

The Analysis of Respiratory Sinus Arrhythmia Using Spectral Analysis and Digital Filtering

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Abstract—Respiratory sinus arrhythmia is the phenomenon by which respiration modulates heart rate in normal humans and in many animals. This investigation was divided into the following three categories: 1) the development of a mathematical model relating respiration to those variations that it causes in heart rate; 2) the use of digital filtering techniques to attenuate fluctuations in heart rate which are due to respiration; and 3) the development of methods that use only heart rate to get information about respiration.

A linear model is used to approximate the relationship between lung volume and heart rate during normal breathing. Segments of controlled-rate normal breathing are analyzed using spectral analysis techniques in order to compute appropriate model parameters for a given subject. Simulations are implemented using the fast Fourier transform, whereby the model accepts normal respiration as its input and develops an instantaneous heart-rate function that is compared with the actual heart rate from a subject.

Based on the spectral content of heart rate relative to respiration during normal breathing, band-stop digital filters are developed and used to process heart-rate data in such a manner that its respiratory arrhythmias are attenuated relative to most nonrespiratory arrhythmias. This filtering is accomplished without the aid of respiratory information. Both recursive and fast Fourier transform digital filtering are utilized. Typical plots of heart-rate and respiration spectral densities and time functions are presented to demonstrate the results.

I. INTRODUCTION

OBSERVATIONS and studies relating to respiration-linked variations of heart rate in man and in some animals have appeared in the literature as far back as 1865, and the topic has continued to be an item of study to the present day [1]–[10]. There has been a great deal of conjecture concerning the reasons that heart rate is modulated by respiration. Three primary mechanisms have been used by most researchers to explain the phenomena: 1) a central nervous system control by direct communication between the respiratory center and the heart-rate centers; 2) an indirect control through blood pressure, which is itself modulated by respiration; 3) a reflex control in response to stretch receptors in the lungs and chest. The latter mechanism has gained large support for causing the most evident and immediate fluctuations in heart rate, but blood pressure control is also thought to play a role,

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perhaps in a secondary or transient manner. Furthermore, as the result of experiments where respiration has been induced without significant lung movement, there is evidence to support the central nervous system control. It would be presumptuous to exclude from a physiological model any of these effects. There may also be other influences linking respiration to heart rate. It is not the purpose of this study to investigate the specific physiological causes of the phenomena, but to investigate the practical problems of mathematically relating respiration to heart rate.

The course of the investigation has been inspired by the desire to see progress made in the following three areas: 1) simulation of human heart rate under various environmental conditions; 2) the ability to distinguish between heart-rate fluctuations that are of respiratory origin and those that are due to other effects; and 3) the capability to derive information about respiration solely by observation of heart rate.

II. BACKGROUND FOR STATISTICAL MODEL

It has long been observed that plots of lung volume and the corresponding instantaneous heart rate have much in common in their frequency of fluctuation. This suggested that knowledge of the rate of breathing over a given time interval would provide much information about the corresponding heart-rate fluctuation around the mean value. Spectral analysis calculations for data obtained from subjects breathing normally indicated definite peaks in both respiration and heart rate near the normal respiration rate of 6 or 7 cycles/min.

The next goal was to compare the power spectral estimates of a heart-rate signal with that of the constant-rate respiration function modulating it. No special respiratory waveforms were sought—just constant-rate breathing for specified lengths of time at the various rates.

Experimental Data

All of the data for the study were provided by the Biomedical Engineering Branch of the USAF School of Aerospace Medicine. Data were provided in various forms on digital and analog magnetic tape reels, on oscillographic recording paper, and on digital computer cards. The instantaneous heart rate was derived from an electrocardiogram (EKG) signal by detecting the time interval between each succeeding *R*-wave peak. The reciprocal of this time interval represents instantaneous heart rate. Specifically, this rate was associated

with the second of the two *R*-wave peaks denoting the interval.

To measure lung volume the subject breathes into a spirometer which produces an analog voltage proportional to lung volume. This voltage is sampled at 0.25 s intervals during the digitizing process. All experimental data were taken while the subject was in the supine position with electrodes placed on both ends of the sternum (breast bone).

III. INITIAL DATA PROCESSING

The raw heart-rate values occur at irregular intervals, whereas the analysis techniques used herein need equal interval samples of the heart-rate function. This was achieved by fitting the experimental data with a p th-order polynomial through each $p+1$ succeeding heart-rate values, and then sampling this polynomial at equal intervals of time. Both $p=2$ and $p=1$ gave good results, and the latter, which is linear interpolation, is simpler and faster.

Even though respiration samples in raw form were at equal intervals, linear interpolation was used to enable the respiration samples to coincide with the equal-interval heart-rate samples. This procedure also facilitates the use of an arbitrary sampling rate, which is necessary for the convenient use of the fast Fourier transforms (FFT) in processing fixed-length data samples where it is desirable to make the total number of data samples equal to 2 taken to some power.

The terminology "frequency content" may need clarification. The word "frequency" here refers to the fluctuations of the heart-rate signal and not to its instantaneous heart-rate value. For a constant heart-rate value of 70 beats/min with no fluctuations, its energy content would be concentrated at zero frequency. Respiration causes fluctuations in heart rate, thereby inducing a power density spectrum with energy at non-zero frequency values. The instantaneous heart-rate values will be referred to in units of *beats/min*, and the frequency of fluctuation of this heart-rate signal will be described in units of *cycles/min*.

Spectral Density Calculations

Both the conventional and FFT methods of computing spectral estimates are developed on pages 11–47 of [11]. Also defined and developed therein are compensation for data truncation effects, effect of time domain sampling on the frequency response, and resolution confidence limits and degrees of freedom for the spectral estimates.

Fig. 1 shows the auto spectral densities for a given subject at the controlled breathing rate of 6 cycles/min. Note the harmonic content of the respiratory signal at multiples of the fundamental; peaks in heart-rate energy content occur at corresponding frequencies.

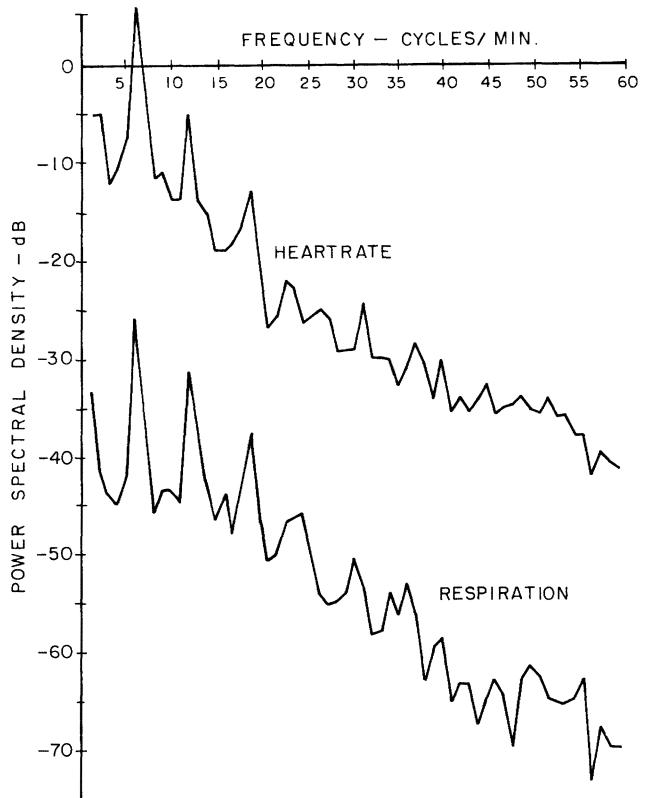


Fig. 1. Spectral densities for 6 cycles/min rate breathing.

IV. A LINEAR MODEL OF RESPIRATORY SINUS ARRHYTHMIA

It can be shown by experimental observations that a nonlinear model is necessary to depict heart rate when special or abnormal respiratory waveforms, such as steps or impulses, are created by a subject in the laboratory. Such strained maneuvers do cause a phenomena having nonlinear characteristics. Typically, most physical systems exhibit a nonlinearity, given the right stimulus, but even these nonlinear systems can be analyzed by quasi-linearization techniques for certain classes of inputs. Thus we seek a linear model for respiratory sinus arrhythmia under the condition of normal breathing. It is anticipated that the linear model will yield valid and useful results for the comparatively mild respiratory maneuvers used by humans most of the time.

Consider the linear system with the input-output relationship shown in Fig. 2. Assume the system can be represented by a transfer function having approximately constant coefficients for the time interval of observation. If the noise input shown were zero, then the transfer function could be found by several conventional methods; e.g., impulse response, $H(j\omega)/R(j\omega)$, etc. [$H(j\omega)$ and $R(j\omega)$ are Fourier transforms of $h(t)$ and $r(t)$, respectively.] However, when $n(t)$ exists these basic methods will include its effect in the measured outputs, and consequently the true nature of the system will not be reflected. Furthermore, it is not always

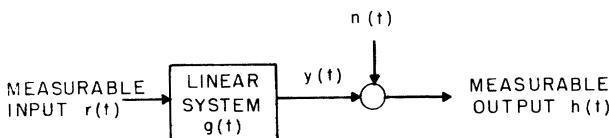


Fig. 2. Block diagram of an example system.

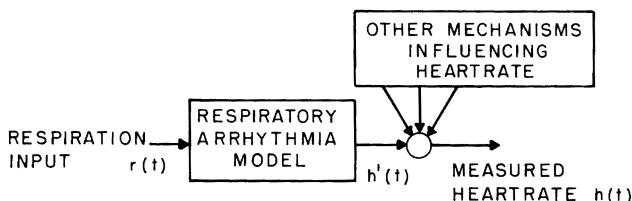


Fig. 3. Block diagram of heart rate influences.

possible to meaningfully stimulate a given physical system with any desired input (such as an impulse or step), as may be the case for the system under study.

Now define a transfer function between $H(j\omega)$ and $R(j\omega)$ as

$$G_{rh}(j\omega) = H(j\omega)/R(j\omega) \quad (1a)$$

and multiply the numerator and denominator of the right-hand side of this equation by $R^*(j\omega)$, the complex conjugate of the Fourier transformed input $r(t)$. The result is

$$G_{rh}(j\omega) = \frac{H(j\omega)R^*(j\omega)}{R(j\omega)R^*(j\omega)} = \frac{P_{rh}^*(j\omega)}{P_{rr}(\omega)} \quad (1b)$$

where $P_{rh}^*(j\omega)$ is the complex conjugate of the cross power between $r(t)$ and $h(t)$, and $P_{rr}(\omega)$ is the auto power of $r(t)$. Assuming that $n(t)$ and $r(t)$ are uncorrelated, then $P_{rh}^*(j\omega)$ is not influenced by the presence of $n(t)$, and since $P_{rr}(\omega)$ does not involve $n(t)$ at all, this estimate of $G_{rh}(j\omega)$ from $h(t)$ and $r(t)$ becomes a good method of overcoming the presence of noise in order to estimate Y/R as specified in Fig. 2.

Now look at Fig. 3. Ideally, the heart-rate signal to record for determining the respiratory arrhythmia model is $h'(t)$ as shown. However, in spite of efforts to take data from subjects whose condition is such that the "other mechanisms influencing heart rate" have a minimal effect (e.g., resting unexcited subjects), some unknown amount of the fluctuations of $h(t)$ does take place because of these nonrespiratory contributions. This is the main reason that (1b) is applicable to these modeling efforts.

Ideally, the input $r(t)$ would be white noise across the response bandwidth of $G(j\omega)$. The auto spectral density of such an input would be constant, and therefore the value of $G(j\omega)$ would be proportional to $P_{rh}^*(j\omega)$. Enough data could be taken in one relatively short run to identify adequately the system parameters. Since a subject cannot create a respiration signal of this nature

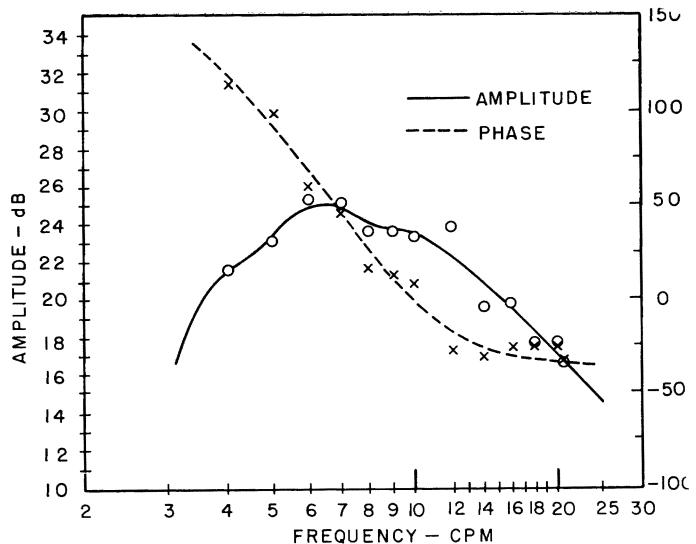


Fig. 4. Frequency response for subject F (16–18 degrees of freedom). Right-hand scale indicates phase in degrees.

while breathing in a relatively normal manner, if at all, it becomes necessary to stimulate the system in some other way. Herein is the basis for the constant-rate respiration data, insofar as their application to modeling efforts is concerned.

The procedure is straightforward. In order to get usable data for identification purposes while driving the system as nearly as possible like it is stimulated during normal breathing, the subject breathes at comfortable depths at constant rates for (ideally) ten minute intervals. The slowest that a subject can breathe for an extended period approaching ten minutes (breathing into the spirometer) is about 3 or 4 breaths/min, and the upper limit seems to be about 20 breaths/min; hyperventilation symptoms cause this upper bound. If the system is driven at 4 cycles/min, and the appropriate spectral densities are computed [see (1)], $G(j\omega)$ for $\omega=2\pi(4)$ rad/min can be found. The process can be repeated for 5 cycles/min and so forth on up to the maximum respiration rate possible. Each such computation determines a set of points on an amplitude and phase plot for $G(j\omega)$. The number of frequencies at which one can thereby specify amplitude and phase for $G(j\omega)$ is at least equal to the number of different sets of constant-rate respiration data that are available for the given subject.

It should be noted that unless the system is adequately driven at a certain frequency, then the value of $G(j\omega)$ computed at this frequency tends to be almost meaningless. This is because the power spectral estimates used for computing $G(j\omega)$ would in this case be near noise levels. Confidence limits for the magnitude and phase estimates for the model must be considered.

A typical frequency response for a given subject is shown in Fig. 4. The identification technique itself was

TABLE I
THE EFFECT OF NOISE FOR 5-CYCLES/MIN RATE BREATHING

SNR	$ G(j\omega) $	Phase (degrees)	Coherence
∞	15.1	82.0	0.965
10	15.3	80.0	0.949
5	15.4	79.2	0.938
2	15.6	77.7	0.912
1	15.8	75.9	0.876

tested insofar as its rejection of nonrespiratory noise in the measured heart rate by deliberately injecting noise into the heart rate before computing the required cross-spectral density between respiration and heart rate (see Fig. 3). White noise having a Gaussian distribution was injected in known ratios of heart-rate variance to noise variance. For ten minutes of 5 cycles/min rate breathing data, Table I summarizes the results of these calculations as a function of signal-to-(injected)-noise ratio (SNR), or original heart-rate variance to injected-noise variance. Note that even for SNR = 1, the computed amplitude and phase compared quite favorably with those for no injected noise (SNR = ∞).

The table also shows the coherence between respiration and heart rate for each SNR. Coherence is defined as

$$\text{Coh } (f) = \frac{|P_{rh}(f)|}{\sqrt{P_{rr}(f)P_{hh}(f)}} \quad (2)$$

where $P_{rh}(f)$ is the cross-spectral density between respiration and heart rate, while $P_{rr}(f)$ and $P_{hh}(f)$ are the auto-spectral densities of respiration and heart rate, respectively. For further discussion of coherence, confidence limits, and degrees of freedom, see pages 60–64 of [11].

V. SIMULATION USING THE AMPLITUDE AND PHASE RESPONSE

The next step is to use this information in a simulation effort. One approach might be to use a Bode plot of the derived frequency response in order to determine an approximate lumped-parameter transfer function which best represents the response. The resulting transfer function could then be set up on an analog or digital (using MIMIC) computer for use with a recorded respiration signal in order to derive a simulated heart rate. This approach was not taken because of its approximate nature. That is, the method is one which is not always straightforward, especially when it comes to getting transfer functions which adequately track both amplitude and phase.

The procedure used to process data through the derived model is mathematically rather basic and is made computationally feasible because of the availability of the FFT. Simply stated, the method consists of transforming an entire time sample of respiration into frequency space, multiplying it by the derived transfer

function for the system, and inverse transforming the result back to the time domain to get heart rate. In essence, the process is digital convolution of $r(t)*g(t)$ implemented by going to the frequency domain and multiplying.

Using a typical set of data, values of amplitude and phase (or their real and imaginary parts) for the derived frequency response are available from about 4 to 20 cycles/min. By extrapolating the amplitude response beyond these edges, zeros usually occur at about 2 and 30 cycles/min; the phase response is also extrapolated as necessary out to these frequency values. Since the amplitude response is relatively small below 4 cycles/min and above 20 cycles/min, these extrapolations have a rather small effect on the output heart rate.

When the FFT is used to transform a time domain function to frequency space, N samples of the time function are used as input, where $N = 2^k$, and k is an integer. If the length of data is T , and the sampling interval is Δt , then Fourier components exist for frequencies of $0, 1/T, 2/T, \dots, N/2T$, so that a total of $N/2+1$ components results. Note that $\Delta t = T/N$, meaning that the maximum frequency just specified is $1/(2\Delta t)$, the Nyquist frequency. In order that corresponding frequency components of $R(j\omega)$ and $G(j\omega)$ can be multiplied, $G(j\omega)$ must be specified at these appropriate frequency intervals. This is done by using linear interpolation between the computed values of $G(j\omega)$.

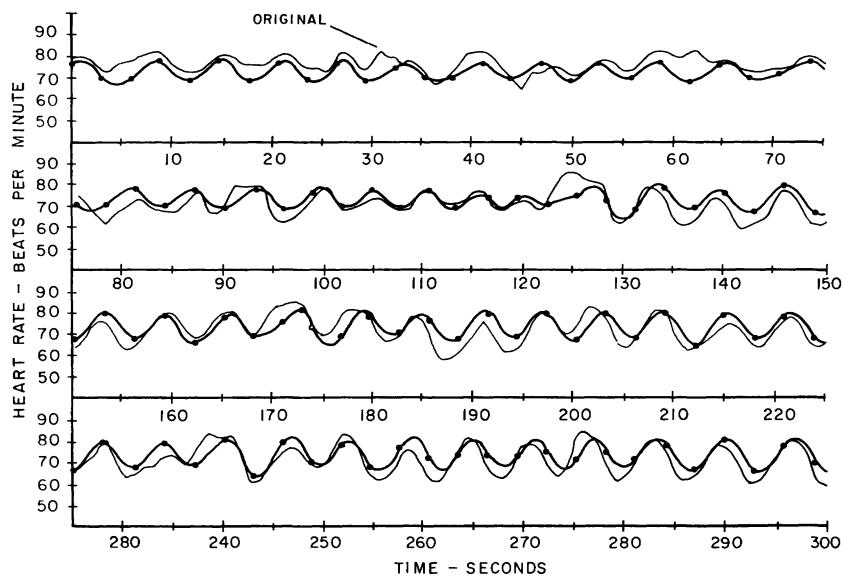
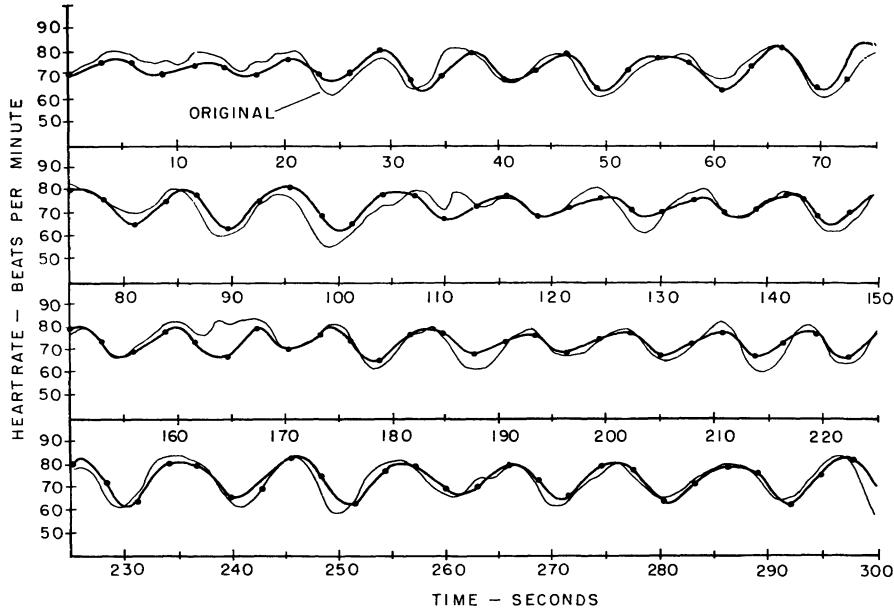
The FFT algorithm utilized has the capability to be used either to take a Fourier transform or to find the inverse of a given Fourier transform. An explanation of the mechanics of using the algorithm in either direction is given in Appendix 2 of [11]. Once the values of $H(j\omega) = R(j\omega) G(j\omega)$ have been computed at the appropriate frequency intervals, the inverse FFT is used to get back to an $h(t)$, which is the desired result. This function can be compared with the actual heart rate.

Obviously, since the value of $G(j\omega)$ is zero for $\omega=0$, the dc value of the derived $h(t)$ is zero. This model makes no attempt to relate the mean value of heart rate to respiration, so that, in order to compare the derived and measured values of heart rate, the mean of the measured heart rate is removed. For display the mean value of the simulated heart rate is made equal to the computed mean of the measured heart rate.

Experimental data taken with no restrictions placed on depth or rate of respiration were used to test the capability of the derived model parameters to simulate accurately actual heart-rate fluctuations during normal breathing. Figs. 5 and 6 show examples of such simulation efforts. Each figure shows five minutes of derived heart rate along with the heart rate actually measured during this interval.

The rms error that was specified for each simulation was computed using

$$\sqrt{\sum_{i=1}^N (h_i - s_i)^2 / N}$$

Fig. 5. Heart rate simulations for subject *F*.Fig. 6. Heart rate simulations for subject *C* with *F* parameters.

where the h_i are the actual heart-rate values, and the s_i are the corresponding simulated values at the total of N sampling instants. Regardless of how small it is, the magnitude of this error is not as meaningful as the relative sizes of errors between different simulation efforts for a given subject. That is, if the model parameters are changed somewhat by smoothing (or less smoothing) or extrapolations, or if parameters for a single subject are determined from different sets of data taken at different times, or in any situation where results need to be compared, then the relative sizes of the rms errors provide one quantitative means of comparison.

In comparing the modeling capability for one subject with that for another, some normalization of this error criterion is needed which makes the comparison mean-

ingful. For example, to compare the absolute values of rms errors for subjects *D* and *E* (3.57 and 6.33, respectively) would be of doubtful value since the respiratory arrhythmias in subject *D* are much weaker than those in *E*, making the modeling capability appear better for *D*. One meaningful normalization weight that reflects the relative strength of the respiratory arrhythmias is the standard deviation of the actual heart rate. The standard deviations for the actual heart rates of subjects *C*, *D*, *E*, and *F* were 6.26, 4.09, 7.42, and 6.63, respectively, and if the previously quoted rms errors are normalized in each case by these values, the following factors result: *C*: 0.69; *D*: 0.87; *E*: 0.85; and *F*: 0.79. For the *C* data modeled with the *F* parameters, the error factor is 0.64.

Such numerical comparisons as those just quoted can serve as a common means of evaluating this particular modeling technique relative to methods developed by other studies.

VI. FILTERING HEART RATE FOR REMOVAL OF RESPIRATORY ARRHYTHMIA

The second of the three stated goals of this model was to develop some means of distinguishing between heart-rate fluctuations that are of respiratory origin and those that are due to other effects, even when respiration is unavailable from the subject. Therefore, based upon the frequency response information previously found, an appropriate filter is sought through which to pass heart rate in order to remove or at least lessen its fluctuations that are caused by respiration, hopefully leaving unaffected most classes of nonrespiratory arrhythmias. The method of doing this will now be discussed.

The results of the spectral density computations previously presented indicate that the frequency band of heart-rate fluctuations caused by normal respiration extends from about 4 cycles/min up to near 25 cycles/min in normal healthy subjects. One way to remove these fluctuations is to band-stop filter the heart rate throughout this region. In practice such a large stop-band is not needed for most normal breathing, because the heart-rate fluctuations caused by this type of breathing tend to be limited to the lower frequencies, corresponding to respiration rates of approximately 5 to 15 cycles/min.

A degree of uncertainty exists as to just how much nonrespiratory heart-rate fluctuation might be inadvertently removed by such filtering. The two main classes of nonrespiratory arrhythmias that are definitely not effected by such band-stop filtering are slow changes in the mean heart rate and rapidly occurring pathological type changes in heart rate. Fortunately, these two types of nonrespiratory arrhythmias are probably the most prevalent. Such stimuli as emotion, exercise, certain drugs, etc., cause shifts in mean heart rate to occur over some period of time, and such low-frequency behavior should definitely not be affected by any operation intended to attenuate only respiratory arrhythmias. Since it is a characteristic of some heart diseases to produce irregular heart beats from time to time (shifting from short intervals to long intervals more or less at random), a rapidly changing instantaneous heart rate results, producing high-frequency fluctuations that should also not be attenuated by a respiratory arrhythmia filter. Unfortunately, it was not possible to get actual heart-rate data with intentionally included nonrespiratory arrhythmias in order to study their spectral characteristics relative to those of respiration. It would be especially desirable if some attempts could be made to induce some nonrespiratory arrhythmias into a heart rate which might have energy in the "respiratory band" as previously described. However, as yet no one has been able to suggest even that such arrhythmias may

consistently exist.¹ With more information available about nonrespiratory arrhythmias, it may be possible to choose more optimum filter characteristics than simple band-stop types. No basis yet exists for defining such a filter in the absence of respiratory information.

For test purposes it was possible to simulate any desired arrhythmia by introducing artificial changes into measured heart-rate data. Examples of the effect that filtering has on some of these arrhythmias will be given later in this section.

Selection of a Filter

Because of the form of available heart-rate data, digital rather than analog filtering is most convenient. In fact, under most circumstances where there is a reasonable choice between the two, the present availability of high-speed digital computers makes digital filtering a desirable alternative for several reasons: 1) physical realizability problems are relatively nonexistent, allowing quite low-frequency filtering along with (if desired) zero or linear phase-shift filtering; 2) results are more accurate and are precisely repeatable; and 3) time-varying filter coefficients are easily programmed. The single most general statement that can be made in favor of digital filtering is that it can precisely implement any filtering operation that can be expressed mathematically, either functionally or graphically.

The type of filter desired for the present purpose is one with a band-reject amplitude characteristic over any preselected frequency range. Zero phase-shift filtering is desirable to compare the filtered signal point-by-point in time with the unfiltered version, as would be the case of subtracting the two in order to get an error signal showing the fluctuations that were filtered out. However, if real-time (on-line) filtering is desirable, then zero phase-shift filtering is not possible. Two methods of band-reject digital filtering, namely Butterworth and FFT filtering, were developed and are described on pages 92-119 of [11]. It is shown that the Butterworth filter can be used: 1) for on-line data processing as the heart rate of some subject is monitored; and 2) for off-line processing to produce zero phase-shift output. However, the FFT method is better for use during off-line filtering, because it provides more ideal realizations of desired filter responses. The FFT method assumes, of course, that a digital computer is available that can handle the FFT operations in a reasonable length of time. Actual data processing using the Butterworth recursive filter is rather simple and fast on most any general purpose digital computer.

Initial Conditions

If filtering calculations are begun by letting previous inputs and outputs be zero, and if the first input is some value such as 80 beats/min, then in effect one puts a large amplitude (relative to the normal heart-rate

¹ See Appendix 1 of [11] for possible evidence of 6-7 cycles/min nonrespiratory arrhythmias.

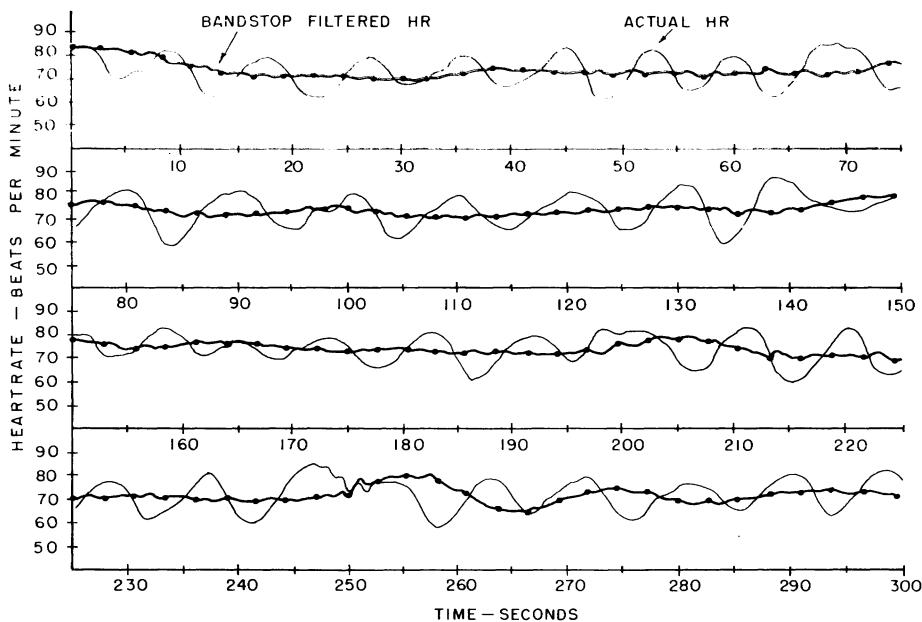


Fig. 7. Heart rate filtering using a fourth-order Butterworth digital filter with a 4-16 cycles/min stopband (nonzero phase).

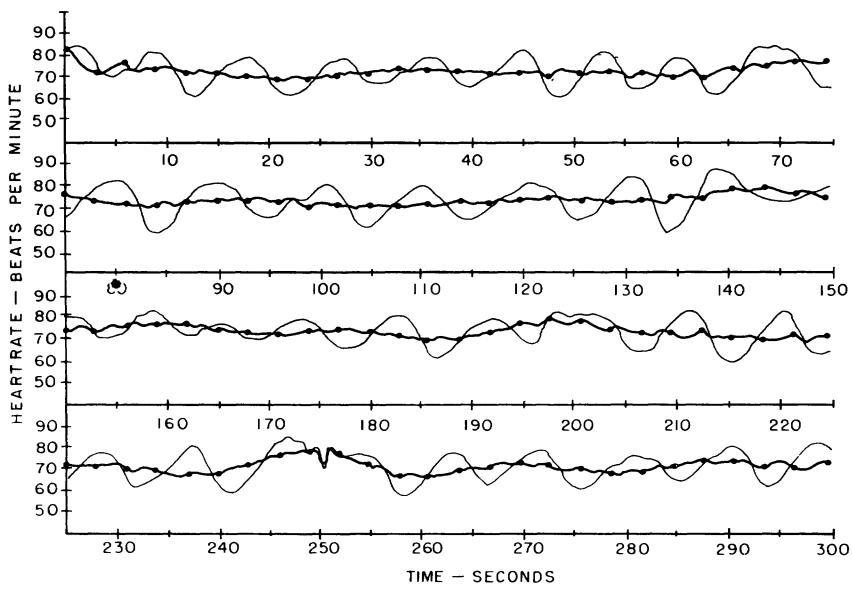


Fig. 8. Heart rate filtering using a 4-14 cycles/min band-stop FFT filter.

changes from one beat to the next) step function into the filter. This causes the output to "ring" for a relatively long period before the transient associated with the step response dies out and valid steady-state filtering becomes available. This transient can be minimized if the "previous" inputs (for negative time) and outputs (the first four) are initially simply set equal to the first input—the 80 beats/min value for the previous example. This is also done at the other end of the input data; i.e., instead of adding on zeros to let the transient die out prior to reversing the data for negative-time filtering, successive constants equal to the last heart-rate value to be processed are used. The transients associated with these operations are quite minor, as shown in Figs. 7 and 8.

VII. THE EFFECT OF FILTERING ON NON-RESPIRATORY ARRHYTHMIAS

In all examples wherein actual heart rate was passed through respiratory arrhythmia filters, it was obvious that low-frequency fluctuations of heart rate were left unaffected by the band-stop filtering operations. This is desirable in order that these slow changes in a subject's mean heart rate can be detected. In order to demonstrate the effect that a typical band-stop filter has on high-frequency arrhythmias, Fig. 9 shows the filtering of a heart-rate sample that has some artificial arrhythmias introduced in it. Such high-frequency changes sometimes occur under normal conditions for any subject regardless of his health. However, these abrupt

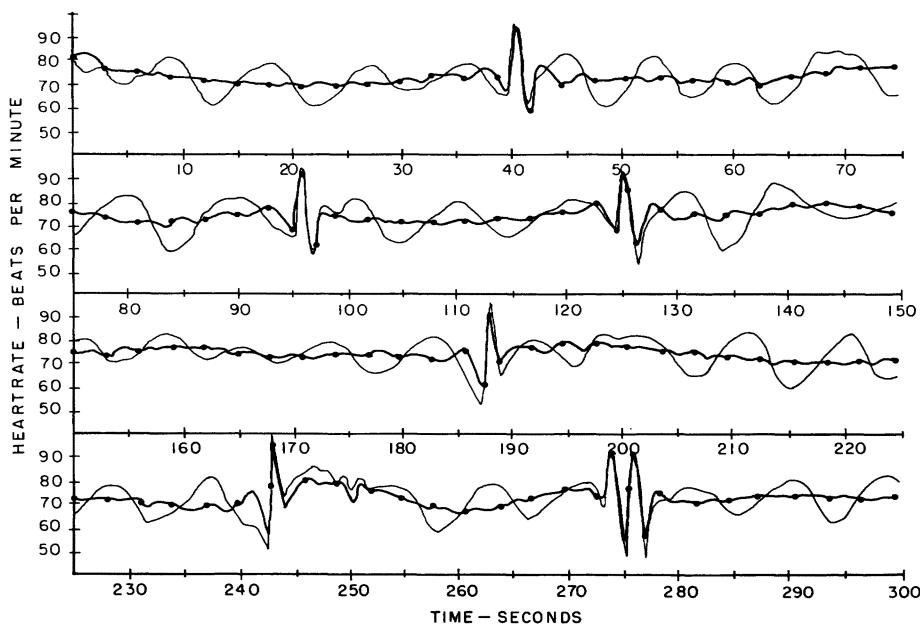


Fig. 9. The effect of a 4-16 cycles/min band-stop filter on some high-frequency artificial arrhythmias.

changes in heart rate often can be associated with certain malfunctions of the heart, and should they occur repeatedly during intensive-care monitoring of a subject a respiratory arrhythmia filter should not attenuate their amplitudes.

A 4-16 cycle/min filter was used for the filtering operation. The first three artificial arrhythmias show the heart rate jumping from normal values to higher values, followed by typical compensatory low values, and then returning back to normal. The filtered heart rate is seen to track the original as desired during these nonrespiratory arrhythmias. The next two arrhythmias show low-high excursions for generality (although this type of change is apparently less common), and the final arrhythmia shows the effect of two successive high-low changes in heart rate.

VIII. RESPIRATORY INFORMATION FROM HEART RATE

The final primary goal of this model was to develop the capability to derive information about respiration solely by observation of heart rate. The investigation has shown that it is not too difficult to use instantaneous heart rate to simply estimate when a breath has occurred. A more stringent aim might be to actually estimate the respiratory waveform itself by using only observed heart rate as an input. Both of these approaches will be discussed below.

Estimation of the Time of Occurrence of Breaths

A rather simple yet effective method of using heart rate to estimate the time of occurrence of each breath has been developed. The technique involves a systematic method of detecting what has sometimes been called the "reflex response" of the heart rate to each breath. That is, during normal breathing each respira-

tory cycle causes the heart rate to fluctuate in a rather consistent manner about whatever "average" exists at the time. A procedure will now be described which senses these fluctuations.

First, the heart rate is low-pass filtered in order to derive a smoothed function which tracks the lower frequency variations in heart rate. This smoothed heart-rate function is then compared point-by-point with the original unfiltered heart rate in order to create two classes of values as a function of time. For purposes of explanation suppose a rectangular waveform is generated that is positive when the original heart rate is greater than the smoothed function and negative when the original heart rate is less than the smoothed function. Each time the rectangular waveform makes a transition from its positive to its negative values, let this denote that a breath has occurred. Fig. 10 shows typical results of this method of breath estimation for normal breathing.

In practice a cutoff frequency of from 5 to 10 cycles/min for the low-pass filter has provided the most sensitive breath-detection capability. These values are higher than might be expected, but the reason that they work best is because rather close tracking of the original heart rate is desirable for sensitivity purposes. That is, even the smallest definite excursion of the actual heart rate from the smoothed function is indicative of respiratory activity, and unless the filtered heart rate is sensitive to most of the lower frequency variations in heart rate, breaths will be missed. Fig. 11 shows an example of the desired behavior of the filtered function relative to the unfiltered heart rate. Of course, if the cutoff frequency is set too high relative to the average respiration rate, the method becomes too sensitive, and consequently useless. The bounds on the method are, therefore, approximately as follows: too low a cutoff frequency (less than about 5 cycles/min for normal

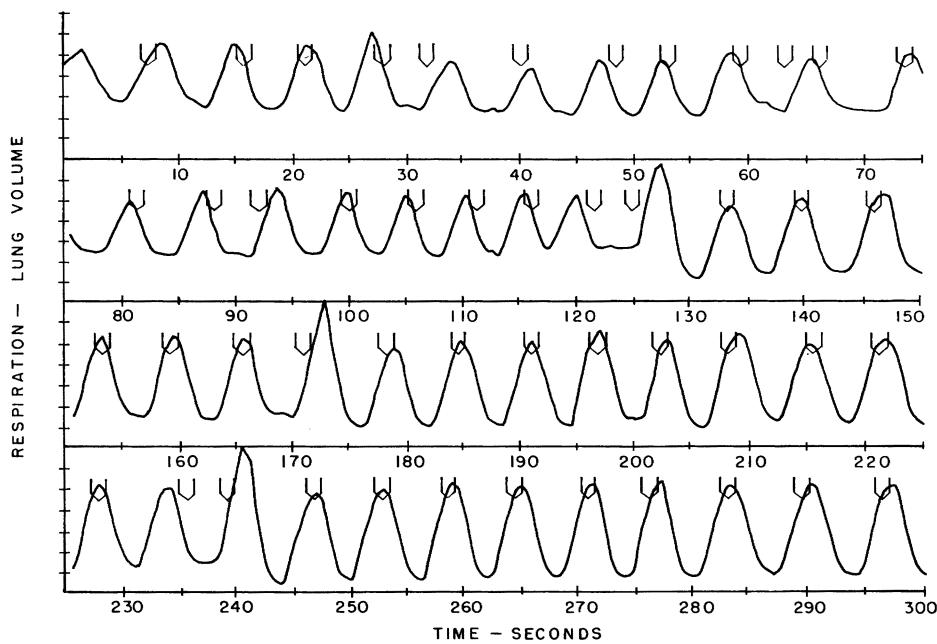


Fig. 10. Actual respiration and estimated times of occurrence of each breath V —subject F .

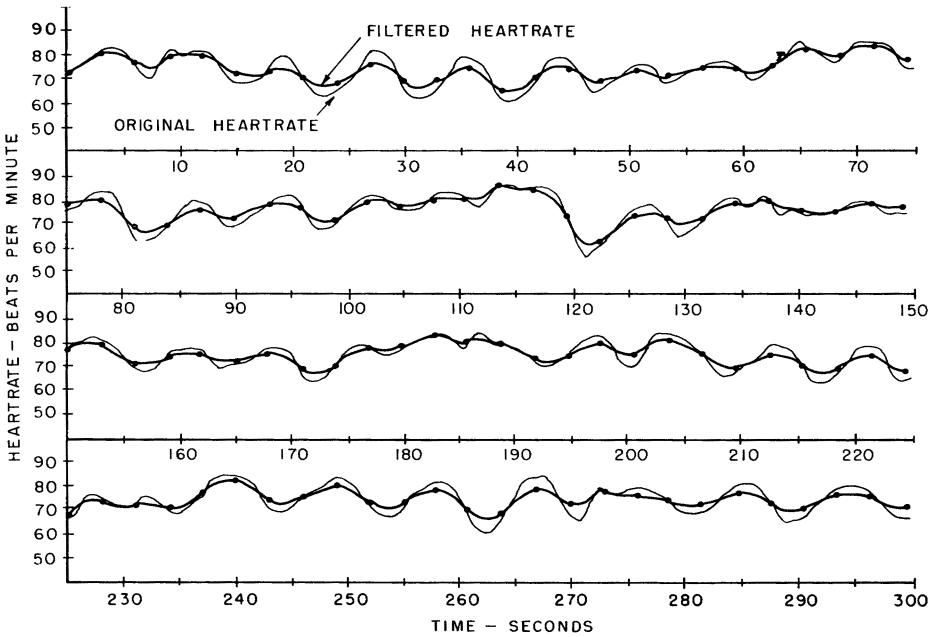


Fig. 11. Low-pass filtering of heart rate.

breathing) generally causes numerous breaths to be missed, and too high a cutoff (greater than about 10 cycles/min) causes false breath detections. Fig. 11 for subject F has a cutoff of 6 cycles/min.

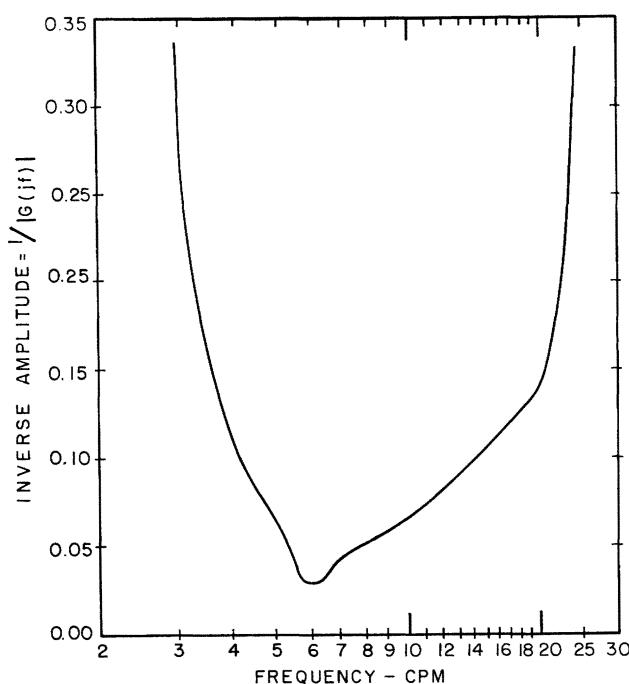
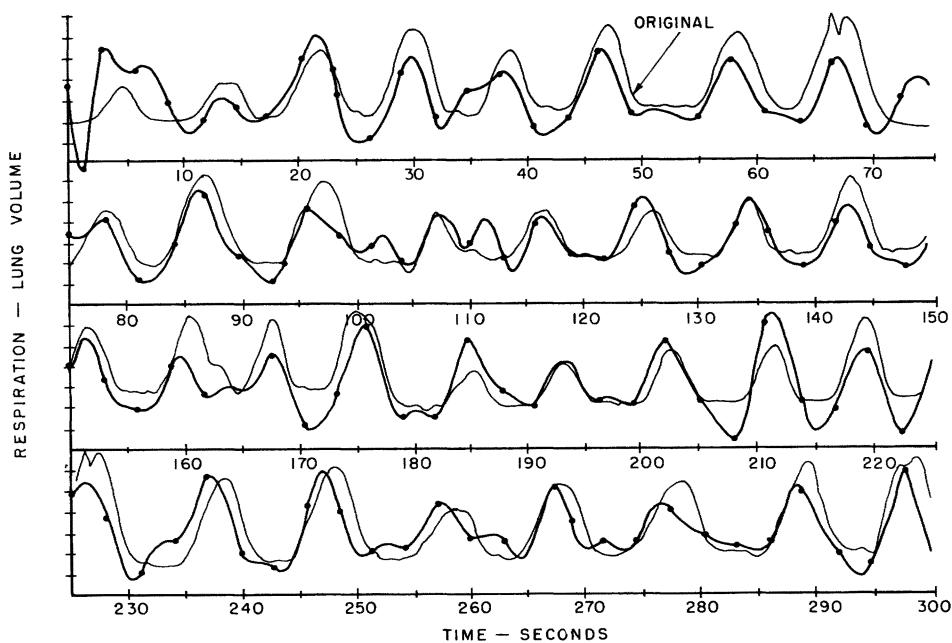
Estimating the Respiratory Waveform

The most difficult thing to do insofar as describing respiration by reference to heart rate is to actually estimate the respiratory waveform. In order to make use of the statistical modeling technique discussed herein, consider the following manipulations:

$$\frac{R(j\omega)}{H(j\omega)} = \frac{RR^*}{HR^*} = \frac{P_{rr}}{P_{rh}^*} = \frac{1}{G} \quad (3)$$

where the parameters are as previously defined. Therefore, a proper linear transform function for the present purpose is the inverse of the transfer function used for deriving heart rate from respiration.

However, a problem immediately arises. Since $G(j\omega)$ goes to (extrapolated) zero in the vicinity of 2 and 30 cycles/min, then its reciprocal gets quite large as these frequencies are approached. Specifically, Fig. 12 shows $1/|G|$ corresponding to the $|G|$ response of subject C . What this behavior actually indicates is as follows: 1) At low frequencies, the heart rate does not contain enough fluctuations to reproduce those observed in respiration; therefore, infinite gain is required. 2) The respiration function for normal breathing, being very "unsinus-

Fig. 12. Inverse amplitude response for subject *C*.Fig. 13. Respiration simulation for subject *C*.

oidal," has much more harmonic content at higher frequencies than does the heart-rate function that it modulates. Again infinite gain is needed for the smoother heart-rate signal to inject these harmonic components into a derived respiration signal. Both of the infinite gains therefore result from the same basic need; i.e., insufficient heart-rate energy in certain frequency ranges to supply that needed to reproduce respiration adequately.

The $1/G$ response can still be used to derive a facsimile of respiration in spite of its instability at low and high frequencies. However, if $1/|G|$ gets too large prior

to "chopping it off" at the band edges, then the resulting impulse response rings excessively. This was in evidence during first attempts to derive an approximation of respiration using this method. However, by restricting the size of $1/|G|$ by using its values only between, say, 4 and 15 cycles/min, and then by smoothing it gradually down to zero either manually or with an appropriate deterministic function, much better results can be had.

One function that has worked well in the smoothing-to-zero operation at the band edges is the hanning function described by

$$S(f) = \frac{M_0}{2} \left[1 + \cos \pi \left(\frac{f - f_0}{f_m - f_0} \right) \right] \quad (4)$$

where $M_0 = 1/|G|$ at frequency $f = f_0$, and f_m = frequency desired for $1/|G| = 0$.

Fig. 13 shows both the original and derived respiration for subject C during uncontrolled normal breathing. As previously stated, certain characteristics of the respiration waveform can never be estimated using this method, because infinite gain is needed in order that the input heart-rate waveform can supply the required harmonic components. For example, the relatively flat portions of the actual respiratory waveform between inhalations are difficult to reproduce, as well as the rather sharply peaked inspiratory maneuvers. However, a good enough facsimile of respiration is produced so that one can usually get an idea as to the general pattern of respiration that is occurring.

The choice of whether to use the breath-time estimation technique described in the previous section or the respiratory waveform estimation method just presented depends on what information about respiration is desired. For simply counting breaths over given intervals to get average respiration rates, then the first method is adequate. Sudden changes in basic respiration rates for a subject can be detected in this manner. One additional advantage of the first method is that no information about specific model parameters need be known for analyzing a given subject.

When model parameters for $G(j\omega)$ have been measured or adequately estimated for a given subject's normal breathing patterns, then the waveform technique previously described can be used. All of the information derivable with the first method is available using this approach, and relative depths of breathing are also available. However, since modeling using $G(j\omega)$ was originally developed primarily for simulation studies, this latter approach is probably not very practical from an operational standpoint.

XI. SUMMARY

This paper summarizes the motivation, applied techniques, and results achieved in a study of respiratory sinus arrhythmia in human beings. The developed model can produce a signal analogous to instantaneous heart rate using the respiration signal as the input to the model. A method is presented whereby breaths are detected using only the observed instantaneous heart rate. Another method is presented whereby the respiratory signal waveform is estimated using observed heart rate as the input signal.

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