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MEASUREMENT OF THE EVOKED CARDIAC RESPONSE: THE PROBLEM OF PRESTIMULUS VARIABILITY

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This paper discusses the problem of prestimulus variability in cardiac activity and its implications for the measurement of evoked cardiac responses (ECRs). Two methods which have been proposed to reduce error arising from respiratory sinus arrhythmia (SA) were reviewed critically. The first approach attempts to control only for the error associated with the sampling of the prestimulus level. This is considered inappropriate since it does not take into account the continuation of SA into the poststimulus period. The second approach reviewed was time series analysis which provides an elegant statistical solution of the problem. Unfortunately, the application of time series analysis has not yet been evaluated for adult cardiac data. A third approach which utilizes an adjustment for SA by employing the actual prestimulus values of an SA cycle was proposed. The application of this technique in the case of a pseudostimulus demonstrated that correction for SA results in significantly smaller 'responses' with less variance than does a conventional procedure which does not take prestimulus variability into account.

1. Introduction

The evoked cardiac response (ECR) has become an important topic for theories concerned with the functional significance of stimulus-bound changes within the autonomic nervous system. Changes in cardiac response amplitude and direction have been ascribed specific relevance for the interrelationship of autonomic and sensori motor activity (Graham and Clifton, 1966; Lacey and Lacey, 1974; Obrist, 1976). Obviously, the use of the ECR in this fashion presupposes that pre- and poststimulus cardiac activity can be differentiated accurately. However, such a differentiation is not always easy, largely because of the variability inherent in cardiac activity.

Cardiac activity is essentially a time series composed of discrete data points. Moreover, the time series appears to represent several different cyclic trends of a

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non-stationary nature, i.e. there may be overall changes in the level of the cyclic fluctuations as a function of time. The periodicity of these cyclic fluctuations may be of a circadian nature involving several hours (Halberg, 1969) or seconds (Sayers, 1975). It is the second of these trends, known as sinus arrhythmia (SA), which is of some consequence for quantification of the ECR, since it is possible for several cycles to occur during a normal analysis period. The bases of SA activity include homeostatic feedback loops responsible for the regulation of respiration (Melcher, 1976), body temperature (Kitney, 1974), blood pressure (Sayers, 1975) and the integration of subcortical and cortical mechanisms (Lacey and Lacey, 1958). Of these, respiratory SA is considered to be the most important and consists of a resting cardiac waveform whith a periodicity of about 3–12 sec and a peak-to-trough amplitude of about 2–20 bpm. Thus a major difficulty in the quantification of the ECR is the differentiation of pre- and poststimulus data points which occur against a background of this waveform. It is with this problem that the present paper is concerned.

2. Error associated with difference scores derived from a mean prestimulus level

The presence of fluctuations in cardiac activity poses obvious problems for the use of difference scores which are derived from a comparison of mean prestimulus level and a number of poststimulus values. First, the mean prestimulus level, derived from a criterion number of values, is influenced by the frequency, amplitude and phase of SA. Thus, even if it were possible to hold frequency and amplitude constant, the prestimulus level will be dependent upon stimulus position in the SA

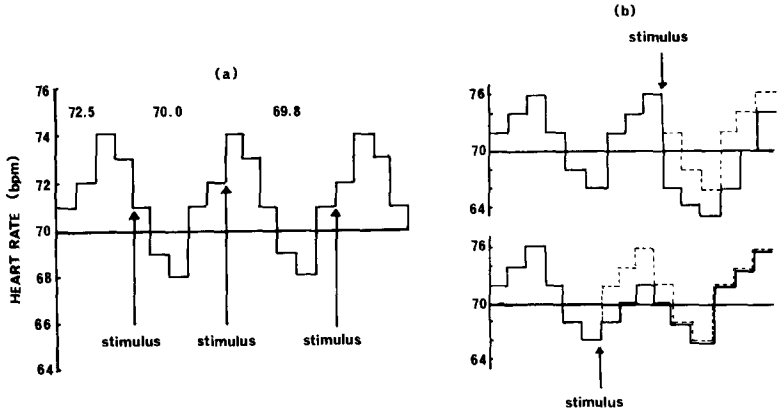

Fig.1. Hypothetical heart rate record showing (a) the effect of stimulus position in the SA cycle on mean prestimulus level (4 beats), and (b) the effect of SA direction on poststimulus activity. The solid line represents observed cardiac activity and the dashed line represents the expected cardiac activity in the absence of stimulation.

Table 1
Means, standard errors and *t*-values for heart rate difference scores derived from a 'pseudo-stimulus'.

	Mean difference scores									
	Poststimulus beats									
	1	2	3	4	5	6	7	8	9	10
Mean	0.04	−0.50	−1.01	−0.37	−0.08	0.42	0.24	−0.49	−0.98	0.10
SE	0.53	0.64	0.57	0.49	0.42	0.43	0.44	0.41	0.53	0.61
t	0.82	−0.79	−1.78	−0.75	−0.18	0.97	0.56	−1.19	−1.85	0.37
	SA-corrected scores									
	Poststimulus beats									
	1	2	3	4	5	6	7	8	9	10
Mean	0.27	0.23	−0.39	0.11	0.39	0.68	0.16	−0.13	−0.49	0.21
SE	0.31	0.44	0.26	0.30	0.31	0.35	0.38	0.41	0.49	0.58
t	0.87	0.51	−1.48	0.35	1.25	1.92	0.42	−0.33	−1.00	0.37

cycle (fig. 1a). Second, if respiratory activity, and hence the characeristics of the SA, are unchanged by stimulus presentation, the phasic components of the ECR will be superimposed upon the SA-bound cardiac activity. The amplitude of such responses will, therefore, be dependent upon the direction of SA at the time of stimulus presentation. This is shown for decelerative responses in fig. 1b. Thus, the ECR represents a summation of the phasic response and the ongoing stimulus-irrelevant cardiac activity. In view of these problems, it is obvious that there is a large source of potential error involved in difference scores derived from a mean prestimulus level. Evidence from several studies suggests that this error does in fact exist. Eisenberg (1975) has demonstrated that for a single subject, statistically significant 'responses' can be obtained from a pseudostimulus, while Williams, Schachter and Tobin (1967) have reported that the direction and rate of change of the prestimulus level contributes significantly to the variance of the poststimulus response. Moreover, Hart (1975) has reported that the shape of an ECR elicited by a 100 dB tone is dependent upon whether the stimulus is presented at peak inspiration or peak expiration.

Since prestimulus variability does appear to influence the shape of the ECR, it is surprising that there have been so few attempts to remove this source of error. One explanation might be the general consensus of opinion that the effects of SA are averaged out when stimulus presentation is random and when the data are collapsed

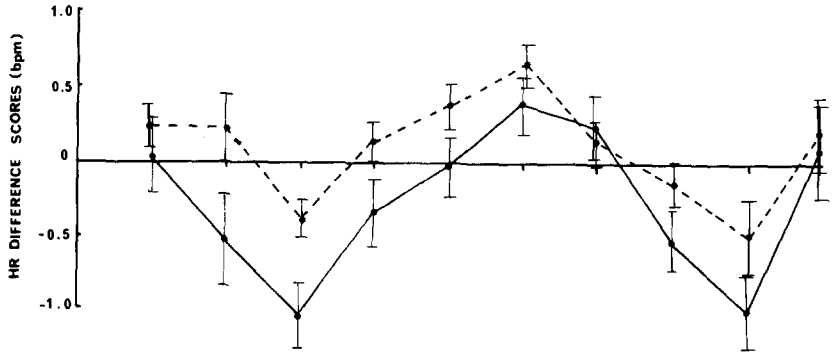

Fig. 2. Beat-by-beat analysis of cardiac activity following a pseudostimulus. The solid line represents the mean difference scores, while the dashed line represents the SA-corrected scores.

across subjects and trials. However, this assumption has never been investigated empirically and some relevant data are presented below.

The technique involved examination of cardiac activity in 75 subjects following a pseudostimulus. [1] This stimulus 'occurred' randomly between minutes three and four of a 5 min pre-experimental rest period. Heart rate difference scores were obtained by subtracting the mean of the last four prestimulus beats from each of the 10 poststimulus beats. The mean difference scores and standard errors, together with t values resulting from a comparison of pre- and poststimulus activity, are shown in the upper section of table 1. The pattern of derived cardiac activity is shown by the solid line in fig. 2, and it can be seen that beats 3 and 9 display deceleration of the order of 1 bpm; such deviations approach statistical significance ($p = 0.08$ and $p = 0.07$, respectively). These findings, together with the implications of fig. 1 and the results of Williams et al. (1967), Hart (1975) and Eisenberg (1975) indicate that the methods which have been proposed to reduce the errors associated with prestimulus variability are worth a closer examination.

3. Reduction of error associated with prestimulus variability

3.1. Extension of the prestimulus sampling interval

Essentially, techniques of this nature attempt to obtain a more reliable measure of the prestimulus level, e.g. mean of the 10 highest prestimulus beats (Engel, 1960), peak rate in a 10 sec interval (Opton, Rankin and Lazarus, 1965) and the

[1] The pseudostimulus technique involves the selection of a point on the cardiac record where no stimulus has occurred and derives a 'cardiac response' from pre- and poststimulus values in the same manner as that employed for actual stimuli.

Method of Mean Cyclic Maxima (Malmstrom, 1968). Although such methods may reduce the variability of the sampled prestimulus level (fig. 1a), they cannot correct for the continuation of a regular cyclic fluctuation such as SA which occurs during the poststimulus period (fig. 1b).

3.2. Time series

This approach considers cardiac data as a discrete stochastic process which may be expressed as some function of the dependency of the observations on time; in turn, this function is superimposed on a background of random variation. The function used to describe this process may be of two basic types. The moving average (MA) model expresses the current observation (z_t) in terms of its dependency on a *finite* number of previous observations. Hence, a moving average model of order 1, MA (1), is expressed:

$$z_t = \mu + u_t - \theta_1 u_{t-1} \tag{1}$$

where μ is the mean of the process, u_t is some random variate, and θ_1 is a fixed parameter of the stochastic process. The autoregressive model (AR), on the other hand, expresses the current observations in terms of the current disturbance and *all* past observations. An autoregressive model of order 1, AR (1), is expressed:

$$z_t = \phi_1 z_{t-1} + \delta + u_t \tag{2}$$

where ϕ_1 is a fixed parameter of the process and δ is a constant. The choice of the appropriate model is governed by a specific pattern of dependency between the observations and time, and this is described by the autocorrelation function of the data (Gottman, McFall and Barnett, 1969; Nelson, 1973). Procedures exist by which the model may be identified and its parameters estimated (Brown, 1962; Box and Jenkins, 1970).

Time series analysis has been applied to cardiac data by Jones, Crowell and Kapuniai (1969, 1970), Jones, Crowell, Nakagawa and Kupuniai (1971) and Lobstein (1974, 1976). The Jones et al. (1969) paper proposed a first-order AR model and later papers by Jones and his colleagues represent attempts to improve the stepwise estimation of the AR coefficients and also to overcome, by adaptive estimation techniques, the problem of changes in the individual parameters of the model. Lobstein's (1976) extension of the original Jones et al. (1969) model consists essentially of a higher-order AR model which uses four ϕ weights. Lobstein has argued that this is necessary since the residuals from the first-order model were found to be dependent upon time. All these procedures are essentially the same once the parameters of the model have been estimated. Using the estimated AR coefficient ϕ, forecasts z_t may be made using equation [2]. The deviations of the actual poststimulus beats from the predicted beats are then taken to represent the stimulus evoked cardiac activity.

A number of basic criticisms of this approach can be made. First, there have

been no attempts to identify the nature of the appropriate statistical model or to show its autocorrelation function. The choice of the AR model was made in terms of its practical advantages over other time series models rather than in terms of the statistical structure of the cardiac data. It should be pointed out that although procedures for identifying the appropriate model have been proposed (Box and Jenkins, 1970; Nelson, 1973); these procedures are extremely complex, and it may be more useful to adopt the approach suggested by Kendal (1973, p. 117). Kendal has argued that the suitability of a chosen model will manifest itself after several applications, since the previous forecasts can be compared with the observed data when it has been collected. This 'test of time' has not, and indeed cannot, be applied to experimental cardiac data. Here, the observed poststimulus beats must always be contaminated by experimental intervention, i.e. a stimulus. It can be argued, therefore, that any time series application to cardiac data should be tested using a pseudostimulus, since this allows the hypothesis that there are no differences between observed and predicted values to be tested. The use of a pseudostimulus has been applied to the adaptive AR model suggested by Jones et al. (1971), where its practical suitability for cardiac data derived from infants was clearly demonstrated.

A second criticism concerns individual variation in the model, both between subjects and trials. The simple AR model ignores two fundamental properties of cardiac data. First, SA represents a seasonal (i.e. cyclic) variation and this is not incorporated into a simple AR model. Models do exist which will take into account seasonal terms which may be either independent of (additive) or dependent on (multiplicative) the mean prestimulus level (Box and Jenkins, 1970; Winters, 1960), but these have not been applied to cardiac data. Second, it is often the case that across trials or even within a small portion of prestimulus data, the time series is nonstationary. Jones et al. (1971) have overcome this problem to some extent by using an adaptive model, the parameters of which are updated prior to each trial. However, Lobstein's (1976) approach does not easily take this problem into account.

The third criticism concerns the number of observations from which the ϕ weights are estimated. Here, there are two conflicting interests; if a higher-order AR model of order p is to be used, there should be sufficient observations (of the order $5p$ to $10p$) for reliable estimation of the ϕ weights (Montgomery and Johnson, 1976, p. 236). However, such a procedure may involve an inconveniently long interstimulus interval of about 60 sec. Both Jones et al. (1971) and Lobstein (1976) avoid the problem of long interstimulus intervals by employing prestimulus sample sizes smaller than those usually recommended (9 and 10 values, respectively). Jones et al. (1971) suggest that the use of an adaptive model is sufficient to overcome the problem of statistical instability which is consequent upon the use of small prestimulus samples. However, it should be pointed out that the procedure employed by both Jones et al. and Lobstein to minimize the effects of unreliable AR coefficients is itself open to criticism. Both of these authors have advocated the use of

the actual observed poststimulus values, or in the case of Lobstein, a weighted product of the observed and the previously predicted values, for the initial estimate of z_t; this is subsequently substituted in equation (2) for z_{t-1}. It seems that this procedure has been adopted in preference to the usual practice of using previous forecasts as the basis of future predictions in order to overcome cumulative forecast errors associated with the unreliability of the AR coefficients. However, since the observed poststimulus beat may bear no relation to the prestimulus process from which the ϕ weights have been estimated this procedure cannot be considered a solution. It will give rise, at the very least, to conservative estimates of the response deviation and perhaps even erroneous ones. One solution may be to extend the notion of adaptive estimation as suggested by Jones et al. (1971) and construct a model from a *lengthy* sample of pre-experimental data for each subject, and update the model prior to each stimulus presentation. This scheme is conceptually similar to the Kalman filter used in control engineering (see Young, 1974), and because of its recursive nature, might be applied to cardiac data.

In conclusion, it would appear that time series models contain the potential for a statistically sophisticated solution to the problem of prestimulus variability. However, there are certain problems which require further investigation. These include the choice of AR models in preference to other time series models which might be more appropriate for cardiac data, the use of adaptive estimation procedures in order to overcome the problems of departures from stationarity and the use of unstable prestimulus sample sizes. Although Jones et al. (1971) have attempted to resolve these problems and have to some extent evaluated their own model, it should be pointed out that their data were derived from infants. As Lobstein (1976) has suggested, the parameters necessary to describe adult cardiac activity may well be different from those which are appropriate in the case of infants.

3.3. Correction for sinus arrhythmia

A technique developed in this laboratory utilizes the SA waveform present in the cardiac record to predict what the poststimulus activity would have been in the absence of a stimulus. The difference between the predicted and observed poststimulus values is then attributed to stimulus intervention. Essentially, the technique involves the determination of the period of the SA cycle (expressed in terms of number of beats, T) just prior to stimulus presentation. The last T prestimulus values are then used as a model for predicting poststimulus activity in the absence of a stimulus. This is shown in fig. 3 in which the dashed line respresents the model prediction. The SA-corrected difference scores are obtained by subtracting each model beat from its corresponding observed beat. In order to illustrate the technique and assess its usefulness, it has been applied to the pseudostimulus data already presented in fig. 2 and table 1. The means, standard errors and t values from this second analysis are presented in the lower section of table 1 and the cardiac response curve by the dashed line in fig. 2. Although beat 6 tended to display signif-

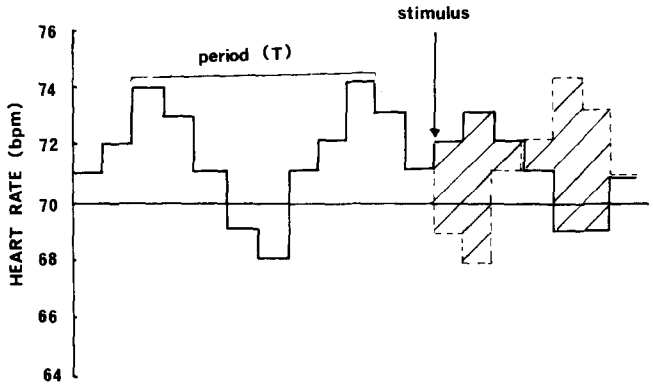

Fig. 3. Hypothetical heart rate record demonstrating the application of the SA-corrected method. The solid line represents observed cardiac activity and the dashed line represents the predicted activity in the absence of stimulation. The hatched area represents the inferred stimulus-evoked changes.

icant acceleration ($p = 0.06$), fig. 2 indicates that in general, the mean amplitude of the difference scores and their variability have both been reduced. A two way analysis of variance in which the main factors were scoring method and beats was performed on the cardiac difference scores. The SA-corrected scoring method resulted in smaller poststimulus difference scores than did the mean difference technique ($F = 3.76$, df = 1,74; $p < 0.05$). [2] The quartic component across beats ($F = 8.68$, df = 1,74; $p < 0.01$) and the scoring method difference in quartic trend ($F = 4.72$, df = 1,74; $p < 0.05$) were also significant. The data were analysed further to determine whether the SA-corrected method resulted in a reduction in variance across poststimulus beats. The total poststimulus variance for each subject was calculated and a comparison made between scoring methods. The SA-corrected method displayed significantly smaller variance than did the mean difference method ($F = 5.15$, df = 1,74; $p < 0.05$). However, it could be argued that since there was a difference between the methods in terms of quartic trend, the Mean Square Successive Difference statistic (Varni, Clark and Giddon, 1971) might be more appropriate. When the variance data were re-analysed using this statistic, there was still a strong tendency for the SA-corrected method to result in a smaller variance ($F = 2.80$, df = 1,74; $p < 0.1$). These results indicate that the SA-corrected

[2] The most likely explanation for the predominance of negative scores obtained when using the mean difference scoring method is to be found in the sampling bias introduced when the arithmetic mean of a number of beats, expressed in bpm, is derived. Thorne, Engel and Holmblad (1976) have shown that mean heart rate derived in this fashion always exceeds the true rate which is obtained from inter-beat intervals. Thus, prestimulus level derived from bpm, is an over-estimation of true heart rate.

method reduces the variance associated with both the trend across beats and the difference scores.

Although the above results are encouraging, it could be argued that an actual rather than a pseudostimulus should have been employed. However, the use of a pseudostimulus possesses two advantages. First, null hypotheses concerning the difference between pre- and poststimulus activity can be formulated. Second, since there is no agreement about what the shape of an ECR *ought* to be, the application of the SA-corrected method to stimulus-evoked cardiac activity would have been of little value, at least in the first instance. Nevertheless, the method has been applied to some recent data in this laboratory. In this experiment, subjects were exposed to pure tones of 45, 60, 75, 90 and 105 dB (re: 0.0002 dyne/cm^2). There were 15 subjects in each intensity group. For each group, a beat-by-beat analysis was performed on the first ten poststimulus beats. Both the SA-corrected and the mean difference techniques were employed and in the latter case, each poststimulus beat was compared with the mean of the four prestimulus beats. Both sets of data were analysed using analysis of variance, and although the intensity effects were significant in both cases (for mean difference, $F = 3.76$, df = 4,70; $p = 0.008$; for SA-corrected, $F = 4.74$, df = 4,70; $p = 0.002$), the proportion of the between subjects error term sums of squares was 11% less for the SA-corrected technique than for the mean difference procedure. For the mean difference technique, this error term constituted 36% of the total sums of squares, while for the SA-corrected, it constituted 25%. Since this error term represents idiosyncratic responses (Wilson, 1967), the findings suggest that ECRs derived from the SA-corrected method are more consistent across subjects. The technique has also been employed in a replication of Hart's (1975) study. Kamath (personal communication, 1977) has shown that ECRs derived from the differences between poststimulus activity and the third from last prestimulus beat are influenced by stimulus position in the respiratory cycle. However, this effect is dissipated when the SA-corrected method is employed. In general, the above findings all suggest that the SA-corrected method results in clearer ECRs when applied to actual experimental data.

Although the SA-corrected technique possesses certain advantages due to its simplicity, there are some limitations. The use of the actual prestimulus values for the prediction of SA activity into the poststimulus period requires a relatively regular prestimulus pattern. Moreover, the technique cannot deal with rapidly changing tonic levels unless some correction factor is introduced to take into account the nonstationary nature of the time series. Furthermore, the method relies to a large extent on visual assessment of the periodicity of the SA waveform. However, two approaches to a computer assessment of SA periodicity are possible. First, if the prestimulus series preceeding each stimulus was differenced using a lag of one (i.e. $x_1 - x_2$, $x_2 - x_3$, ..., $x_{n-1} - x_n$), the positions of the peaks and troughs of the SA cycle would be seen as changes in the sign of the difference scores. The mean SA period could then be assessed as the mean number of beats between each successive peak or trough. An alternative approach involves calculating the variances

for a sample of prestimulus data lagged from 2 to 12. The period of the SA would be represented by the lag with the smallest associated variance.

4. Discussion

This paper has examined the problem of cardiac prestimulus variability when measuring the ECR. The presence of error associated with ECRs derived from a mean prestimulus level has been discussed and attempts to reduce this error have been reviewed. Although the pseudostimulus approach did not reveal the occurrence of 'significant response', the difference scores and their associated variances were shown to be larger than those derived by the SA-corrected method. It is suggested, therefore, that when detailed information concerning the exact nature of the ECR is required, methods which attempt to reduce prestimulus variability should be employed. This would seem to be particularly important in habituation studies, for example, where a distinction between responses and nonresponses is often of prime interest. It should be noted that the use of either the SA-corrected technique or time series analysis should not alter the *shape* of for example, the conditioned cardiac response or the cardiac response component of the orienting response. Moreover, it should not influence the form of the habituation function. Rather, these techniques reduce the error which arises from prestimulus variability and therefore, increases the sensitivity of the ECR to experimental manipulations.

Before making any general recommendation regarding the appropriateness of any of the techniques discussed in this paper, a general assumption that underlies all of these approaches should be recognised. These techniques assume that the ECR may be represented as deviations of poststimulus beats from some form of prestimulus activity. The response consists, therefore, solely of deviations in cardiac activity due to stimulus intervention. This does not necessarily imply that the ECR and the stimulus are causally related in a direct fashion. Indeed, changes in respiratory or somatic activity may well be responsible for part, if not all, of the ECR (Obrist, 1976). The presence of these involuntary cardio-reflexes does not invalidate any of the methods, although this source of response activity ought to be considered when assessing the interaction of the ECR with psychological variables. Reflexive sources of cardiac activity may only be teased out of the overall pattern of the ECR by the use of multivariate comparisons of the ECR with the responses derived from other physiological response systems thought to be reflexively linked to cardiac activity. It should also be pointed out that all three methods discussed here are concerned only with amplitude changes in cardiac activity. Deviations will also result from a phase difference between pre- and poststimulus beats. None of the methods can correct for errors associated with a phase shift which occurs early during the poststimulus period. If changes of phase are suspected, alternative forms of analysis such as Fourier analysis (Sayers, 1975) should be applied. This will probably necessitate the collection of longer pre- and poststimulus series.

Three approaches which attempt to reduce the error associated with prestimulus variability within cardiac data have been discussed. The first attempts to reduce the sampling error associated with mean prestimulus level by extending the sampling interval. However, this procedure does not take into account the continuation into the poststimulus period of a regular cyclic fluctuation. Both time series analysis and the SA-corrected method, on the other hand, attempt to do this by the construction of a prestimulus model. Although time series analysis results in a statistically more elegant approach and has been shown to be effective for infant cardiac data (Jones et al., 1971), the appropriateness of the autoregressive model for adult cardiac data has yet to be demonstrated. There are several statistical procedures which could be used to assess the fit of such a model, but a more relevant test might be that of a pseudostimulus analysis using a number of subjects and trials. This would be of particular importance when time series analysis is applied across groups of subjects (Lobstein, 1976), since the statistical basis for such an analysis is less robust. If errors of forecasting are demonstrated using a pseudostimulus technique, modifications to the original model could be considered. Alternative schemes such as the Holts-Winters procedure for small samples with seasonal fluctuations (Montgomery and Johnson, 1976; Kendal, 1973, p. 126) might also be considered. Time series analysis does, on the other hand, possess advantages over the SA-corrected technique in that it is readily amenable to on-line computer application. Whether the SA-corrected technique can be similarly automated relies on the programming of a procedure which would identify the periodicity of the SA waveform prior to stimulus presentation. One major advantage of the SA-corrected technique is that it avoids many of the statistical assumptions necessary in the application of time series analysis to cardiac data. In conclusion, it would appear that techniques such as those discussed in this paper are necessary for deriving ECR scores but that those available at present await demonstrations of reliability.

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