conjugate pole pairs falling close to the unit circle, which were not considered relevant as decision criteria as they are nearly the same in all patients. However, the second complex conjugate pole pair falls closer to the unit circle for patients with coronary artery disease compared with normal patients, as shown in Table 2. This indicates that the diastolic heart sounds of diseased patients contain more energy between 400 and 800 Hz than those of normal patients.

All these findings are in agreement with the theoretical studies which showed that coronary occlusions produce

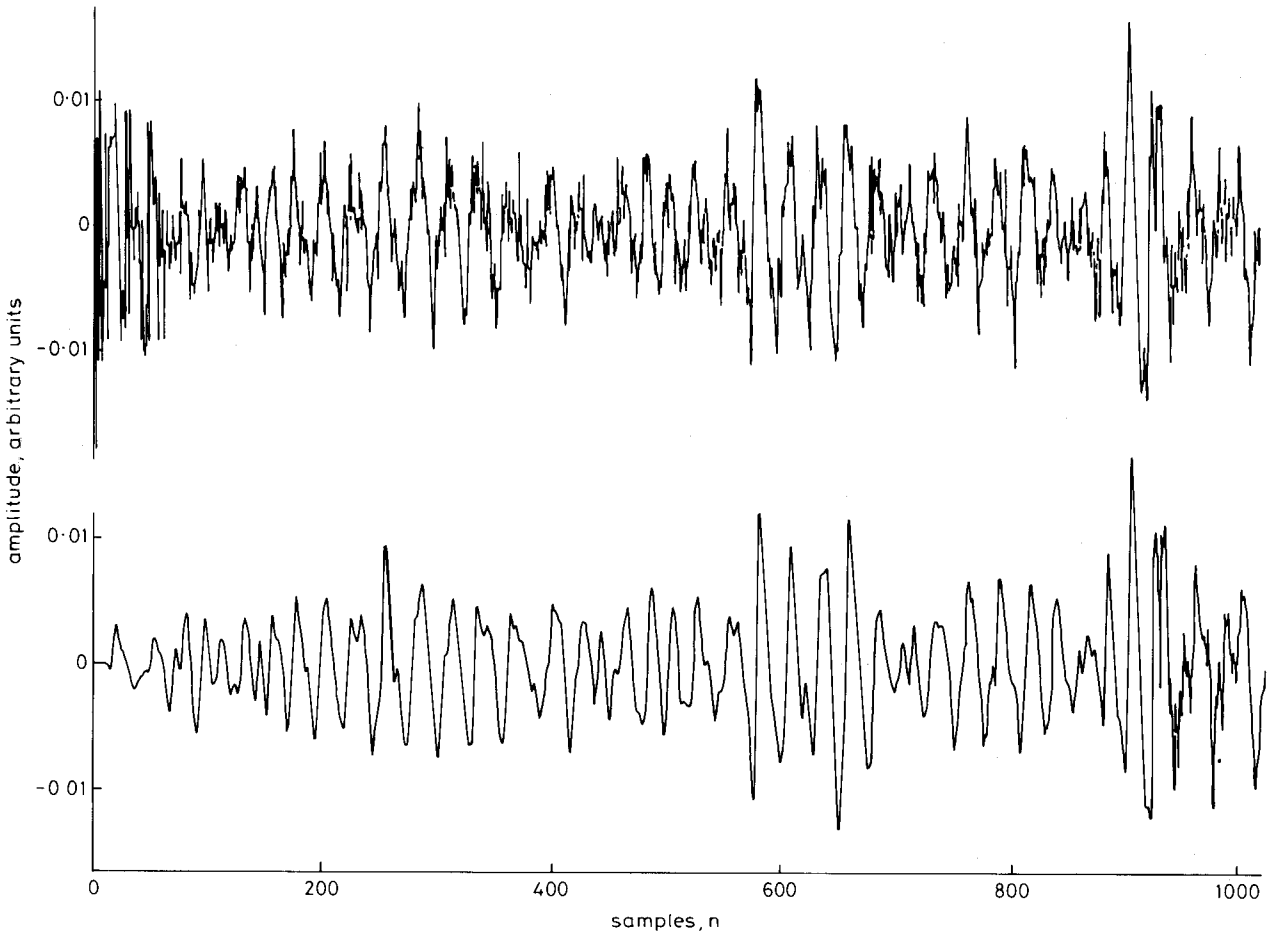

Fig. 1 *Upper curve: diastolic heart sounds recorded during a single period at the patient's bedside. Lower curve: filtered diastolic heart sound signal (ALE output)*

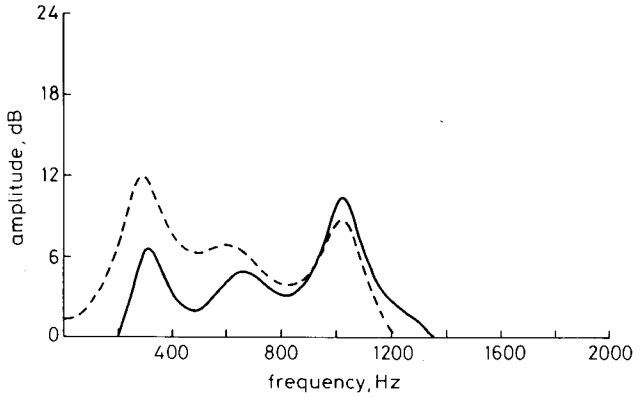

Fig. 2 *PSD function obtained from the AR and ARMA models applied to the isolated diastolic heart sounds of CAD patient 12602 after adaptive line enhancement. Solid line: AR spectrum; broken line: ARMA spectrum*

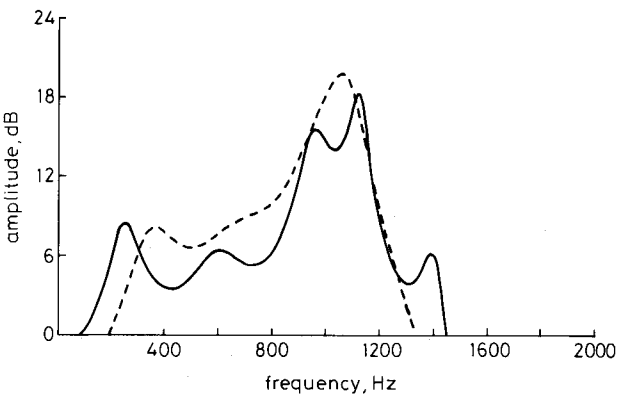

Fig. 4 *PSD function obtained from the AR model applied to the isolated diastolic heart sounds of CAD patient 14002. Solid line: after ALE; broken line: before ALE*

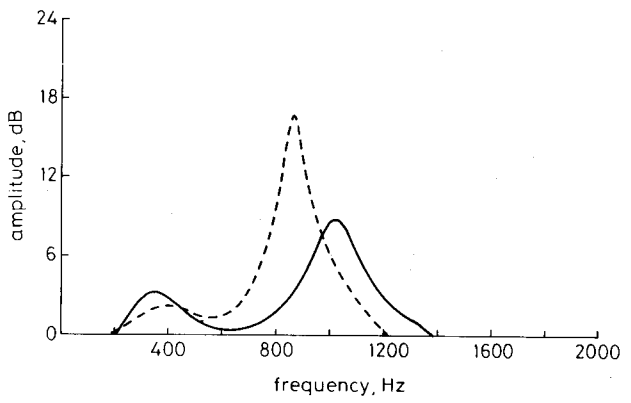

Fig. 3 *PSD function obtained from the AR and ARMA models applied to the isolated diastolic heart sounds of normal patient 12604 after adaptive line enhancement. Solid line: AR spectrum; broken line: ARMA spectrum*

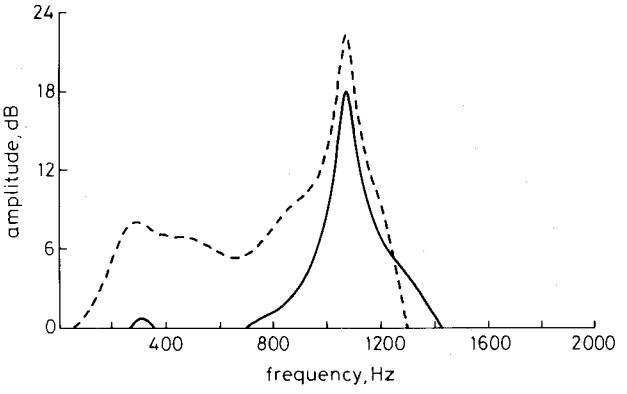

Fig. 5 *PSD function obtained from the AR model applied to the isolated diastolic heart sounds of normal patient 14004. Solid line: after ALE; broken line: before ALE*

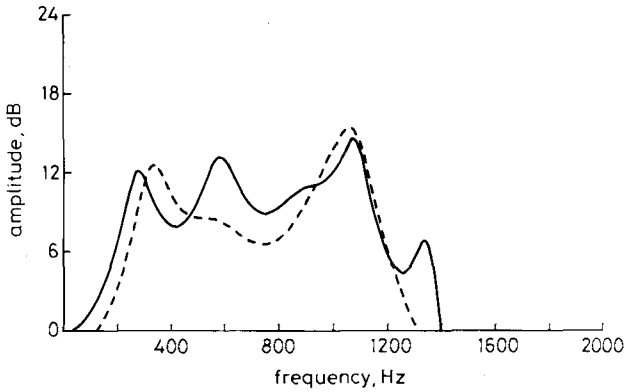

Fig. 6 *PSD function obtained from the ARMA model applied to the diastolic heart sounds of CAD patient 14002. Solid line: after ALE; broken line: before ALE*

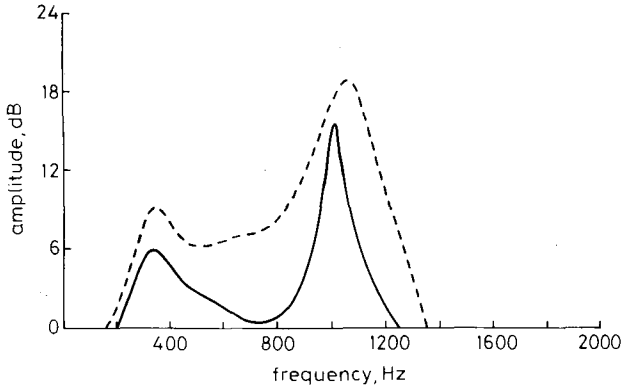

Fig. 7 *PSD function obtained from the ARMA model applied to the isolated heart sounds of normal patient 14004. Solid line: after ALE; broken line: before ALE*

resonances with frequencies ranging between 200 and 1200 Hz (WANG *et al.*, 1990; TIE, 1990).

Before differentiating between diseased and normal patients, a *t*-test (to test means) (KENDALL and STUART, 1977) was employed to determine whether the decision parameters have the same mean and whether the differences in the means were statistically significant. Although both the spectral peak parameter *ap*, as well as the pole parameter and *z*, showed significance values (*Q*-values in Table 3) less than 0·001, the parameter *z* was most significant. In addition to the Student's *t*-test, the Kolmogorov-Smirnov (K-S) test was applied to the decision parameters *ap* and *z*. Table 3 shows the K-S distance *D* between the cumulative distribution functions of normal/diseased patient decision parameters and the related significance level *S* which should be less than or equal to 0·01. All decision parameters were found to be significant in differentiating diseased patients from normal patients. Finally, the first two moments of the decision parameters were calculated. Table 4 shows that these moments were considerably different for normal and diseased patients.

The diagnostic effectiveness of the decision parameters

Table 3 T, K-S test results of normal/abnormal decision parameters

Parameters	*T*-value	*Q*	*D*	*S*
ap(AR)	5·48	0·0001	0·748	0·0003
z(AR)	4·87	0·0001	0·809	0·0001
ap(ARMA)	3·35	0·0001	0·569	0·0021
z(ARMA)	4·51	0·0001	0·774	0·0004

z is the second pole pair among the second and third pole pair
T-value shows Student's *t*-test
Q shows the significance of the *t*-test
D shows the K-S distance between two group cumulative distributions
S shows the significance of K-S test

Table 4 First two moments of normal/abnormal patient parameters

Parameters	Condition	Mean	Variance
ap(AR)	CAD	513·86	125 230·2
	normal	116·36	14 525·55
z(AR)	CAD	0·814	0·00313
	normal	0·649	0·00109
ap(ARMA)	CAD	729·12	802 096·1
	normal	171·25	16 622·15
z(ARMA)	CAD	0·812	0·00618
	normal	0·675	0·0066

was estimated by constructing curves of sensitivity against specificity for these parameters. Fig. 5 shows curves constructed from only the parameter *z* obtained with the AR model (after ALE), because the AR and ARMA methods showed essentially the same diagnostic performance. Each point on the curve represents the combination of true positives against false negatives estimated for a given threshold value of the parameter. It is obvious from this figure that this curve provides a good basis for determining the usefulness of a decision parameter. For example, in Fig. 8 selecting a threshold of 0·72 for the amplitude of *z*2 leads to a sensitivity of 93 per cent and a specificity of 90 per cent. Using this threshold value, three of 30 abnormal and one of 10 normal subjects were incorrectly diagnosed.

The incorrectly diagnosed normal patient 12502 was, in fact, a pseudonormal assumed to be normal. Considering the incorrectly diagnosed diseased patients, 13304 had two 100 per cent LAD occlusions. These blockages would permit negligible blood flow and thus can be expected to appear normal. There was no clear trend among the other two misdiagnosed patients, 13504 and 13104, except that both were postangioplasty patients.

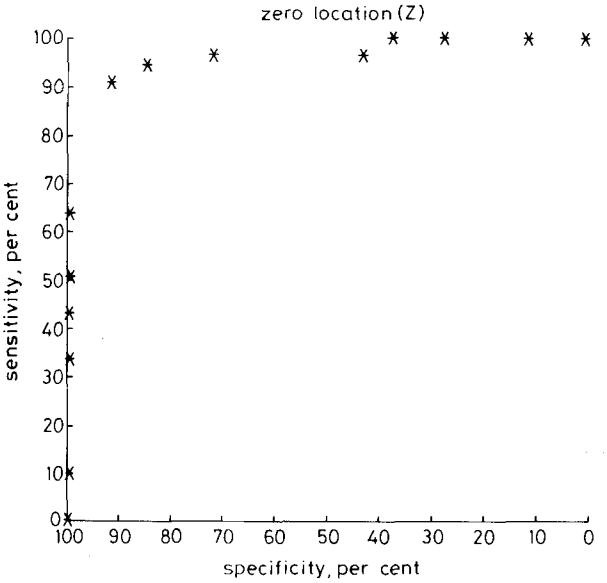

Fig. 8 *Diagnostic performance of parameter z showing sensitivity as a function of specificity*

None of the patients had aortic regurgitation, mitral stenosis or other audible diastolic murmurs. Further work involving the development of additional signal-processing techniques will be necessary to overcome these problems.

These results compare quite favourably with other noninvasive methods for detecting coronary artery disease. For example, the sensitivity of the cardiointegram (CIG) technique developed by TEICHHOLZ *et al.* (1984) was found to be 73 per cent with a specificity of 78 per cent. This approach was considered to be a moderately useful noninvasive method to detect coronary artery disease. Although the sensitivity of the thalium stress test is 83 per cent, and its specificity is 90 per cent, it is costly and takes time

(JOHNSON, 1985). However, our approach as described above is based on measurements associated with turbulence, which is closely related to stenosis, and not on symptoms as with other noninvasive approaches. Thus, it is likely that our approach will be able to detect coronary occlusions before they become large enough to induce symptoms, allowing considerably more flexibility in treatment.

4 Conclusion

In this study, the ALE filtering method was to reduce background noise from diastolic heart sounds recorded in a relatively noisy room (patient's bedside). The performance of filtering method was evaluated using AR and ARMA modelling of the filtered sounds, and both methods showed essentially the same diagnostic performance. Results obtained when comparing groups showed that the spectral energy distribution differed markedly between normal and diseased patients with the energy between 400 and 800 Hz being greater for diseased patients. For normal subjects, it was found that the second poles of the AR and ARMA methods were farther from the unit circle than those of diseased patients. The ALE filter was shown to improve this differentiation with either spectral method. The curve of sensitivity against specificity using the AR second poles as a decision criterion shows that this method can be used successfully to noninvasively detect coronary artery disease. The assessments based on the PSD function parameters and poles of the AR and ARMA methods correctly identified the diagnostic state of 39 out of 43 patients.

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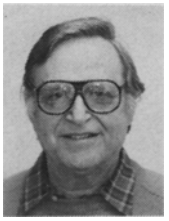
Metin Akay was born in Sivas, Turkey. He received the BS and MS degrees in Electrical Engineering from Bogazici University, Istanbul, Turkey, in 1981 and 1984. From 1984 to 1986 he continued in the Ph.D. programme at Bogazici University. In 1986 he joined the Biomedical Engineering Department at Rutgers University, where he held a fellowship and was a teaching assistant. He received his Ph.D. from Rutgers University in 1990. He is currently a Visiting Assistant Professor at Rutgers University. His areas of interest are adaptive signal processing, detection and estimation theory and their application to biomedical signals.



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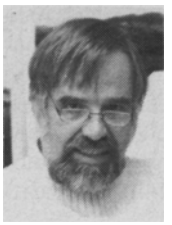

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Erratum

Medical & Biological Engineering & Computing, Vol. 29, No. 5, September 1991, 548–553 and Vol. 29 No. 6, November 1991, 629–633.

'Programmable implantable device for investigating the adaptive response of skeletal muscle to chronic electrical stimulation' by L. Callewaert, B. Puers, W. Sansen, J. C. Jarvis and S. Salmons

The names of J. C. Jarvis and S. Salmons were omitted from the contents list on the front cover of the September 1991 issue and from the author index in the November 1991 issue.

The correct contents entry should have been:

Communication

Programmable implantable device for investigating the adaptive response of skeletal muscle to chronic electrical stimulation: L. Callewaert, B. Puers, W. Sansen, J. C. Jarvis and S. Salmons **548**

The full entry in the author index should have been:

Callewaert, L., Puers, B., Sansen, W., Jarvis, J. C. and Salmons, S. Programmable implantable device for investigating the adaptive response of skeletal muscle to chronic electrical stimulation (Communication) **548–553**

The following two entries should also have been included:

Jarvis, J. C. (see **Callewaert, L., 548–553**)
Salmons, S. (see **Callewaert, L., 548–553**)