CARDIAC HABITUATION TO AN AUDITORY STIMULUS IN WELL AND MALNOURISHED INFANTS

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ABSTRACT

CARDIAC HABITUATION TO AN AUDITORY STIMULUS
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By

Barry Marshall Lester

This study examined the effect of nutritional insult on the magnitude and habituation of the orienting response (OR) in one-year-old male infants. Twenty well nourished and twenty malnourished infants from the lower social-economic class in Guatemala City, Guatemala, were presented with twenty trials of a pure tone stimulus. The infants were full-term, full birth weight, clinically normal and suffered no major illnesses during the first year of life. The design was balanced for the order of stimulus presentation. Twenty subjects were presented with ten trials of a 750 Hz tone followed by five trials of a 400 Hz tone and five trials of the 750 Hz tone. For the remaining twenty subjects this order was reversed. The tones were presented at 90 db for five seconds with a randomized inter-trial interval. The major dependent variable was heart rate deceleration and all infants were tested while in an awake and alert state.

The results showed that for the well nourished infants each tone sequence was characterized by a large initial OR followed by rapid habituation. In contrast, there was no change in heart rate deceleration to any of the tone sequences for the malnourished infants. These infants showed an attenuation or complete absence of the OR. The results were taken as evidence of a fundamental attentional deficit associated with malnutrition that probably interferes with learning. Such a deficit, if it persists into childhood may account for the often reported poor performance of malnourished children on standard psychological tests.

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By

Barry Marshall Lester

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To Freda who said that I could

and to Hi who made sure that I did.

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INTRODUCTION

Nearly two-thirds of the world's children suffer from some form of protein-calorie malnutrition (Habicht, 1972). Of recent concern is the possibility that malnutrition may have deleterious effects on mental development, particularly when nutritional insult is suffered early in life. Several studies have demonstrated that children who were malnourished during infancy score significantly lower on tests of psychological performance than their well nourished counterparts (Barrera-Moncada, 1963; Cabak & Najdanvic, 1965; Monckeberg, 1968; Stoch & Smythe, 1968). For example, Stoch and Smythe (1968) compared the performance of well nourished and malnourished samples on the New South African Individual Scale over an eleven year period. Sixty percent of the malnourished group fell below the level of the lowest child in the control group. Only one child in the malnourished group exceeded the mean of the control group.

Infant Studies

Nevertheless, few attempts have been made to directly assess the effects of malnutrition on behavior during the first few years of life. Moreover, most studies which

have focused on infancy have been confined to reporting gross differences in psycho-motor functioning through the administration of infant scales. Cravioto and Robles (1965) administered the Gesell Developmental Schedules to infants hospitalized for malnutrition at various ages. When admitted to the hospital all infants showed developmental retardation. The effectiveness of nutritional rehabilitation varied as a function of the infant's age upon admission. Infants admitted at 37-42 months of age showed significant improvement as indicated by the Gesell Developmental Quotient (DQ) score. Those admitted under six months of age continued to evidence developmental lag even after 6 1/2 months of hospitalization.

Similarly, Chase and Martin (1970), compared the psycho-motor performance of infants who were hospitalized for malnutrition during the first year of life with a well nourished control group. No differences in performance on the Yale Developmental Scale were found between control children and those hospitalized before four months of age. However, children who were hospitalized after four months of age showed decrements in performance when compared with both the control group and those children hospitalized after four months of age. Finally, Stoch and Smythe (1963), reported DQ differences of up to twenty points between well and malnourished two year olds and other studies have also reported depressed infant scale performance among malnourished infants (Barrera-Moncada, 1963; Geber & Dean, 1956;

Monckeberg, 1968; Pollitt & Granoff, 1967). However, the age specific effects of malnutrition are not clear since factors such as the age of onset of malnutrition (particularly as related to age of weaning) and the length of the disease have not been studied in relation to psychological performance.

Brockman and Ricciuti (1971) used a simple sorting task to compare the cognitive development of Peruvian infants hospitalized for marasmatic malnutrition with a well nourished control group. The authors suggested that the poor performance of the malnourished infants reflected a retardation in the acquisition of concepts. Moreover, performance was lower among malnourished infants less than 24 months of age than among malnourished infants greater than 24 months.

While the above studies suggest a relation between early nutritional inanition and subsequent intellectual development, there are at least two levels on which they may be criticized. First, malnutrition does not occur independent of other salient biological and social circumstances (Klein, Lester, Yarbrough, & Habicht; 1971, Birch & Gussow; 1970). Malnutrition seldom is found among the wealthy! Consequently, effects due to malnutrition per se are often confounded with other intervening factors such as social class and prenatal, perinatal and postnatal infection. These factors have been shown to interact with nutritional effects in the prediction of psychological performance,

(Klein, et al., 1971). Nevertheless, malnutrition is related to mental development even when other environmental variables are controlled (Klein, Lester, Yarbrough, & Habicht, 1972).

Another non-nutritional factor which may complicate the interpretation of previous studies is the fact that many of the malnourished children were hospitalized for varying lengths of time during nutritional rehabilitation. Thus, nutritional and institutional affects may have been confounded.

A second level of criticism of the above studies relates to the use of infant developmental scales as the major dependent measure of the effects of malnutrition. These scales are predominantly made up of motor test items and while they are useful as descriptors of gross psychomotor functioning they do not adequately assess cognitive functioning. In other words, infant scales do not specify the underlying psychological processes that may be affected by malnutrition. Moreover, their power to predict later intellectual performance is often reported to be low (Bayley, 1970).

Some studies have attempted to differentiate various types of performance through the further breakdown of mental and motor scores into component sub-scales, such as language or adaptive behavior. However, it is still not clear what psychological mechanisms are affected by malnutrition to produce a deficit in language performance (Chase &

Martin, 1970). This same logic can be applied to the Brockman and Ricciutti study (1971): How does malnutrition affect concept aquisition? Does an inability to discriminate among task stimuli result from a deficiency in inter-sensory organization as Brockman and Ricciutti (1971) suggest, or does some more fundamental process such as attention underlie poor discrimination performance?

Clinical observation of malnourished infants has repeatedly indicated that nutritional insult interferes with attentional processes. Indeed, apathy is one characteristic behavioral feature of the clinical syndrome of malnutrition (Birch & Gussow, 1970). The malnourished infant is universally found to be unresponsive to stimulus changes in the environment and to lack the exploratory behavior, curiosity, and general activity so typical of the healthy infant (Correa, 1908, DeSilva, 1964; Geber & Dean, 1956; Stoch & Smythe, 1968). The magnitude of apathy in the malnourished infant gives meaning to the statement that "Once a child can be persuaded to smile he is well on the way to recovery" (Clark, 1951).

Nevertheless, the clinical observation of attentional deficits in malnourished infants has not been systematically investigated with experimental methodologies. Therefore, the purpose of the present study was to provide an experimental test of the hypothesis that the infant's ability to respond appropriately to impinging environmental stimuli is affected by nutritional insult.

Attention and Habituation

Habituation refers to centrally mediated response decrement following the repeated presentation of a novel stimulus that is not accounted for by peripheral mechanisms such as receptor or effector fatigue. Habituation has become increasingly popular among infant psychologists as a technique to evaluate a number of different processes such as sensory development, learning, perceptual development, attention, memory, and information processing (Jeffrey & Cohen, 1971).

Habituation is related to selective attention. It enables the organism to actively ignore or filter non-salient aspects of the environment and to simultaneously increase receptivity to high priority events. If the case were otherwise, the result would be biologically disastrous since the organism would be quite at the mercy of all stimuli bombarding it at any moment without being able to discriminate biologically important from biologically unimportant stimuli.

The habituation paradigm is also attractive as a possible indicator of the integrity of the central nervous system (CNS) in infants suffering from nutritional insult. This hypothesis, that response decrement is related to central processes and is, therefore, relevant in the assessment of early cognitive development, derives from the theoretical notions of Sokolov (1963). Sokolov suggested that

the organism preserves information about stimuli by forming a "neural model" of the intensity, quality, duration, and order of presentation of stimuli. This model becomes more developed with each repetition of the stimulus. Each new stimulus is then compared to the model and if stimulus input coincides with its neural model, that stimulus is "recognized" and no orienting response (OR) occurs. However, if there is a discrepancy between an incoming stimulus and the neural model for that stimulus (if there is a mismatch between model and input) an OR is elicited. Thus, with repeated presentations of the same stimulus, a progressive response decrement is indicative of model acquisition. It follows that rate of response decrement should be related to the speed of model acquisition.

If the process of habituation depends upon CNS involvement in the form of model building of prior input (Sokolov, 1963), one might expect the rate of habituation to be a sensitive measure of CNS integrity. This hypothesis receives marginal support from a study by Bronshtein, Antonova, Kamenetskaya, Luppova, and Sytova (1958) in which the habituation of sucking suppression to auditory input did not occur in about half of the infants suffering trauma at birth, whereas it did occur in three quarters of the normals. In addition, slower habituation was reported in "suspect babies" even after the signs of trauma had disappeared and the infants were judged to be clinically

normal. In the same study, absence of habituation to sound stimuli was reported in hydrancephalic infants. Similarly, Brackbill (1971) presented an auditory stimulus to a single anencephalic infant and found no evidence of habituation of the startle response. Lastly, Eisenberg, Coursin, and Rupp (1966) reported that whereas normal infants required from 20 to 37 trials to habituate to a change in tone, two "suspect babies" required almost twice as long, while two high-risk babies showed no evidence of habituation.

Rate of habituation has also been studied in premature infants. While Peiper (1963) reported habituation of a respiratory response to auditory stimuli in premature babies, Polikanina and Probatova (1965) and Polikanina (1961) reported no habituation of the OR to sound in premature babies; habituation did occur in full term babies. Finally, in a discussion of the Russion research on early insult Bronshtein et al. (1958) reported that premature babies evidenced a delay in the rate of habituation. While the habituation paradigm has been used to discriminate normal from "suspect babies" or premature babies, this method has not been used in the assessment of infants suffering from nutritional insult.

The present investigation compared orientation and habituation to an auditory stimulus in well nourished infants and in infants suffering from malnutrition. The

design involved the presentation of 10 trials of a pure tone followed by 5 trials of a second tone of a different frequency followed by 5 trials of the original tone.

The importance of this sequence of stimulus presentation is that it permits a distinction between response decrement due to central mechanisms and response decrement due to peripheral mechanisms. Thompson and Spencer (1966) have outlined nine parametric characteristics of habituation which, if met, would rule out response decrement due to peripheral mechanisms. Applying these characteristics to the present investigation would predict: (1) an OR followed by response decrement during the presentation of the first 10 trials; (2) response recovery or dishabituation to the change in tonal frequency (trials 11-15); (3) habituation of dishabituation, or response decrement to this change in tone; (4) a second dishabituation when the original tone is again presented (trials 16-20); and (5) response decrement of this second dishabituation during trials 16-20.

Attention and Heart Rate

Cardiac deceleration was the primary dependent variable in the present study. Cardiac deceleration as an attentional measure was first reported by the Laceys' (1959) and was viewed as the organism's way of increasing its receptivity to external events. The Laceys' suggested that cardiac deceleration accompanies stimulus intake whereas cardiac acceleration is related to the startle or defensive

reaction. Graham and Clifton (1966) further reinforced the notion that the cardiac component of the OR is deceleration.

An alternative interpretation of cardiac deceleration is that a decrease in heart rate reflects a lowering of general somato-motor activity; that heart rate decreases as the organism becomes quiet (Obrist, Sutterer & Howard, 1969). Some support for this hypothesis was reported in a study in which cardiac decelerations to visual stimuli were accompanied by decreases in motor activity (Kagan, 1971). The clear implication is that cardiac deceleration and acceleration are manifestations of different phenomena. Whereas cardiac deceleration is viewed as an attentional response accompanied by motor quieting, cardiac acceleration is indicative of behaviors that co-vary with increased motor activity, such as fear or boredom.

Cardiac deceleration as an attentional measure has also been employed in studies involving the presentation of auditory stimuli as in the present investigation (Bridger, 1961; Horowitz, 1971). Since attentional processes were of major concern in this study, cardiac deceleration was the primary dependent measure although other indices of cardiac activity were also computed.

Pilot Study I

Method

The purpose of the first pilot study was to determine the feasibility of an experimental design in which pure tone stimuli are presented to well nourished and malnourished infants.

The subjects were eight children ranging from 8 months to 40 months of age. Four children fully recuperated following severe marasmus comprised the experimental group. Four other children matched for age and sex comprised the control group. Both experimental and control children were from the lower social-economic class (SES) of Guatemala City.

The children were presented with 20 trials of a 400 Hz pure tone followed by 10 trials of a 750 Hz pure tone, followed by 15 trials of a 400 Hz pure tone. All tones were presented for five seconds at 65 db. The intertrial interval (ITI) was randomized with a mean of 14 seconds and a range of 10 to 18 seconds. The dependent variable was heart rate calculated as a change score in cardiac activity from prestimulus to stimulus onset periods. The precise method of data reduction is described in the main study.

Results and Discussion

Table 1 presents the mean heart rate decelerations, accelerations and range of heart rate by blocks of five trials for the well and malnourished groups. The magnitude of the heart rate decelerations during the 20 presentations of the first tone is of particular interest. The well nourished children showed a classic habituation pattern; the magnitude of the decelerations was substantial during the first block of trials, then gradually leveled off by the final block. In contrast, the previously malnourished children showed no such habituation pattern. Their slight heart rate decelerations remained stable across all presentations of the first tone.

During the 10 presentations of the change in tonal frequency the well nourished group showed an increase in the magnitude of heart rate accelerations as compared to the malnourished group. These data suggested that the tonal change may have had a disquieting effect on the well nourished, and that in contrast to the malnourished group, the well nourished children recognized the change.

These findings are also supported by the heart rate variability data. The changes in rate variability reflected substantially more heart rate activity during the entire experimental procedure for the well nourished than for the malnourished group. Indeed, the three heart rate measures taken together showed a general stability and lack of

TABLE 1.--Analyses of Heart Rate Responses by Clocks of Five Trials for Children in Pilot Study I.

Group	S1*	S2	S3	S4	$\bar{x}$	T1**	T2	$\bar{x}$	S1	S2	S3	$\bar{x}$
Mean Heart Rate Deceleration												
Well Nourished (N=4)	8.45	3.75	4.30	1.05	4.39	2.75	2.45	2.60	1.85	2.30	3.35	2.50
Malnourished (N=4)	3.70	2.35	2.40	2.90	2.81	2.45	3.15	2.80	1.70	1.70	2.80	2.07
Mean Heart Rate Acceleration												
Well Nourished (N=4)	0.40	1.35	3.80	1.95	1.87	2.70	4.05	3.37	3.30	1.20	2.45	2.32
Malnourished (N=4)	1.40	2.40	1.00	1.90	1.67	2.85	2.35	2.60	2.50	2.30	0.90	1.90
Mean Change In Heart Rate Variability												
Well Nourished (N=4)	3.50	3.05	2.85	4.05	3.36	3.70	3.65	3.67	3.60	2.80	3.60	3.33
Malnourished (N=4)	4.85	1.90	6.40	3.25	4.10	3.95	5.60	4.77	3.25	3.60	6.50	4.45

* S = 400 Hz
** T = 750 Hz

activity for the previously malnourished children, whereas for the controls, both accelerations and decelerations varied with stimulus presentation and were accompanied by consistent and marked increases in the range of heart rate activity.

These data on four previously malnourished children as compared to their controls suggested that habituation to pure tones might be sensitive to differences in attentional processes between adequately nourished and poorly nourished infants. These data were promising in that the differences were obtained even though the experimental subjects had been physically rehabilitated from malnutrition.

However, given the small number of subjects and the large variability in the ages, it was decided to conduct a second pre-test. In addition, the second pre-test was designed to test a louder tone volume as it was felt that 65 db was not sufficiently intense. Finally, the question of whether or not there are differences in pre-stimulus or basal heart rate between well and poorly nourished infants was examined in Pilot Study II.

Pilot Study II

Method

The subjects for this study were 16 male infants with a mean age of 13 1/2 months drawn from institutions in Guatemala City. Eight of these infants were malnourished at the time of admittance to the institution and were supposedly rehabilitated although according to the Gomez (1956) system, they were suffering from second and third degree malnutrition. The eight remaining babies were better nourished, suffering from first degree malnutrition (Gomez, 1956). Table 2 shows the means for weight, height, head circumference and age for the relatively well nourished and malnourished groups.

The experimental procedure involved 20 trials of a 750 Hz pure tone followed by 10 trials of a 400 Hz tone followed by 10 trials of a 750 Hz tone. The tones were presented at 90 db for five seconds with the ITI randomized as in Pilot Study I.

Results and Discussion

To compare pre-stimulus heart rate between the experimental and control groups, the mean heart rate during the 10 seconds preceding the presentation of the first

TABLE 2.--Age and Anthropometry of Infants in Pilot Study II.

Groups	Age (Mos.)	Weight (lbs.)	Height (cms.)	Head Circumference (cms.)
Well Nourished	$\bar{X}$=13.75	$\bar{X}$=17.40	$\bar{X}$=71.85	$\bar{X}$=44.57
(N=8)	SD=7.81	SD=3.28	SD=5.81	SD=2.07
Malnourished	$\bar{X}$=13.25	$\bar{X}$=14.71	$\bar{X}$=68.42	$\bar{X}$=43.14
(N=8)	SD=3.10	SD=3.03	SD=5.34	SD=1.46

stimulus was calculated for each group. The basal heart rates were, for the experimental group; $\bar{X}$ = 144.64, SD = 3.57; and for the control group; $\bar{X}$ = 137.47, SD = 5.11. A *t* test revealed no significant difference in prestimulus heart rate between the two groups [$t(14) = 1.40$].

Figure 1 presents the heart rate decelerations to the pure tone stimuli for the well and malnourished infants. The results showed that the mean HR deceleration on trial 1 was larger for the well nourished infants than for the malnourished infants [$t(14) = 3.67$, $p < .01$]. Likewise, the well nourished group showed larger mean HR decelerations than the malnourished group on the first and second dishabituation trials; trials 21 and 31 [$t(14) = 1.76$, $p < .05$ and $t(14) = 1.77$, $p < .05$, respectively].

Furthermore, whereas the well nourished infants clearly responded to the changes in tonal frequency, the malnourished infants did not. Within group comparisons between each dishabituation trial and the immediately preceding trial revealed that the well nourished group showed a greater mean HR deceleration on trial 21 than on trial 20 [$t(7) = 2.16$, $p < .05$] and on trial 31 than on trial 30 [$t(7) = 2.18$, $P < .05$]. For the malnourished infants, these differences were not significant.

These findings lend further support to the use of the habituation procedure for investigating the effects of nutritional insult. Specifically, the OR appeared to be

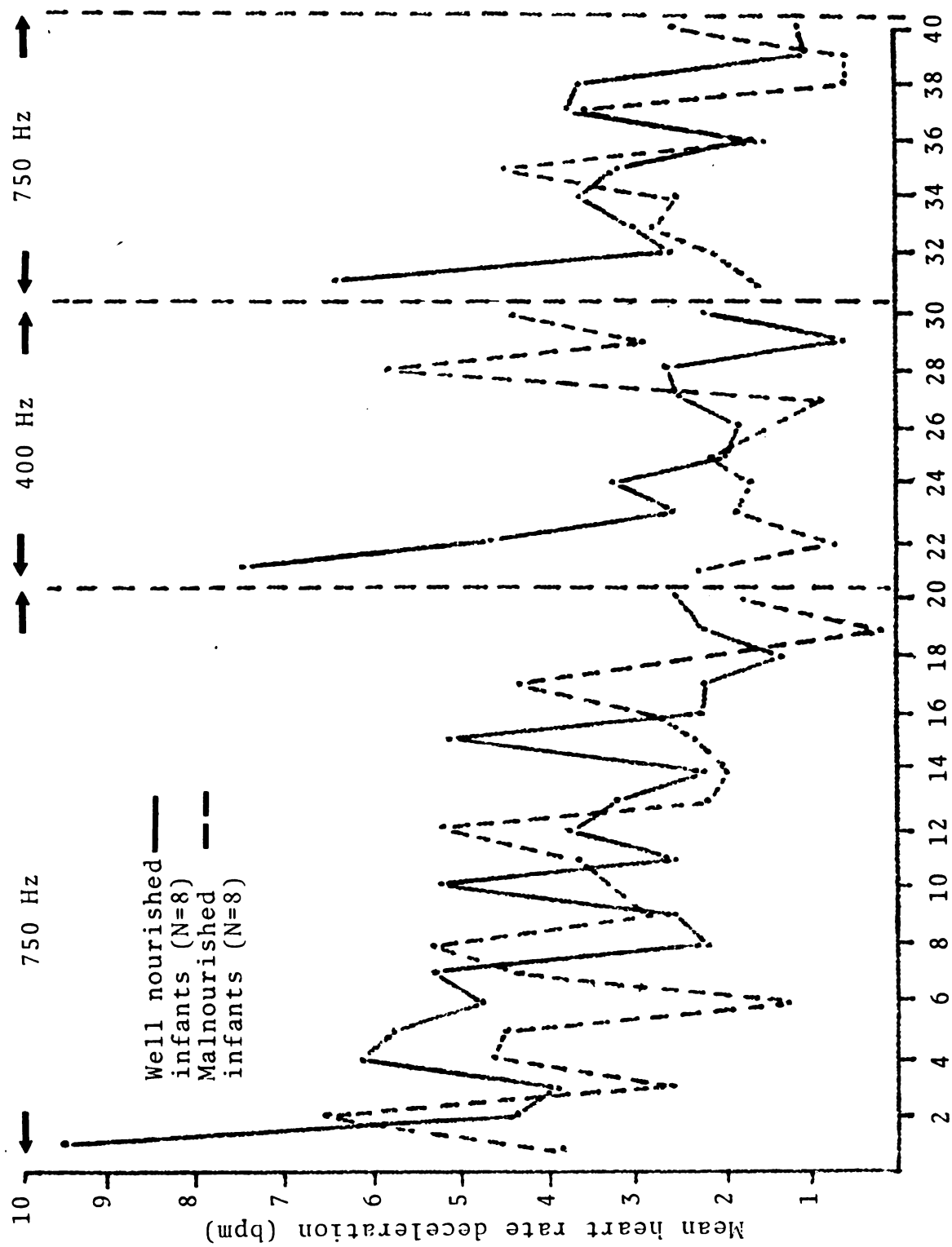

Figure 1.--Heart Rate Deceleration to Pure Tone Stimuli for Infants in Pilot Study II.

attenuated or completely absent in infants suffering from malnutrition. Furthermore, this attenuation of the OR persisted from the first presentation of a pure tone to subsequent changes in the frequency of the tone. However, because of the possible confounding effects of institutionalization and the relatively small sample size a more comprehensive study was planned. In addition, another pre-test was planned to investigate HR recovery time.

Pilot Study III

Method

The third pilot study was conducted to determine HR recovery time as it was not clear from the preceding studies whether or not the ITI was sufficiently long to allow for full HR recovery following each stimulus presentation. For this experiment, 5 well nourished and 5 poorly nourished one-year-old girls were presented with 10 trials of a 750 Hz pure tone at 90 db with a 30-second ITI.

Results and Discussion

HR recovery was calculated by dividing each 30-second ITI into six time blocks of 5 seconds each and comparing the mean HR during each time blocks with the mean HR during the 5 seconds preceding the stimulus. For example, the mean HR during the 5 seconds preceding trial 1 was compared with the mean HR for the six 5 second periods following trial 1.

The results showed, first, that each subject showed a fairly consistent HR recovery. The range of HR recovery time was usually within 8 seconds for any given subject across the 10 trials. Second, there were no differences in HR recovery time between the poorly nourished and adequately

nourished groups. The mean time for HR to reach the pre-stimulus level and remain steady at that level was approximately 15 seconds for both groups with a range of 7 to 22 seconds.

The results of this experiment suggested that the ITI should be increased over that used in the previous experiments. Accordingly, it was decided to randomize the ITI with a mean of 20 seconds and a range of 15 to 25 seconds. This allowed ample time for HR recovery before the onset of the next stimulus. Furthermore, this ITI was appropriate for both well and malnourished infants as HR recovery time did not differ between these groups.

However, as a result of lengthening the ITI to a mean of 20 seconds, the procedure consisting of 40 trials as in pilot study II could not be used as the experimental session would be too long. The length of the experimental session of pilot study II was approximately 15 minutes and several babies cried toward the end of the session. Given that these subjects were drawn from the same sample as those selected for the main study it was decided to limit the experimental session to 10 minutes. For this reason, the design for the main study called for 20 rather than 40 trials.

Two malnourished infants from the same sample were administered the 40 trial procedure as in pre-test 2 but with the lengthened ITI of mean 20 seconds. Both babies

cried shortly after the 20th trial (approximately 10 minutes). In addition, an analysis of HR data for these two babies showed no substantial changes in HR activity in the latter trials within each tone sequence. The final design is detailed below.

Main Study

Method

Subjects

The subjects were forty, one year old, male infants equally divided among two nutritional status groups. Subjects were recruited through State run medical clinics in Guatemala City, Guatemala. These clinics are located in the slums or "barrios" providing free prenatal, perinatal and postnatal care for those families, all of whom are extremely poor, living in the "barrios." Mothers who brought their infants to one of these clinics for a regular check-up were contacted by a Guatemalan research assistant. The study was briefly explained to the mothers and those who consented to participate were asked a series of questions to assess the social-economic characteristics of the family and the medical history of the infant.

Presented in Table 3 are the social-economic characteristics of the sample. As can be seen from Table 3, there were no differences between the well and malnourished infants with respect to the age or education of the parents or the occupation of the father. The parents were 25 to 30 years of age and received approximately four years of formal education. The fathers were mostly unskilled or semi-skilled

TABLE 3.--Social-economic Characteristics of Infants in Main Study.

Group	Age				Years of School				Frequency of Type of Occupation of the Father		
	Mother		Father		Mother		Father				
	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	Unskilled	Semi-skilled	Skilled
Well Nourished	23.90	4.06	32.29	9.38	4.20	2.26	4.52	2.29	8	5	7
Malnourished	24.00	4.67	29.70	7.44	3.73	2.05	3.88	2.44	8	6	6

laborers with typical occupations including factory workers, masons, messengers, shoe repairmen, tailors, and farm laborers.

Information concerning the income of the family was not collected because pre-testing found mothers extremely reluctant to divulge these data. In addition, because of the tremendous variability in income among those mothers who did consent to answer, it was felt that the income measure was unreliable and only served to intimidate the mothers. Thus, the social-economic status of the sample was controlled by the fact that all families lived in the "barrios" and therefore attended the free medical clinics, and by the similarity among the well and malnourished groups with respect to the age and education of the parents and the occupation of the father.

The medical histories of the infants in the sample revealed that all infants were born at full gestation with no prenatal, perinatal or postnatal complications. All births were single with no differences between the well nourished groups in mean birth weight ($\bar{X}$ = 6.54, SD = .93 for the well nourished group, $\bar{X}$ = 6.42, SD = .96 for the malnourished group). For the mothers of the malnourished infants, 70% received prenatal care and 95% breast fed their infants. For the mothers of the well nourished group, 75% received prenatal care and 85% breast fed their infants. Finally, at the time of testing, 57% of the malnourished

infants were still being breast fed whereas 47% of the well nourished infants were being breast fed.

Determination of Nutritional Status

Nutritional anthropometry is concerned with measures of physical growth as indices of variation in nutritional status. The most common measures are weight, height and head circumference (Jelliffe, 1966). Weight is considered to be the key anthropometric measurement as loss of body weight is usually the first anthropometric sign of malnutrition, particularly at younger ages (Jelliffe, 1966). Nutritional status is determined by comparing measured anthropometry from a sample with known standards of reference, if local standards are not available. These general standards are expressed as a percentage of the norm for a given age.

For the present study, the individual weights of the Guatemalan infants were compared with the Harvard norms (Jelliffe, 1966) and the norms obtained from Mexico City (Gomez, 1956). In addition, Gomez (1956) provided a system for categorizing degree of malnutrition. For first degree malnutrition, body weight is between 76%-90% of the theoretical average of the child's age. For second degree malnutrition, the weight is between 61%-75% of the average age, and for third degree malnutrition weight is not more than 60% for the average age.

To determine whether or not body weight was sufficiently distributed among lower class Guatemalan infants

to select a well and a malnourished group for testing, anthopometric data was collected for 134 infants. These infants were nine- to eleven-month-old males and females and were measured at the medical clinics described above. Of the 134 infants, twenty-six were excluded as they did not meet one or more of the medical criteria outlined above, the major reason for exclusion being low birth weight. Presented in Figure 2 is the frequency distribution of the weight for age values for the remaining 108 infants according to the Gomez (1956) norms. This distribution is virtually identical to that obtained from the Harvard Standards (Jelliffe, 1966). From this sample forty male infants were selected for testing. Table 4 shows the mean values for age, weight, height, and head circumference for the well and malnourished groups. As can be seen from this table, the malnourished infants were significantly lighter, shorter, and had smaller head circumferences than did the well nourished infants. The mean percent of average weight for age was 76.4% (range of 56%-75%) for the malnourished infants and 94.4% (range of 89%-100%) for the well nourished infants. According to the Gomez (1956) system, the malnourished infants were suffering from second and third degree malnutrition whereas the well nourished infants were within the average expected weight for age.

The anthropometry information was collected by two Guatemalan research assistants who had been previously

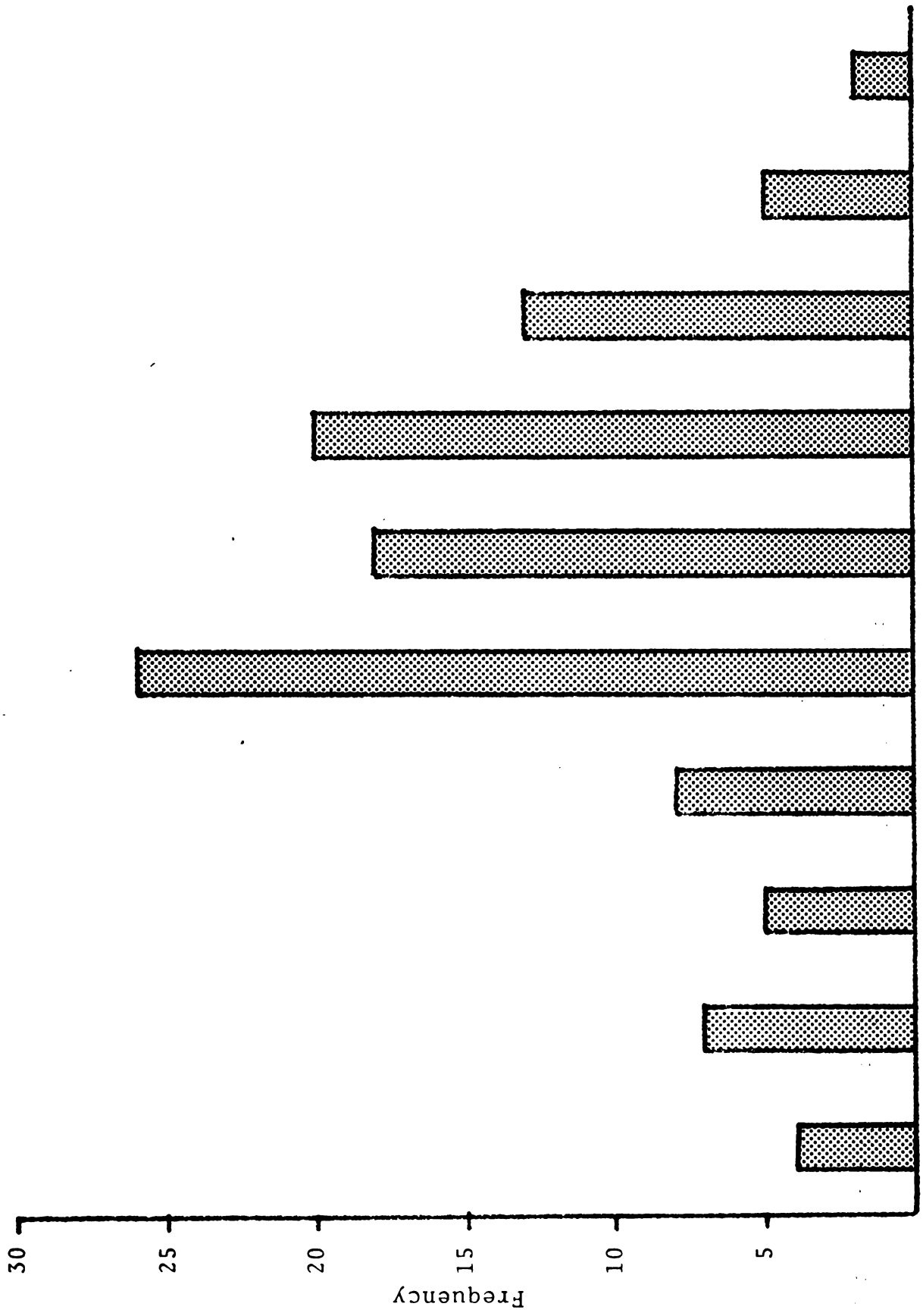

Figure 2.--Percent Weight for Age Distribution for 108 Lower Class Guatemalan Infants.

TABLE 4.--Age and Anthropometry of Infants in the Main Study.

Measure	Well Nourished (N=20)	Malnourished (N=20)	F (df=1,38)
Age (mos.)	$\bar{X}$ = 12.38 SD = 1.30	$\bar{X}$ = 12.28 SD = 1.92	00.05
Weight (lbs.)	$\bar{X}$ = 20.76 SD = 1.92	$\bar{X}$ = 15.44 SD = 1.42	98.44 $p < .001$
Height (cms.)	$\bar{X}$ = 72.19 SD = 2.87	$\bar{X}$ = 67.77 SD = 2.89	23.50 $p < .001$
Head Circumference (cms.	$\bar{X}$ = 45.48 SD = 1.34	$\bar{X}$ = 43.87 SD = 1.35	14.29 $p < .001$

trained to make these judgments. The reliability of the two assistants was established by comparing their percent of agreement for ten of the infants. The percentages of agreement for the three anthropometric measures were 100% for weight (±.10 lb), 90% for height (±.5 cm) and 90% for head circumference (±.5 cm).

Procedure

Mother and infant were brought to the infant laboratory at the Institute of Nutrition of Central America and Panama (INCAP) in Guatemala City. The study was explained to the mother in detail in a waiting room where the electrodes were also attached to the infant. Mother and infant were then brought into a white, sound attenuated experimental chamber (2.9m x 1.9m x 2.0m) where the infant was seated on the mother's lap. The auditory stimulus was delivered from a speaker mounted on the wall .72m directly above the infant's head. The mother did not hear the stimulus as she listened to pre-recorded music through a set of head phones. Also, in the experimental room were two coders seated behind a partition with a one-way screen opposite the infant and at a distance of 1.8m.

The experimental design is shown in Table 5. Subjects were randomly assigned to one of two orders of stimulus presentation. Order A consisted of 10 trials of a 750 Hz pure tone followed by 5 trials of a 400 Hz pure tone, again followed by 5 trials of a 750 Hz pure tone. Order B con-

TABLE 5.--Experimental Design of Main Study.

Groups	Adaptation	Experimental Session Sequence I Trials 1-10	Sequence II Trials 11-15	Sequence III Trials 16-20	Order of Stimulus Presentation
10 Well Nourished Males	30 seconds basal HR	750 Hz pure tone at 90db.	400 Hz pure tone at 90db.	750 Hz pure tone at 90db.	Order A
10 Mal-nourished Males	30 seconds basal HR	750 Hz pure tone at 90db.	400 Hz pure tone at 90db.	750 Hz pure tone at 90db.	
10 Well Nourished Males	30 seconds basal HR	400 Hz pure tone at 90db.	750 Hz pure tone at 90db.	400 Hz pure tone at 90db.	Order B
10 Mal-nourished Males	30 seconds basal HR	400 Hz pure tone at 90db.	750 Hz pure tone at 90db.	400 Hz pure tone at 90db.	

sisted of 10 trials of the 400 Hz tone, 5 trials of the 750 Hz and 5 trials of the 400 Hz tone. All tones were produced at 90 db by a General Audiometric Oscillator. The stimulus was presented for 5 seconds, using Lafayette timers. The ITI was randomized automatically with a mean of 20 seconds and a range of 15 to 25 seconds through the combined use of a Lafayette Probability Generator and Lafayette Counter.

The dependent measure of HR was monitored with a set of Beckman bio-potential electrodes placed slightly above the infant's nipples and a ground located above the navel. Prior to stimulus administration, 30 seconds of basal heart rate was collected to adapt the subject to the situation. Cardiac responses were recorded in an adjoining room with a Beckman type-R dynograph with cardiotachometer. Behavioral observations were recorded on the polygram simultaneously with the physiological activity. Behavioral observations were coded by manually pressing buttons on the button box. These observations were coded by one observer in the experimental room according to pre-defined behavioral categories. The other observer collected similar data for inter-observer reliability. Four behaviors were coded, one involving the infant's orientation to the source of the stimulus. The other three were measures of the state of the infant and were adapted from Brackbill & Fitzgerald (1969). The four categories are described as follows:

1. Behavioral Orientation Reaction (BOR): The infant's head and/or eyes are turned toward the stimulus.

2. Awake: The infant appears generally happy and peaceful. There is little to moderate gross motor activity, i.e. movements involving the whole body. The eyes are characterized by a bright, shiny appearance; respiration is relatively regular and vocalizations are not of an "unhappy" variety.

3. Drowsy-Sleep: During the drowsy state the infant's body relaxes as he gradually falls asleep, then suddenly jerks awake. The eyelids flutter and the eyes, when visible, have a glassy appearance. As the infant falls asleep, the body is more relaxed, eyes are usually closed and respiration is slow and regular. Occasional body movements in the extremities and in the face, such as twitches, grimaces, smiling, sucking, etc., may be observed. In addition, one can sometimes see conjugate movements of the eyeballs.

4. Fuss-Cry: During the fuss state, there is a considerable amount of gross motor activity. The infant may writhe or squirm, respiration becomes irregular and vocalizations are those of the cranky, fuss variety. In the cry state, these same behaviors continue and may become exaggerated.

In all, forty-two infants were brought to the laboratory for testing. Two infants cried throughout the experi-

ment and were not included in the sample. Each of the forty infants who comprised the sample received the entire twenty trial procedure. While the sleep state never occured, some infants did fuss or cry. Fussing or crying occured 13 times out of the 400 trials for the well nourished group and seven times out of 400 trials for the malnourished group. This represented 3% and 1% of the trials respectively. Since these trials were dropped from all subsequent analyses (a total of 2%), the data described below are those for those trials in which all infants were in the awake state.

Inter-observer reliability between the two observers was collected for 15 of the infants. The percentages of agreement were 91% for the BOR, 100% for awake, and 100% for fuss-cry. As mentioned previously, the sleep state did not occur. These percentage agreements are similar to those reported by Brackbill and Fitzgerald (1969).

Data Reduction

Three indices of heart rate activity were computed for each trial: heart rate deceleration, heart rate acceleration and heart rate variability. Heart rate deceleration was calculated as the mean of the two lowest beats during the five second pre-stimulus period minus the mean for the lowest two beats during the five second stimulus presentation period. Heart rate acceleration was the difference between the mean of the two highest beats

during the pre-stimulus period minus the mean of the two highest beats during the stimulus presentation. To calculate changes in heart rate variability, the range for the stimulus presentation period (mean of the two highest beats minus the mean of the two lowest beats during stimulus presentation) was subtracted from the base range (mean of the two highest beats minus the mean of the two lowest beats for the pre-stimulus period). Reliability of the scoring of these measures was determined for five of the infants. The two assistants reached 99% agreement for each HR measure (± 1 beat per minute).

For the BOR measure, two variables were calculated, the duration of the BOR in seconds for each trial and a simple count of the number of babies who evidenced the BOR for each trial. Inter-scorer reliability for these were 92% and 99% respectively.

Results

To test for the effects of the order of stimulus presentation, the three dependent HR variables were subjected to an Order X Trials analysis of variance (ANOVA) with repeated measures over the last factor for each of the three trial sequences for each nutrition group. These sequences are referred to as I (trials 1-10), II (trials 11-15), and III (trials 16-20). Thus, for tone sequence I the ANOVA took the form 2 (Order) X 10 (Trials) whereas for

the sequences II and III a 2 (Order) X 5 (Trials) ANOVA was used. The results showed no effects due to order of stimulus presentation in either the well or malnourished group for any of the HR measures.

For the well nourished group HR decelerations to sequence I revealed a significant trials effect [$F(9,144) = 8.82$, $p < .001$] with no significant order effect or order by trials interaction. Significant main effects for trials were also found for sequences II [$F(4,64) = 12.35$, $p < .001$] and III [$F(4,60) = 6.66$, $p < .001$] with no significant order or order by trials interaction. [The results are presented in Appendix Tables 1-3.]

For the malnourished infants, HR decelerations to the three tone sequences also revealed no significant main effect for the order factor. However, the main effect for trials as well as the order by trials interaction were also not significant for any of the tone sequences. [Tables 4-6 of the Appendix contain the results of these analyses.] These data show that HR decelerations were not affected by the order of stimulus presentation for either the well or the malnourished groups. However, whereas the well nourished infants showed a significant response decrement to each of the three tone sequences, HR decelerations among the malnourished infants did not change across the repeated presentations of any tone.

For the measure of HR acceleration in the well nourished group sequence I revealed a significant trials

effect [$F(9,144) = 2.36$, $p < .025$] with no significant order effect or order by trials interaction. The significant trials effect was due to an increase in HR accelerations during the latter trials in sequence I. For sequences II and III HR accelerations showed no significant main effects or interactions. [The results are presented in Appendix Tables 7-9.] For the malnourished group no significant main effects or interactions were found for any of the tone sequences. [See Appendix Tables 10-12.] Thus, HR accelerations were also not affected by the order of stimulus presentation either among the well or malnourished infants.

Similar results were obtained for the HR variability measure. The results for the well nourished group revealed no order effects for any of the tone sequences although each sequence did reveal significant main effects for the trials factor [$F(9,144) = 3.02$, $p < .05$ for I; $F(4,64) = 12.96$, $p < .001$ for II; and $F(4,60) = 5.80$, $p < .001$ for III]. None of the order by trials interactions were significant. [See Appendix Tables 13-15.] For the malnourished group, there were no significant main or interaction effects for any tone sequence for the HR variability measure. [These results are presented in Appendix Tables 16-18.]

The results for the three HR measures showed that cardiac activity was not affected by the order of stimulus

presentation for either the well or the malnourished groups. The data also showed that for well nourished infants, there was a decrease in the magnitudes of both HR decelerations and HR variability for each tone sequence and a slight increase in the magnitude of HR accelerations during the last few trials of sequence I. In contrast, no change in HR activity was found among the malnourished infants for any of the tone sequences.

These differences were explored further by comparing cardiac responses between the two groups within each tone sequence with the order conditions collapsed. For sequence I the statistic was a 2 (Nutrition) X 10 (Trials) ANOVA with repeated measures over the last factor. For the other tone sequences the repeated measures ANOVA was a 2 (Nutrition) X 5 (Trials) model.

Figure 3 presents the mean HR decelerations to the pure tone stimuli in the well and malnourished groups. The ANOVA for sequence I revealed a significant trials effect [$F(9,324) = 3.42$, $p < .001$] and a significant trials by nutrition interaction [$F(9,324) = 5.61$, $p < .001$]. [See Appendix Table 19.] These effects were due to differences in the magnitude of HR decelerations on the first trial between the two groups. This is evidenced by the fact that when the ANOVA was performed on trials 2-10 no significant main or interaction effects were found. [See Appendix Table 20.] In addition, an ANOVA for trials 6-10 showed

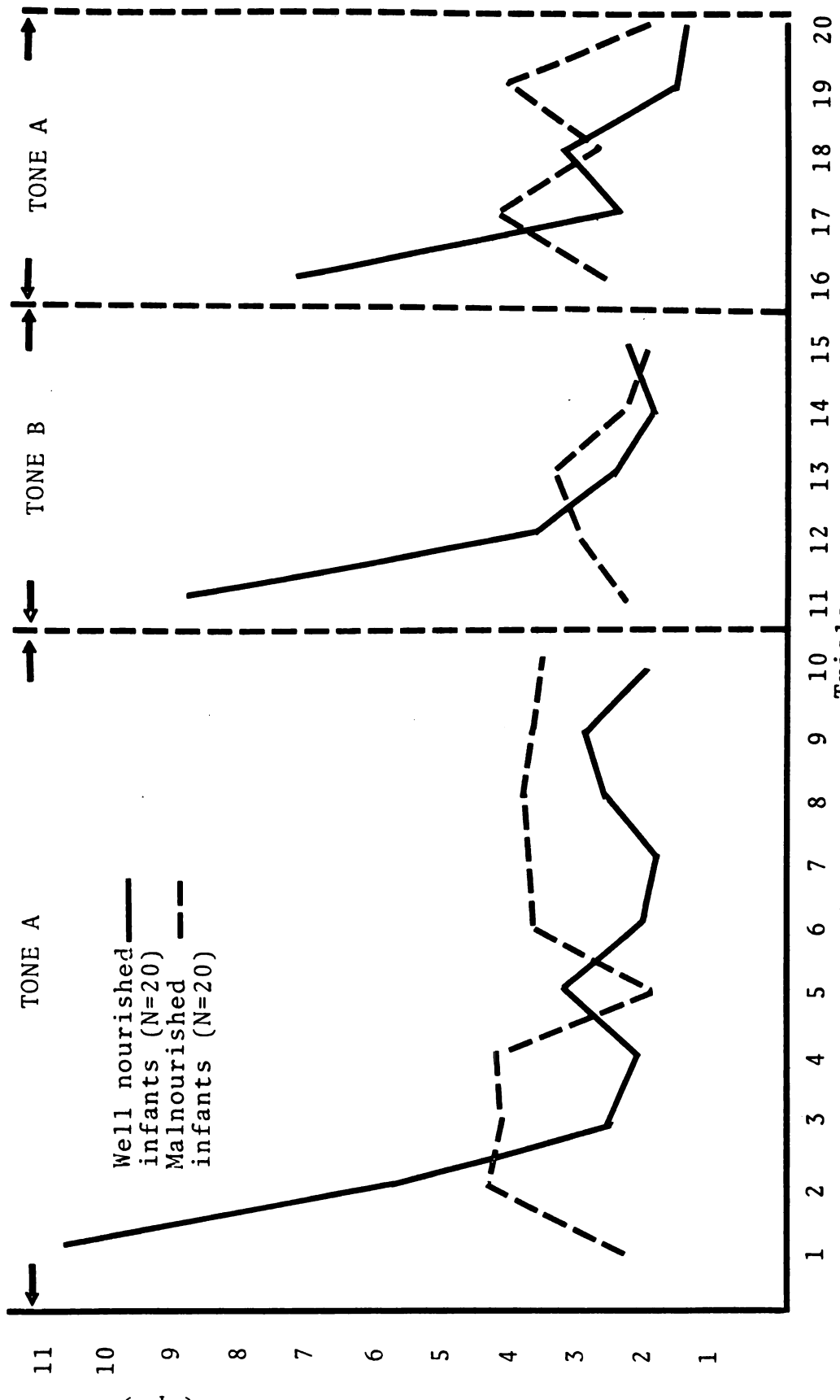

Figure 3.--Heart Rate Decleration to Pure Tone Stimuli for Infants in Main Study.

a significant main effect for the nutrition factor [$F(1,37) = 5.36$, $p < .05$] with no significant main or interaction effects. [See Appendix Table 21.] As can be seen in Figure 3, the mean decelerations on trials 6-10 were larger in the malnourished than in the well nourished group. This reflects the fact that the well nourished infants had habituated by the fifth trial whereas the malnourished infants maintained the same level of responding throughout the entire tone sequence.

This same pattern of results emerged from the ANOVAs for sequences II and III. For sequence II the analysis revealed a significant trials effect [$F(4,132) = 3.98$, $p < .001$] and a significant nutrition by trials interaction [$F(4,132) = 5.52$, $p < .001$]. [See Appendix Table 22.] The ANOVA for trials 12-15 showed no significant main or interaction effects suggesting that the differences were accounted for by the differences in the magnitude of the decelerations on trial 11 between the two groups. [See Appendix Table 23.] Similarly, the ANOVA for sequence III revealed significant effects for the trials factor [$F(4,132) = 3.32$, $p < .025$], and the nutrition by trials interaction [$F(4,132) = 4.78$, $p < .001$]. [See Appendix Table 24.] This was due to the larger deceleration in the well nourished groups on trial 16 as an ANOVA for trials 17-20 showed no main or interaction effects. [See Appendix Table 25.]

The responses to the two dishabituation stimuli (trials 11 and 16) were examined by comparing the mean HR deceleration of each dishabituation stimulus with the mean deceleration of the immediately preceding trial. For the well nourished infants, the mean HR deceleration was larger on trial 11 than on trial 10 [$F(1,18) = 27.88$, $p < .001$] and was larger on trial 16 than on trial 15 [$F(1,16) = 32.88$, $p < .001$]. For the malnourished infants, no significant increase in the magnitude of HR deceleration was found to either dishabituation stimulus.

As is clear from Figure 3, HR decelerations to the pure tone stimuli were characterized by large initial OR's followed by rapid habituation among the well nourished infants. On the other hand, the malnourished infants showed an attenuation or absence of the OR which was unaffected by further presentations of the same stimulus.

Figure 4 shows the mean HR accelerations to the pure tone stimuli in the two groups. The ANOVA for sequence I revealed a significant nutrition by trials interaction [$F(9,324) = 2.18$, $p < .05$] with no significant main effects for either the nutrition or trials factors. [See Appendix Table 26.] The interaction was due to an overall increase in the magnitude of HR accelerations during the last five trials of sequence I in the well nourished group. An ANOVA for these trials showed a significant trials effect [$F(1,37) = 4.19$, $p < .05$] with no other significant main or interaction

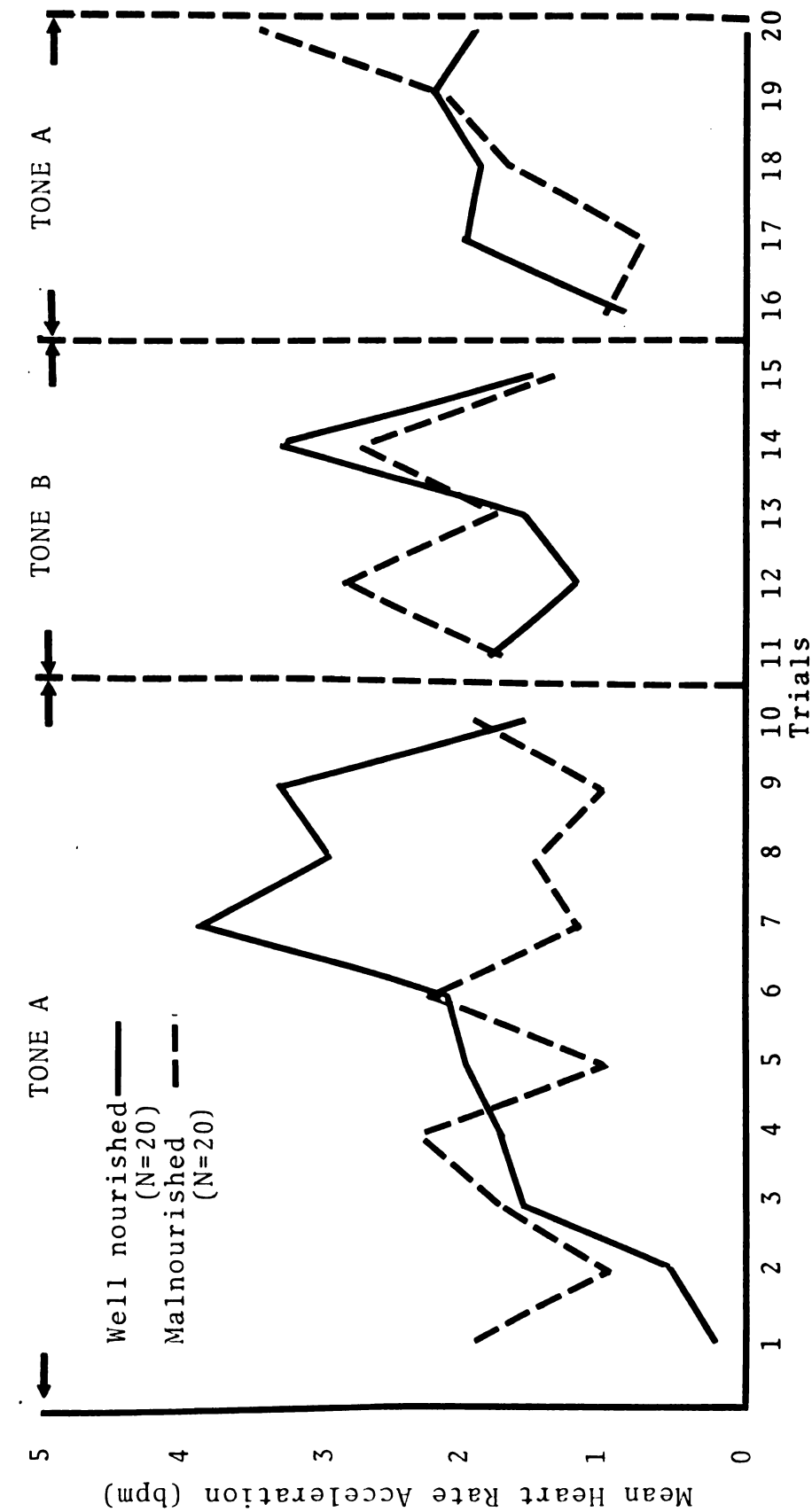

Figure 4.--Heart Rate Acceleration to Pure Tone Stimuli for Infants in Main Study.

effects. [See Appendix Table 27.] This increase in HR accelerations during the last five trials was probably due to the fact that the well nourished infants had already habituated to the stimulus and were growing restless. For sequences II and III main or interaction effects were not found to be significant between the two groups. [See Appendix Tables 28-29.] Also, the two dishabituation trials (11 and 16) produced no significant increase in mean HR acceleration from the previous trial in either group.

The results for the HR variability measure are presented in Figure 5. The ANOVA for sequence I showed a significant main effect for trials [$F(9,324) = 2.27$, $p < .025$] with no significant main or interaction effects. [See Appendix Table 30.] The elimination of the first trial from the analysis, however, resulted in no main or interaction effects. This suggested that the response decrement in variability was due to group differences in the magnitude of HR change on trial 1. [See Appendix Table 31.] For sequence II, significant effects were found for the trials factor [$F(4,132) = 5.54$, $p < .001$] and the nutrition by trials interaction [$F(4,132) = 4.40$, $p < .005$]. [The results are shown in Appendix Table 32.] Again, an ANOVA for trials 12-15 showed no significant main or interaction effects. [See Appendix Table 33.] Similarly for the last tone sequence none of the main effects were significant although there was a significant nutrition by trials interaction [$F(4,132) =$

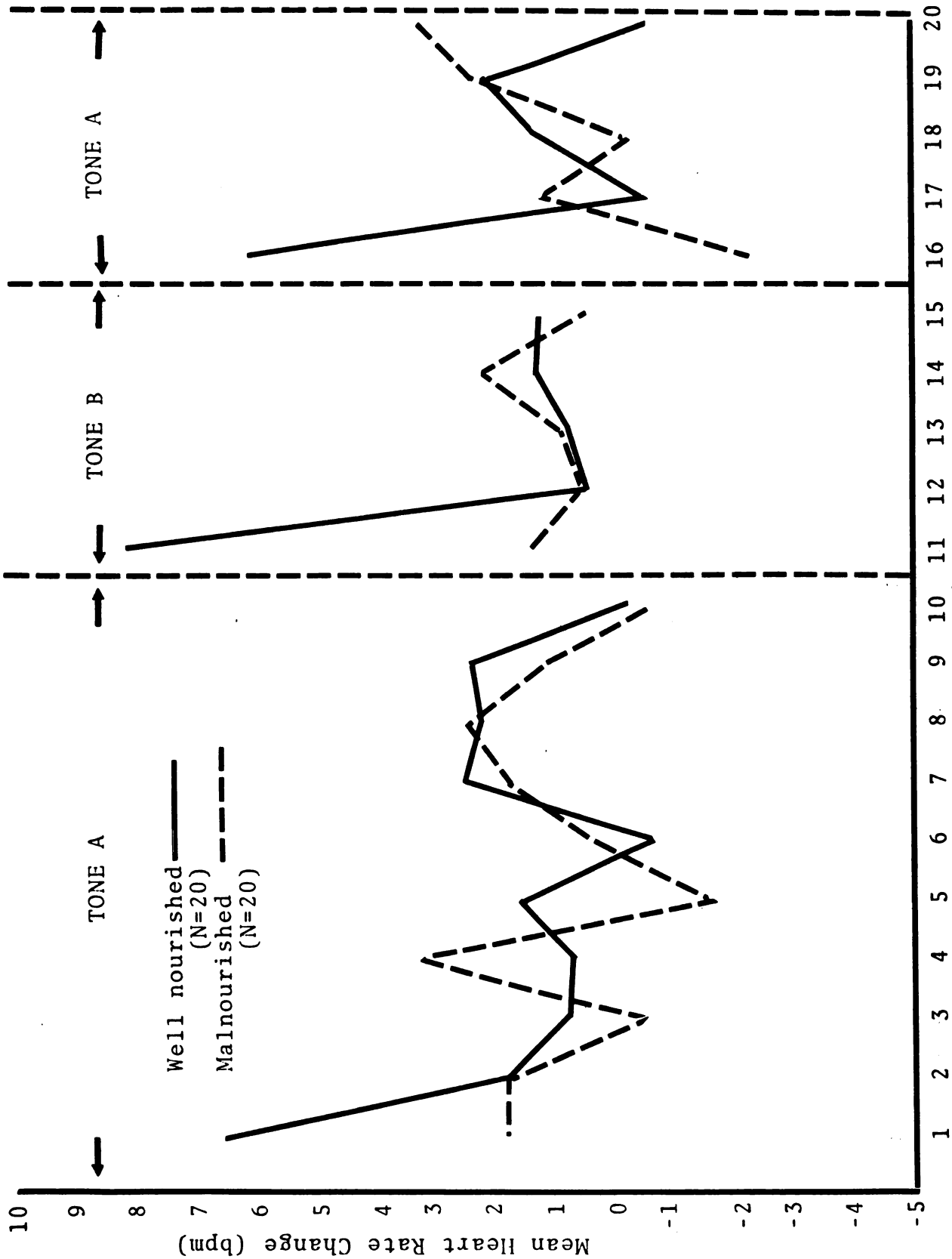

Figure 5.--Heart Rate Variability to Pure Tone Stimuli for Infants in Main Study.

5.24, $p < .001$]. [See Appendix Table 34.] When the ANOVA was performed over trials 17-20, the interaction effect was no longer significant. [This can be seen from Appendix 35.]

Analysis of responses to the dishabituation stimuli revealed a significant increase in HR variability from trial 10 to trial 11 [$F(1,18) = 40.79$, $p < .001$] and from trial 15 to trial 16 [$F(1,16) = 6.41$, $p < .025$] for the well nourished group. For the malnourished group similar comparisons were not significant.

The results of the HR variability measure are similar to those found for HR deceleration. For the well nourished infants the reaction to the first presentation of the stimulus in each sequence was characterized by a substantial increase in HR activity from the pre-stimulus to the stimulus onset period. Over repeated trials, however, changes from pre-stimulus to stimulus onset periods habituated. The malnourished infants, on the other hand, showed no changes in HR activity from pre-stimulus to stimulus onset periods across any trials.

The relation among the three HR measures are presented in Table 6. Within each tone sequence significant correlations (Pearsonian) were found between all cardiac measures for both the well and the malnourished infants. Increases in the magnitudes of both HR decelerations and accelerations were associated with increases in HR activity

TABLE 6.--Correlations Among Heart Rate Measures by Tone Sequence for Infants in Main Study.

Group	Variable	Tone Sequence Sequence I Decel	Acel	Var	Sequence II Decel	Acel	Var	Sequence III Decel	Acel	Var
Well Nourished	Decel	-	-.24**	.48**	-	-.25**	.58**	-	-.29**	.54**
	Acel	-	-	.33**	-	-	.22*	-	-	.22*
Mal-nourished	Decel	-	-.34**	.47**	-	-.34**	.57**	-	-.32**	.47**
	Acel	-	-	.18*	-	-	.25*	-	-	.21*

* p .05
** p .01

from pre-stimulus to stimulus onset periods. That is, HR change in general was related to both larger decelerations and accelerations to the stimuli. In addition, the measure of deceleration and acceleration are consistently negatively correlated within each tone sequence for both the well and malnourished groups.

A final note with respect to the HR data has to do with the possibility that any observed differences might be a function of differences in basal autonomic activity. This was tested by comparing the mean HR during the ten seconds preceding the first trial between the well and malnourished groups. Analysis of variance showed that pre-stimulus HR was not significantly different between the well nourished ($\bar{X}$ = 134.32, SD = 12.37) and the malnourished ($\bar{X}$ = 138.95, SD = 13.28) infants.

The final dependent variable was the BOR measure. This response was evidenced by relatively few infants on each trial. Furthermore, responses across trials tended to occur in the same infants. Figure 6 presents, for each trial, the percentage of well and malnourished infants who responded. As can be seen from Figure 6, the only trial on which at least half of the infants responded was the first trial for the well nourished group. This response habituated and showed some recovery on the two dishabituation trials, 11 and 16. The malnourished infants never responded above the 10% level on any trial. Thus, while the BOR

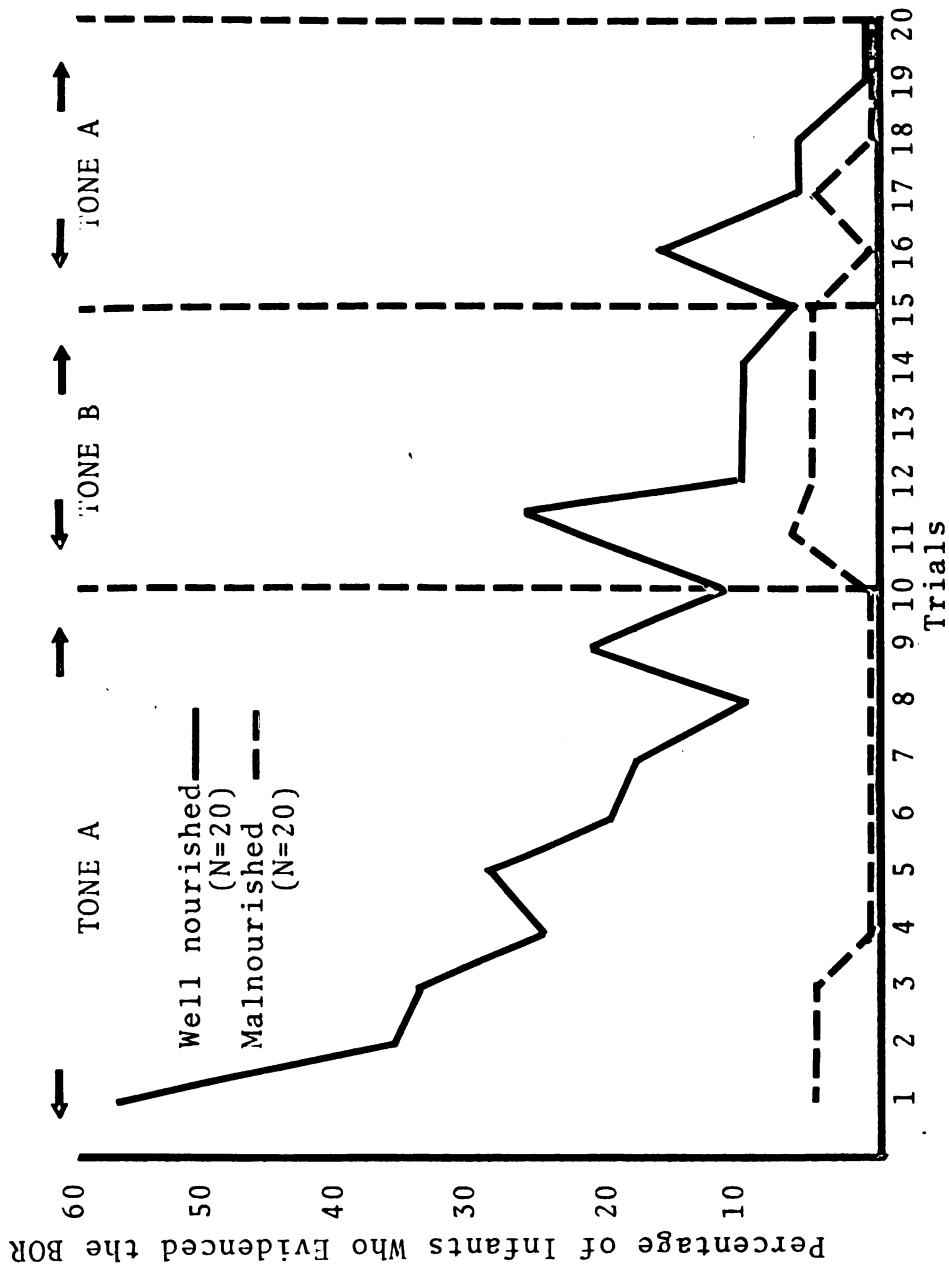

Figure 6.--Percentage of Infants in Main Study who Showed the Behavioral Orientation Reactor (BOR).

measure showed a general lack of responsivity among both groups, an inspection of the figure still shows differences in BOR magnitude and habituation between the well and malnourished infants.

A related issue is whether or not the magnitude of HR decelerations is greater among infants who evidence a simultaneous BOR than among those who do not. A *t* test was calculated between the mean HR deceleration among the well nourished infants on trial 1 who showed the BOR and the mean HR deceleration on trial 1 among the well nourished infants who showed no BOR. These means were 11.54 for the BOR group (SD = 4.30) and 8.75 for the non BOR group (SD = 3.24) and were not significantly different.

Discussion

The results from this study reveal a clear nutritional effect on the infants ability to respond to and process impinging novel stimulation. The well nourished infants showed a classic habituation reaction to the repeated presentation of a novel stimulus followed by dishabituation and subsequent rehabituation to qualitative changes in that stimulus. The initial presentation of the tone resulted in a large OR which diminished rapidly. Response recovery, or dishabituation occurred to the two sequences in which the frequency of the tone was changed with the intensity of the tone at a constant level. Response decrement following the two dishabituation stimuli was also

rapid suggesting that the habituation of the OR in the well nourished group was due to a neural inhibitory process rather than to effector or receptor fatigue.

These results are similar to those reported by Lewis and Spalding (1967) in which HR decelerations averaged nine bpm to the initial presentation of an auditory stimulus and Moffitt (1968) who reported decelerations of up to twenty bpm in response to speech sounds. The decelerative responses in these two studies were obtained in six month old infants. Studies with older infants have reported similar HR changes to auditory stimuli although these studies have been criticized for their method of data reduction (Graham & Jackson, 1970).

In contrast to the classic habituation response of the well nourished infants, the malnourished infants showed a general lack of responsivity. HR decelerations did not change following the repeated presentation of the tones. This lack of responsivity can be best described as an attenuation or, in some cases, a complete absence of the orienting response. HR decelerations on the initial trial of each tone sequence did not differ from those on subsequent trials within the same tone sequence. Nor was there any evidence of dishabituation to the changes in tonal frequency. Deceleration responses to the dishabituation stimuli were not different from responses immediately preceding the presentation of these stimuli. The only instance

in which HR decelerations were larger among the malnourished than among the well nourished group was during the last five trials of the first tone sequence. This was due to the fact that the well nourished infants had essentially stopped responding or habituated to the tone whereas the malnourished infants were still manifesting the same minimal level of deceleration on trials six through ten as they did on trials one through five.

That the well nourished infants had habituated to the tone by thr fourth is also evidenced by the HR acceleration data. The only trials on which differences between the two groups were found for the acceleration response were these same trials, five through ten. Here the well nourished infants showed an increase in accelerations across the same trials. It seems likely, therefore, that this increase in accelerations for the well nourished infants was due to the fact that they had already habituated (as evidenced by the deceleration data).

The notion that HR acceleration reflects stimulus rejection and HR deceleration stimulus intake has been suggested by several investigators (e.g. Lacey, 1959). The correlation results obtained from the present study further supports the notion that accelerations and decelerations do indeed measure different processes. As shown in Table 6 the correlations between accelerations and decelerations within each tone sequence for both the

well and malnourished infants were in a negative direction. This also indicates that the results are not an artifact of the method of data reduction.

The results from the HR variability measure are similar to those of the deceleration data. For the well nourished infants, the initial presentation of the tone was associated with an increase in cardiac activity over the pre-stimulus level. Cardiac activity then decreased with subsequent presentations of the same tone. The dishabituation stimuli produced an increase in cardiac activity which also decreased over the remaining trials. For the malnourished infants, cardiac activity did not change from pre-stimulus to stimulus onset levels following the presentation of the initial tone, nor was there a change in activity when the malnourished infants were presented with the dishabituation stimuli. As was evident in Figure 5 the HR activity measure was characterized by more variability than the deceleration curve. This is because the activity measure is sensitive to both changes in acceleration and deceleration as evidenced by the positive correlations between HR activity and both decelerations and accelerations (see Table 6).

The results of this measure of cardiac activity can be compared with the results of studies by Porges, in which HR variability was related to attentional processes. Porges, Arnold, and Forbes (1973) found that in newborns, HR vari-

ability increased to tone onset and went on to suggest that magnitude of pretrial HR variability may be an index of attentional responsivity to the environment. The variability measure in the Porges et al. study was the mean HR variance calculated within a given period, such as trial onset. The present study used a change score in cardiac activity from pre-stimulus to stimulus onset periods. Therefore, the issue of resting HR variability and subsequent attentional responses cannot be addressed by the present data. However, both the study by Porges and the present investigation found an increase in cardiac change (variability or activity) to the onset of the tone. Thus, overall cardiac change, independent of the direction of the change, that is, deceleration or acceleration, may be a useful measure of attention. Such a measure would be particularly appropriate for research with newborns, in whom the directionality of the HR response to external stimulation is clearly ambigious (c.f. Graham & Jackson, 1970).

To sum the results of the HR data, well nourished infants were found to exhibit the expected OR and habituation of the OR as measured by changes in both cardiac decelerations and activity when presented with a pure tone stimulus. The same two measures also revealed response recovery or dishabituation to changes in tonal frequency. The malnourished infants, on the other hand, showed little

or no evidence of the OR for either measure, nor was response recovery demonstrated to the dishabituation stimuli. HR accelerations were minimal or absent among both groups of infants with the exception of the latter trials in the first tone sequence for the well nourished infants, which was perhaps a reflection of increasing boredom with the testing.

The BOR measure yielded few responses among either group, although as was indicated in Figure 6 a greater percentage of well nourished than malnourished infants did evidence the BOR. Further, the percentage of well nourished infants who showed the response declined over trials and increased to the two dishabituation stimuli. For the malnourished infants neither response decrement nor response recovery was noted.

It is important to note that the mean deceleration among the well nourished infants who evidenced the BOR was not different from those in whom the response was not observed. This finding sheds doubt on the statement by Bridger and Reiser (1959) that a behavioral response provides an independent measure of the infant's reception of the stimulus and should be used to validate cardiac responses. A more likely explanation is that the BOR is a poorer measure of central processes than is the HR response.

Implications

The basic finding in this study, that the OR is diminished or absent in infants suffering from malnutrition provides strong evidence for a fundamental attentional deficit associated with nutritional insult. As mentioned previously, the OR is the organism's initial response to stimulation and provides the mechanism for selective attention. It enables the organism to detect and respond to salient aspects of the environment. OR attenuation or absence, then suggests an inability to respond to and process impinging novel stimulation. This confirms the clinical observations of Birch and Gussow (1970) and others that the apathy and lack of responsivity of the malnourished infant is due to a lack of stimulus receptivity.

That magnitude of the OR is related to learning is suggested from recent studies by Zeiner and Schell (1971) and Ingram and Fitzgerald (1972). In the former study, in which a visual conditional stimulus was used, more rapid aquisition of the conditional discrimination was found among high OR magnitude adults. The Ingram and Fitzgerald investigation reported similar findings in a study of classical conditioning of the skin potential response to an auditory stimulus in infants. Here conditioning was demonstrated only in high OR magnitude subjects. If magnitude of the OR does indeed predict learning, then absence or attenuation of the OR may be indicative of a learning deficit.

Support for this position was reported in a review of the Soviet experiments on the OR in mentally defective children (Lynn, 1967). The OR of these children to low and medium intensity stimuli was frequently absent. In an experiment in which a verbal instruction was preceded by a neutral stimulus, normal children produced an OR to the neutral stimulus and were conditioned to it whereas mentally defective children showed neither the OR, nor evidence of subsequent conditioning. These results were taken as an indication of deficits in cortical functioning.

The role of the cortex in orienting was also evaluated in the Brackbill (1971) study in which an anencephalic infant evidenced a strong OR that did not habituate. Her conclusion was that "the cortex may not be important to the elicitation of a response, but it is essential to inhibition of response" (1971, p. 12). While these results may appear contrary to the Soviet investigations both can be explained according to the model proposed by Sokolov (1963). According to this model the OR can occur either through direct stimulation of the recticular activating system (RAS) or as mediated by the cortex.

This also permits comparison of the performance of the malnourished infants in the present study with that of the mentally defective children reported in the Soviet literature. Absence of the OR in both groups may be due to lack of communication between the cortex and the RAS

or to a deficit in the RAS or cortex itself. Although the explanation of the absence of the OR remains obscure the implication that OR absence or attenuation retards learning seems warranted. This suggests that the kind of deficits found in the present study, if they persist into childhood, can account, at least partially, for the often reported poor performance of malnourished children on standard psychological tests.

Obviously, whether or not the effects of malnutrition are reversible cannot be determined from the present study. Research with animals has shown that malnutrition can lead to structural damage to the central nervous system (Dobbing, 1970; Lester, 1970). Moreover, there is mounting evidence to suggest that the consequences of malnutrition are most severe for the developing brain. For example, in one study, reduction of adult rat body weight by 40 to 50 percent failed to produce a reduction in brain weight (Peters & Boyd, 1966). The chemical composition of the brain as measured by both RNA and DNA concentrations was also found to be unaffected by similar reductions in body weight (Lehr & Gayet, 1963). On the other hand, when rats were malnourished during the period of rapid brain growth, which occurs entirely postnatally during the first twenty one days, severe and permanent effects were reported. Deficits in brain weight were found by Dobbing (1968) and Benton, Moser, Dodge, & Carr (1966). The latter study also

reported a reduction in total brain lipids, phospholipids, and cholesterol. Winick and Noble (1966) found a reduction in the rate of cell division and in the total number of brain cells. Other studies on the developing rat brain have related malnutrition to deficits in protein synthesis (Zamenoff, Marthens, & Margolis, 1968), a reduction in protein content (Chase, Lindsley, & O'Brien, 1969) and incomplete myelination (Chase, Dorsey, & McKhann, 1967). Finally, studies on the developing pig brain have yielded similar CNS damage as a result of malnutrition (Stewart & Platt, 1968; Dobbing, 1968).

Dobbing (1970) has taken this evidence to suggest a vulnerable periods hypothesis which states that in any species brain development may be most susceptible to damage if insult occurs during the period of rapid brain growth. In terms of such an hypothesis, the human brain would be most sensitive to nutritional insult from the last trimester of pregnancy through the first few years of life.

There is some support from human studies for the idea that the effect of nutritional insult on intelligence is most severe when insult is suffered early in life. Studies by Chase and Martin (1970), Cravioto and Robles (1965), and Stoch and Smythe (1963) which were reviewed earlier found that performance on an infant scale was lower in infants who suffered malnutrition during infancy

than among infants for whom insult occurred later in life. For example, in the report by Cravioto and Robles (1970), DQ performance recovered among infants hospitalized between 37 to 42 months of age whereas infants who were hospitalized prior to six months showed no recovery in DQ during comparable periods of hospitalization. While the interpretation of the data from these studies remains equivocal due to inadequate controls for confounding variables such as social class, hospitalization, and parental education, they do suggest that the infancy period may be one of particular susceptibility to the long term consequences of nutritional insult.

It is also interesting to note that the kind of learning deficit suggested by the results of the present study, that of a deficiency in attentional process, has also been associated with low birth weight infants (Caputo & Mandell, 1970). While studies of low birth weight infants suffer from some of the same problems as do studies of malnourished infants, that is, confounding effects of social class and the exclusive use of infant developmental scales, there is a striking similarity between the effects of low birth weight and malnutrition. Several authors have suggested that the consequences of low birth weight include disturbances in brain functioning (Caputo & Mandell, 1970; Wiener, 1965). Moreover, there is also evidence to suggest that attentional processes are affected by low birth weight.

For example, Drillen (1961) as well as Pasamanick (1965) found lack of concentration to be one of the major handicaps of school aged children born of low birth weight.

Finally, several studies have shown that reading is one of the primary abilities to be affected by low birth weight (DeHirsch, Jansky, & Langford, 1964; Rabinovitch, Bibace, & Caplan, 1961). These authors have interpreted their findings as reflecting a deficit in the infant's ability to receive information from the environment. These findings lend further support to the notion that early insult in general may have long term consequences for intellectual development and specifically that a direct result of early insult may be a deficit in attentional processes.

Conclusions

There are two main conclusions to be drawn from this study. First, infantile malnutrition affects the infant's basic ability to respond to and process impinging novel stimulation. The absence or attenuation of the OR reflects an attentional deficiency that probably has serious consequences for subsequent learning. Thus, there is the image of the malnourished infant as "out of touch" or unresponsive to his environment. It seems extremely likely that such an attentional handicap may impair learning and be responsible for the often reported poor performance of malnourished children on standard psychological tasks.

Obviously, the kind of research called for is to examine the long term consequences of this deficiency. Longitudinal studies which follow malnourished infants through school age are most appropriate. Simultaneously, the question of the reversibility of the effects of malnutrition could be approached through studies in which intervention is an experimental treatment at both younger and older ages.

Another approach is to expand the design of the present study to further elaborate the nature of this attentional deficit. The issue of sex differences, for example, was not addressed in the present investigation. It would also be useful to determine to what extent the effect demonstrated in this study was a function of the modality tested. Studies exploring the habituation of the OR with visual stimuli, for example, would contribute to our understanding of the generality of the deficit.

With older children, the Zeaman and House (1963) model might prove fruitful in the investigation of the relation between attention and nutritional insult. This theory assumes that discrimination learning requires the acquisition of two responses, an attentional response which precedes an instrumental response. By plotting learning curves these authors found that while rate of learning, once it starts, is generally independent of the number of trials, the number of trials for learning to start varies

considerably. It is this early portion of the curve, the number of trials for learning to start, that is presumed to be controlled by attentional processes; instrumental processes take over once learning has begun. If attentional processes are affected by malnutrition one might predict that the number of trials for learning to start is related to the nutritional status of the child. Likewise, rate of learning, once it starts, should be relatively unaffected by nutritional insult.

Finally, one would like to examine the effect of nutritional insult on other behaviors. Are all learning situations similarly affected? If magnitude of the OR is indicative of learning, then conditionability should be affected by malnutrition both in terms of the rate of aquisition of a conditioned response and as related to OR magnitude. It might also be interesting to examine the extent to which this deficit can be overcome in the face of nutritive reinforcement. The procedures employed by Papousek (1968) are appropriate as they involve the elaboration of conditioned head turning with food as reinforcement.

The second major conclusion from this study is a methodological one. This is simply that the habituation paradigm was effective in uncovering differences due to malnutrition. This technique could readily be applied to other infant risk populations, such as low birth

weight infants and has the advantage of measuring specific cognitive processes as opposed to the DQ approach. The use of psychophysiological measures should also be underscored as sensitive indicators of possible dysfunction. Had the present study relied solely on behavioral measures the dramatic differences between well and malnourished infants would probably have been obscured.

The importance of these findings can hardly be exaggerated. The probability that two thirds of the world's children may be suffering from intellectual impairment as a result of poor feeding is staggering! That governments are aware of the problem and have chosen to do nothing about it is disgraceful! How is it possible that there are over ten million Americans living on diets having less than two thirds of the minimum nutrients required for adequate health (U.S. Senate Select Committee on Nutrition and Human Needs, 1969)? Nutritional rehabilitation programs must include psychological as well as physical intervention as we are well aware of the potentially devastating effects of stimulus deprivation often associated with institutional care. It is only through such programs that we can help the suffering infant relearn to interact with his environment. In so-called developing countries, such as Guatemala, the infant is particularly vulnerable as early weaning on protein deficient diets often leads to nutritional inanition within the first year of life.

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APPENDIX

APPENDIX TABLE 1.--Summary of Analysis of Variance of Mean Heart Rate Deceleration for Tone Sequence I in Well Nourished Infants by Order of Stimulus Presentation and Trials.

Source	SS	df	MS	F	P
Order	71.12	1	71.12	3.22	
Error	353.44	16	22.09		
Trials	1246.42	9	138.49	8.82	.001
Trials X Order	131.22	9	14.58	.93	
Error	2261.16	144	15.70		
Total	4063.37	179			

APPENDIX TABLE 2.--Summary of Analysis of Variance of Mean Heart Rate Deceleration for Tone Sequence II in Well Nourished Infants by Order of Stimulus Presentation and Trials.

Source	SS	df	MS	F	P
Order	6.94	1	6.94	.70	
Error	158.98	16	9.94		
Trials	628.00	4	157.00	12.35	.001
Trials X Order	37.11	4	9.28	.73	
Error	813.69	64	12.71		
Total	1644.72	89			

APPENDIX TABLE 3.--Summary of Analysis of Variance of Mean Heart Rate Deceleration for Tone Sequence III in Well Nourished Infants by Order of Stimulus Presentation and Trials.

Source	SS	df	MS	F	P
Order	0.00	1	0.00	0.00	
Error	369.60	15	24.64		
Trials	385.32	4	96.33	6.6	.001
Trials X Order	98.17	4	24.54	1.68	
Error	867.29	60	14.45		
Total	1720.38	84			

APPENDIX TABLE 4.--Summary of Analysis of Variance of Mean Heart Rate Deceleration for Tone Sequence I in Malnourished Infants by Order of Stimulus Presentation and Trials.

Source	SS	df	MS	F	P
Order	2.65	1	2.65	.11	
Error	420.61	18	23.37		
Trials	115.51	9	12.83	.69	
Trials X Order	233.91	9	25.99	1.41	
Error	2995.89	162	18.49		
Total	3768.56	199			

APPENDIX TABLE 5.--Summary of Analysis of Variance of Mean Heart Rate Deceleration for Tone Sequence II in Malnourished Infants by Order of Stimulus Presentation and Trials.

Source	SS	df	MS	F	P
Order	5.71	1	5.71	.17	
Error	516.29	15	34.42		
Trials	24.87	4	6.22	.30	
Trials X Order	94.38	4	23.60	1.13	
Error	1256.56	60	20.94		
Total	1897.82	84			

APPENDIX TABLE 6.--Summary of Analysis of Variance of Mean Heart Rate Deceleration for Tone Sequence II in Malnourished Infants by Order of Stimulus Presentation and Trials.

Source	SS	df	MS	F	P
Order	46.94	1	46.94	1.82	
Error	412.18	16	25.76		
Trials	101.27	4	25.32	1.68	
Trials X Order	49.44	4	12.36	.82	
Error	966.49	64	15.10		
Total	1576.32	89			

APPENDIX TABLE 7.--Summary of Analysis of Variance of Mean Heart Rate Acceleration for Tone Sequence I in Well Nourished Infants by Order of Stimulus Presentation and Trials.

Source	SS	df	MS	F	P
Order	6.08	1	6.08	.25	
Error	392.56	16	24.54		
Trials	219.47	9	24.39	2.36	.025
Trials X Order	48.35	9	5.37	.52	
Error	1486.89	144	10.33		
Total	2153.35	179			

APPENDIX TABLE 8.--Summary of Analysis of Variance of Mean Heart Rate Acceleration for Tone Sequence II in Well Nourished Infants by Order of Stimulus Presentation and Trials.

Source	SS	df	MS	F	P
Order	3.21	1	3.21	.39	
Error	131.29	16	8.21		
Trials	57.04	4	14.26	1.25	
Trials X Order	26.96	4	6.74	.59	
Error	731.60	64	11.43		
Total	950.10	89			

APPENDIX TABLE 9.--Summary of Analyses of Variance of Mean Heart Rate Acceleration for Tone Sequence III in Well Nourished Infants by Order of Stimulus Presentation and Trials.

Source	SS	df	MS	F	P
Order	22.61	1	22.61	2.59	
Error	130.80	15	8.72		
Trials	18.93	4	4.73	.43	
Trials X Order	29.05	4	7.26	.66	
Error	655.89	60	10.93		
Total	857.28	84			

APPENDIX TABLE 10.--Summary of Analysis of Variance of Mean Heart Rate Acceleration for Tone Sequence I in Malnourished Infants by Order of Stimulus Presentation and Trials.

Source	SS	df	MS	F	P
Order	.02	1	.02	.00	
Error	107.70	18	5.98		
Trials	46.22	9	5.14	.73	
Trials X Order	65.08	9	7.23	1.03	
Error	1133.70	162	6.70		
Total	1352.72	199			

APPENDIX TABLE 11.--Summary of Analysis of Variance of Mean Heart Rate Acceleration for Tone Sequence II in Malnourished Infants by Order of Stimulus Presentation and Trials.

Source	SS	df	MS	F	P
Order	22.49	1	22.49	1.64	
Error	205.59	15	13.67		
Trials	38.99	4	9.75	1.18	
Trials X Order	35.60	4	8.90	1.08	
Error	496.71	60	8.28		
Total	798.87	84			

APPENDIX TABLE 12.--Summary of Analysis of Variance of Mean Heart Rate Acceleration for Tone Sequence III in Malnourished Infants by Order of Stimulus Presentation and Trials.

Source	SS	df	MS	F	P
Order	1.34	1	1.34	.30	
Error	71.11	16	4.44		
Trials	88.16	4	22.04	2.45	
Trials X Order	64.38	4	16.09	1.79	
Error	576.67	64	9.01		
Total	801.66	89			

APPENDIX TABLE 13.--Summary of Analysis of Variance of Mean Heart Rate Variability for Tone Sequence I in Well Nourished Infants by Order of Stimulus Presentation and Trials.

Source	SS	df	MS	F	P
Order	12.72	1	12.72	.30	
Error	678.64	16	42.42		
Trials	657.37	9	73.04	3.02	.005
Trials X Order	404.39	9	44.93	1.24	
Error	5202.86	144	36.13		
Total	6955.98	179			

APPENDIX TABLE 14.--Summary of Analysis of Variance of Mean Heart Rate Variability for Tone Sequence II in Well Nourished Infants by Order of Stimulus Presentation in Trials.

Source	SS	df	MS	F	P
Order	20.54	1	20.54	1.15	
Error	286.72	16	17.92		
Trials	809.11	4	202.28	12.96	.001
Trials X Order	103.07	4	25.77	1.65	
Error	998.62	64	15.60		
Total	2218.06	89			

APPENDIX TABLE 15.--Summary of Analysis of Variance of Mean Heart Rate Variability for Tone Sequence III in Well Nourished Infants by Order of Stimulus Presentation and Trials.

Source	SS	df	MS	F	P
Order	1.32	1	1.32	.04	
Error	533.10	15	35.54		
Trials	501.26	4	125.31	5.80	.001
Trials X Order	58.53	4	14.63	.68	
Error	1295.87	60	21.58		
Total	2390.08	84			

APPENDIX TABLE 16.--Summary of Analysis of Variance of Mean Heart Rate Variability for Tone Sequence I in Malnourished Infants by Order of Stimulus Presentation and Trials.

Source	SS	df	MS	F	P
Order	.01	1	.01	.00	
Error	972.89	18	54.05		
Trials	377.85	9	41.98	1.42	
Trials X Order	326.05	9	36.23	1.23	
Error	4790.41	162	29.57		
Total	6467.20	199			

APPENDIX TABLE 17.--Summary of Analysis of Variance of Mean Heart Rate Variability for Tone Sequence II in Malnourished Infants by Order of Stimulus Presentation and Trials.

Source	SS	df	MS	F	P
Order	.51	1	.51	.01	
Error	733.51	15	48.90		
Trials	32.25	4	8.06	.33	
Trials X Order	154.65	4	38.66	1.60	
Error	1451.21	60			
Total	2372.13	84			

APPENDIX TABLE 18.--Summary of Analysis of Variance of Mean Heart Rate Variability for Tone Sequence III in Malnourished Infants by Order of Stimulus Presentation and Trials.

Source	SS	df	MS	F	P
Order	31.21	1	31.21	1.21	
Error	414.09	16	25.88		
Trials	240.62	4	60.16	1.54	
Trials X Order	62.84	4	15.71	.40	
Error	2502.13	64	39.40		
Total	3250.90	89			

APPENDIX TABLE 19.--Summary of Analysis of Variance of Mean Heart Deceleration for Tone Sequence I by Nutritional Status and Trials.

Source	SS	df	MS	F	P
Nutrition	.17	1	.17	.01	
Error	847.82	36	23.55		
Trials	533.93	9	59.33	3.42	.001
Trials X Nutrition	875.62	9	97.29	5.61	.001
Error	5622.18	324	17.35		
Total	7879.72	379			

APPENDIX TABLE 20.--Summary of Analysis of Variance of Mean Heart Rate Deceleration for Tone Sequence I (Trials 2-10) by Nutritional Status and Trials.

Source	SS	df	MS	F	P
Nutrition	67.46	1	67.46	2.66	
Error	913.71	36	25.38		
Trials	152.38	8	19.05	1.06	
Trials X Nutrition	134.55	8	16.82	.94	
Error	5162.98	288	17.93		
Total	6431.09	341			

APPENDIX TABLE 21.--Summary of Analysis of Variance of Mean Heart Rate Deceleration for Tone Sequence I (Trials 6-10) by Nutritional Status and Trials.

Source	SS	df	MS	F	P
Nutrition	106.41	1	106.41	5.36	.05
Error	733.99	37	19.84		
Trials	11.92	4	2.98	.18	
Trials X Nutrition	7.84	4	1.96	.12	
Error	2458.27	148	16.61		
Total	3318.44	194			

APPENDIX TABLE 22.--Summary of Analysis of Variance of Mean Heart Rate Deceleration for Tone Sequence II by Nutritional Status and Trials.

Source	SS	df	MS	F	P
Nutrition	57.25	1	57.25	2.75	
Error	687.92	33	20.85		
Trials	265.31	4	66.33	3.98	.001
Trials X Nutrition	368.05	4	92.01	5.52	.001
Error	2201.74	132	17.51		
Total	3580.28	174			

APPENDIX TABLE 23.--Summary of Analysis of Variance of Mean Heart Rate Deceleration for Tone Sequence II (Trials 12-15) by Nutritional Status and Trials.

Source	SS	df	MS	F	P
Nutrition	10.01	1	10.01	.59	
Error	607.45	36	16.87		
Trials	45.39	3	15.13	.84	
Trials X Nutrition	24.91	3	8.30	.46	
Error	1940.45	108	17.97		
Total	2628.20	151			

APPENDIX TABLE 24.--Summary of Analysis of Variance of Mean Heart Rate Deceleration for Tone Sequence II by Nutritional Status and Trials.

Source	SS	df	MS	F	P
Nutrition	.13	1	.13	.01	
Error	828.72	33	25.11		
Trials	199.37	4	49.84	3.32	.025
Trials X Nutrition	287.12	4	71.78	4.78	.001
Error	1981.39	132	15.01		
Total	3296.75	174			

APPENDIX TABLE 25.--Summary of Analysis of Variance of Mean Heart Rate Deceleration for Tone Sequence III (Trials 17-20) by Nutritional Status and Trials.

Source	SS	df	MS	F	P
Nutrition	34.11	1	34.11	1.42	
Error	840.13	35	24.00		
Trials	95.84	3	31.95	1.86	
Trials X Nutrition	39.51	3	13.70	.77	
Error	1806.79	105	17.21		
Total	2816.38	147			

APPENDIX TABLE 26.--Summary of Analysis of Variance of Mean Heart Rate Acceleration for Tone Sequence I by Nutritional Status and Trials.

Source	SS	df	MS	F	P
Nutrition	20.44	1	20.44	1.45	
Error	506.36	36	14.07		
Trials	109.46	9	12.16	1.44	
Trials X Nutrition	165.65	9	18.41	2.19	.025
Error	2734.02	324	8.44		
Total	3535.93	379			

APPENDIX TABLE 27.--Summary of Analysis of Variance of Mean Heart Rate Acceleration for Tone Sequence I (Trials 6-10) by Nutritional Status and Trials.

Source	SS	df	MS	F	P
Nutrition	74.97	1	74.97	4.19	.05
Error	661.90	37	17.89		
Trials	13.52	4	3.38	.36	
Trials X Nutrition	81.19	4	20.30	2.17	
Error	1381.88	148	9.34		
Total	2213.47	194			

APPENDIX TABLE 28.--Summary of Analysis of Variance of Mean Heart Rate Acceleration for Tone Sequence II by Nutritional Status and Trials.

Source	SS	df	MS	F	P
Nutrition	2.30	1	2.30	.21	
Error	362.08	33	10.97		
Trials	64.05	4	16.01	1.64	
Trials X Nutrition	28.08	4	7.02	.72	
Error	1290.86	132	9.78		
Total	1747.37	174			

APPENDIX TABLE 29.--Summary of Analysis of Variance of Mean Heart Rate Acceleration for Tone Sequence III by Nutritional Status and Trials.

Source	SS	df	MS	F	P
Nutrition	.04	1	.04	.01	
Error	225.87	33	6.84		
Trials	73.12	4	18.28	1.82	
Trials X Nutrition	31.71	4	7.93	.79	
Error	1325.99	132	10.05		
Total	1656.72	174			

APPENDIX TABLE 30.--Summary of Analysis of Variance of Mean Heart Rate Variability for Tone Sequence I by Nutritional Status and Trials.

Source	SS	df	MS	F	P
Nutrition	57.23	1	57.23	1.24	
Error	1664.30	36	46.23		
Trials	675.67	9	75.07	2.27	.025
Trials X Nutrition	418.55	9	46.51	1.41	
Error	10723.70	324	33.10		
Total	13539.45	379			

APPENDIX TABLE 31.--Summary of Analysis of Variance of Mean Heart Rate Variability for Tone Sequence I (Trials 2-10) by Nutritional Status and Trials.

Source	SS	df	MS	F	P
Nutrition	9.97	1	9.97	.24	
Error	1505.23	36	41.81		
Trials	388.05	8	48.51	1.40	
Trials X Nutrition	257.03	8	32.13	.92	
Error	10010.57	288	34.76		
Total	12170.85	341			

APPENDIX TABLE 32.--Summary of Analysis of Variance of Mean Heart Rate Variability for Tone Sequence II by Nutritional Status and Trials.

Source	SS	df	MS	F	P
Nutrition	58.84	1	58.84	1.86	
Error	1041.31	33	31.55		
Trials	454.68	4	113.67	5.54	.001
Trials X Nutrition	361.08	4	90.27	4.40	.001
Error	2707.53	132	20.51		
Total	4623.43	174			

APPENDIX TABLE 33.--Summary of Analysis of Variance of Mean Heart Rate Variability for Tone Sequence II (Trials 12-15) by Nutritional Status and Trials.

Source	SS	df	MS	F	P
Nutrition	8.53	1	8.53	.26	
Error	1202.47	36	33.40		
Trials	18.82	3	6.27	.30	
Trials X Nutrition	20.74	3	6.91	.33	
Error	2249.43	108	20.83		
Total	3499.98	151			

APPENDIX TABLE 34.--Summary of Analysis of Variance of Mean Heart Rate Variability for Tone Sequence III by Nutritional Status and Trials.

Source	SS	df	MS	F	P
Nutrition	4.36	1	4.36	.15	
Error	979.75	33	29.69		
Trials	125.42	4	31.36	1.06	
Trials X Nutrition	622.20	4	155.55	5.24	.001
Error	3919.36	132	29.69		
Total	5651.09	174			

APPENDIX TABLE 35.-- Summary of Analysis of Variance of Mean Heart Rate Variability for Tone Sequence III (Trials 17-20) by Nutritional Status and Trials.

Source	SS	df	MS	F	P
Nutrition	84.74	1	84.74	3.26	
Error	909.76	35	25.99		
Trials	71.97	3	23.99	.80	
Trials X Nutrition	138.51	3	46.17	1.54	
Error	3154.89	105	30.05		
Total	4359.87	147			