Comparison of cardiovascular and behavioral responses to near-threshold acoustic stimuli in baboons

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Three male baboons were trained to respond in an auditory psychophysical procedure to obtain reliable estimates of their auditory thresholds. Subsequently, animals were implanted with femoral catheters and retested at near-threshold levels of a 1.0-kHz tone. Beat-by-beat information was obtained on changes in interbeat interval (IBI) and both systolic and diastolic blood pressure (SBP and DBP) during tone presentations. IBIs decreased as stimulus intensity increased when animals correctly detected the stimuli, whereas no changes in IBI occurred when the animals did not detect the stimuli or when the response lever was removed in a probe session. No changes in SBP were observed, and one animal showed significant increases in DBP as stimulus intensity increased. Thresholds estimated from the overall functions relating changes in IBI to changes in stimulus intensity were approximately 7-9 dB higher than thresholds estimated from behavioral psychometric functions.

Sensory psychophysical detection experiments that have also measured cardiovascular responses have taken two directions. One area of research has studied how cardiovascular variables affect the detection of stimuli (e.g., Lacey & Lacey, 1970). Such studies have, for example, manipulated cardiovascular levels by altering heart rate (e.g., Saxon & Dahle, 1971) or have presented stimuli at different portions of the cardiac cycle (Birren, Cardon, & Phillips, 1963). The second body of research has studied how stimulus detection affects cardiovascular responses, with an eye toward using cardiovascular measures as "objective" indices of sensory detection (e.g., Gerber, Mulac, & Lamb, 1977; T. Suzuki, 1978; Zeaman & Wegner, 1956). These studies have manipulated stimuli along an intensity or frequency dimension and examined functional relationships with cardiovascular changes.

For auditory detection in particular, such overt behavioral indices as the "I heard it," the head nod, or a hand raise have long been used to assess hearing, while such physiological responses as respiration (Walter & Porges, 1976), brainstem evoked responses (J. Suzuki & Yamane, 1982), or heart rate changes (Butterfield, 1962) have been more or less neglected. Although there is general agreement that cardiovascular response magnitude can vary directly with stimulus intensity at

suprathreshold intensities (e.g., Suzuki, 1978; Zeaman & Wegner, 1956), there has been some controversy regarding the use of cardiovascular changes (principally heart rate changes) as an index of stimulus detection at near-threshold levels of auditory stimuli (Borton & Smith, 1980). The present study addressed this problem by providing data on the correlations among changes in heart rate, blood pressure, and a behavioral detection response in nonhuman primates during the performance of a psychoacoustic threshold-detection procedure.

METHOD

Subjects

The subjects were three male baboons (*Papio cynocephalus*), weighing 17-22 kg. None of the animals had any history of acoustical or other trauma. One of the subjects (SI) previously had received intramuscular injections of ketamine twice weekly for 6 weeks in an unrelated experiment. All subjects were TB tested at 3-month intervals with ketamine used as a tranquilizer. The animals were maintained on a 22-h restricted feeding regimen with supplemental monkey chow and fresh fruit provided daily after experimental sessions.

Apparatus

During threshold training and testing sessions, each animal was housed in a double-walled foam-insulated concrete-block acoustical chamber with inside dimensions of 1 × 1.3 × 1.7 m. The attenuation characteristics of each chamber ranged from 60 dB at 250 Hz to greater than 100 dB at 2 kHz and above. A 76 × 97 cm intelligence panel inside the chamber contained a response lever, a red cue light, and a tube feeder for delivery of banana pellets. With the animal positioned facing the panel, the response lever rested in front of the animal's right hand, and the feeder tube in front of his mouth. Acoustic stimuli were delivered via an Altec wide-range speaker suspended from the ceiling and located directly over the animal's head approximately 80.5 cm above ear level.

Pure-tone signals were generated by a Leader oscillator passed through a Coulbourn electronic switch (20-msec rise and fall times)

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and programmable attenuator, a Crown D-75 amplifier, and the speaker. The system was calibrated with a General Radio sound-level meter (No. 1981B) by a 0.5-in. condenser microphone placed at ear level where the baboon's head would normally be and at a 90-deg incidence angle to the sound source.

A specially designed microcomputer system was employed as an on-line data processor for transmitting real-time beat-by-beat physiological information to a larger DEC PDP-8/E minicomputer system, which in turn controlled the experiment and stored all physiological and behavioral data on hard disk for later evaluation and statistical analysis. The microcomputer system employed a high-speed Burr-Brown A/D converter that was software controlled to provide a sampling rate of 303 samples/sec for each variable, selected to ensure accurate detection of the systolic and diastolic components of the pressure wave.

Procedure

A reaction time procedure (Hienz, Turkkan, & Harris, 1982) was employed in which a flashing red cue light signaled the start of each trial. A baboon pressed the lever to initiate a trial, signaled by the flashing light becoming steady. The animals were trained to continue holding the lever down, releasing the lever only when a tone burst (1.5 sec duration) was presented from the overhead speaker. Each tone burst was programmed to occur randomly between 1.0 and 7.3 sec after initiation of the lever-holding response. Lever releases during the 1.5-sec tone period were defined as correct stimulus detections and were reinforced by the immediate delivery of one or two banana-flavored food pellets. Releases prior to or after the tone period were not reinforced. All trials were separated by a 1-sec intertrial interval (ITI), during which the cue light was off; responses during the ITI delayed the next trial by 1 sec.

Auditory thresholds were determined by randomly varying the intensity of the test stimuli from trial to trial for a block of 140 trials, with four to five such blocks comprising each session. Four intensity levels, 10 dB apart, were randomly presented at a test-tone frequency of 1.0 kHz. Interspersed among these trials were catch (no-tone) trials, which occurred randomly with a probability of .2. These trials were used to determine the false-alarm rate of each subject. Thresholds were corrected for such "guessing" by defining the threshold as that intensity producing a detection score halfway between the false-alarm rate and 100%. Four to five such threshold estimates were obtained during each session. For all animals, testing was conducted until performance stability was obtained, as indicated by stable thresholds (± 2 dB), false-alarm rates below 20%, and no systematic trends in the data. For two subjects (AL and MA), auditory threshold functions for frequencies ranging from .62 to 40.0 kHz had been previously published (Hienz et al., 1982). Subject SI had been previously tested at frequencies ranging in octave steps from .250 to 16.0 kHz.

Surgery

After stable performances were obtained, the animals underwent surgery for arterial catheterization in order to measure systolic and diastolic blood pressure (SBP, DBP) and interbeat intervals (IBIs) on every heart beat. A silicone coated polyvinyl catheter was inserted into the femoral artery under 1.75% halothane anesthesia according to a surgical procedure adapted from Werdegarr, Johnson, and Mason (1964). The distal end of the catheter was tunneled under the skin, exited in the interscapular area, and was fitted with an 18-ga Luer stud adapter. The arterial catheter was connected to a Statham transducer (P23De) mounted at heart level. The proximal end of the arterial catheter was advanced to just above the iliac bifurcation and below the renal arteries. Patency of the arterial catheter was maintained by a continuous infusion of heparinized saline (5,000 USP units per liter) at a constant rate of 4 ml/h, and by a more rapid infusion (125 ml/h) for one min once each day. Daily calibration of the system was accomplished without dismantling the components by integrating a mercury manometer through a series of three-way valves (Findley, Brady, Robinson, & Gilliam, 1971).

After a 2-week surgical recovery period, auditory thresholds were redetermined at 1.0 kHz using the reaction time procedure. In the postsurgical series of sessions, the behavioral procedure was modified so that the pretone foreperiod was synchronized to end at a heart beat occurrence. This synchronization of tone onset with a heart beat prevented tones from occurring at varying portions of the cardiac cycle, a manipulation that has been shown to affect reaction time performance (e.g., Birren et al., 1963).

Data Analysis

From each tone presentation trial, values for IBI, SBP, and DBP were obtained at the last pretone heart beat, all complete heart beats during the tone presentation, and the first posttone heart beat. Change scores were used as an index of cardiovascular response magnitude. IBI change scores were calculated by subtracting the first IBI after tone onset from the preceding pretone IBI. Positive changes indicate heart rate acceleration; negative changes indicate deceleration. Systolic and diastolic blood pressure changes were calculated by subtracting the last pretone pressure value from the first tone pressure value, with positive changes thus indicating increases in blood pressure. Reaction times typically ranged from 400-1,000 msec. When reaction times were short, the first heart beat after tone onset (first tone beat) often fell into the posttone beat category. Data presented in the results section for cardiovascular changes during the tone period are based upon the first tone beat only.

RESULTS

Figure 1 illustrates changes in both signal detectability and reaction time. Mean percent correct lever releases (Figure 1, left) and average median reaction time for correct releases (Figure 1, right) are shown as a function of the intensity of a 1.0-kHz tone for the three subjects. All subjects showed increases in signal detectability as tone intensity increased, describing familiar psychometric functions. The displacement of these functions along the abscissa indicates differences in threshold sensitivity among the animals, with Subject AL the most sensitive and Subject SI the least sensitive. Behavioral thresholds, defined as that intensity producing a detection level halfway between the catch trial rate and 100%, were -0.8 , 4.9 , and 16.5 dB SPL for Subjects AL, MA, and SI, respectively. Reaction time, on the other hand, generally decreased as stimulus intensity increased, with reaction times approaching asymptote at the highest stimulus intensity employed.

Cardiovascular responsiveness during the same sessions is plotted in Figure 2, where IBI (left), SBP (center), and DBP (right) change scores are shown as a function of tone intensity for the three subjects. Since not all subjects received the same tone intensities, a within-subject repeated measures analysis of variance was used to assess the significance of the cardiovascular changes. For all three subjects, the magnitude of IBI response increased with increasing tone intensity. Since pretone IBIs were constant across tone intensities, this indicates an increasingly accelerative heart rate response to tones [Subjects AL, MA, and SI yielded F values (4,8) of 10.47, 26.93, and 11.10, respectively; $p < .01$ in all cases].

Figure 1. Mean percent correct lever releases (left) and average median reaction time (right) as a function of tone intensity for three subjects. Each data point is the average of three sessions. Catch (no tone) trial response levels are shown to the left.

Figure 2. Changes in interbeat interval (left), systolic blood pressure (middle), and diastolic blood pressure (right) for the first tone beat after tone onset as a function of tone intensity. Each data point is an average of three sessions for each subject.

Figure 3. Changes in interbeat interval (left), systolic blood pressure (middle), and diastolic blood pressure (right) for the first tone beat after tone onset as a function of tone intensity. The upper graphs show data for trials in which each subject released the lever. The lower graphs show data for trials in which no lever release occurred. Each data point is an average of three sessions for each subject.

Figure 4. Change in interbeat interval for Baboon AL as a function of tone intensity in sessions in which the response lever was present (filled data points) and for a probe session in which the lever was removed (open data points).

Maximal changes in IBI occurred at the highest tone intensity for all animals, and averaged -132.54 msec (an average heart rate increase of 17.81 beats/min). Systolic blood pressure did not show significant changes as a function of tone intensity, and only Subject MA showed a significant increase in DBP with tone intensity [$F(4,8)=9.99$, $p < .01$]. The largest increases in systolic and diastolic blood pressure also generally occurred at the highest tone intensities and averaged $+4.18$ and $+5.64$ mmHg, respectively.

Figure 3 shows the IBI data of Figure 2 subdivided according to whether or not a lever release occurred at each tone intensity. When lever releases occurred, IBIs at the first tone heart beat showed increasingly acceleratory changes as tone intensity increased; no such trends were evident when subjects failed to release to the lever. Thus, acceleratory IBI changes occurred only when followed by a lever-release response. To clarify this point further, Subject AL was exposed to a probe session in which the lever was removed and tones of 1.5-sec duration were presented at the average intertrial interval of his previous sessions. Stimulus intensity was varied randomly from trial to trial, and each tone presentation was

followed by pellet delivery, except during catch trials. Changes in IBI as a function of tone intensity are shown in Figure 4 for this probe session (open circles) and for previous sessions in which the lever was present (closed circles). Again, acceleratory IBI changes occurred when followed by a behavioral response, whereas no such changes occurred when the lever was not available.

The relationship between changes in IBI and behavioral responding is further shown in Table 1, where correlations between absolute IBI and reaction time are given for each tone intensity. Each coefficient is the average across the three sessions for each subject (Fisher's r to z transformation; Snedecor & Cochran, 1967). For each animal, the highest positive correlation occurred with the most intense tones, with lower intensity tones having smaller coefficients. Thus, shorter IBIs occurred with shorter reaction times at the higher tone intensities. This relationship is shown in Figure 5 as a scatterplot of absolute IBI as a function of reaction time for Baboon SI. Filled data points denote the two higher tone intensities, and their clustering toward the short-latency, short-IBI region of the graph indicates the high correlation between these two measures at the higher tone intensities. Similar results were obtained with the other two animals.

For each subject, two methods were used to estimate hearing thresholds based on changes in IBI. First, a critical difference in IBI from its control (catch trial) level was determined for each IBI function of Figure 1, using Dunnett's *a posteriori* test with a significance level of .05. The "cardiovascular" threshold was defined as that intensity producing the critical difference as determined by interpolation, yielding thresholds of 9.2, 8.2, and 23.1 dB SPL for animals AL, MA, and SI, respectively. These threshold estimates averaged 6.6 dB higher than the behavioral threshold estimates. The second method of threshold estimation based on IBI changes was analogous to the behavioral method: The largest change in IBI was set at a 100% response, while the IBI change for catch trials was assumed to represent a 0% change. Threshold estimates based on interpolation to the 50% point of such functions were 9.2, 13.5, and 22.9 dB SPL for Subjects AL, MA,

Table 1
Correlation Coefficients (Pearson r) of the Relationship Between Absolute Interbeat Interval and Reaction Time as a Function of Tone Intensity

Subject	Catch		Intensity (dB SPL)											
			-8		2		12		22		32		42	
	r	n	r	n	r	n	r	n	r	n	r	n	r	n
AL	-0.30*	17	0.33	21	0.28†	125	0.49**	26	0.67**	7				
MA	0.37	12	0.44	15	0.34†	86	0.23	47	0.86†	13				
SI	0.08	33					0.13	44	0.38†	87	0.39†	44	0.86†	23

*Average of three sessions, Fisher's r to z . ** $p < .05$. † $p < .01$.

Figure 5. Scatterplot of the relationship between absolute interbeat interval and reaction time for Baboon S.I. All data points are from trials in which a lever release occurred, with each point representing one trial. Different symbols denote different tone intensities: □, ○, ■, ● represent trials in which tone intensities were 12, 22, 32, and 42 dB SPL, respectively; "X" represents trials in which no tones were presented (catch trials).

and SI, respectively. These threshold estimates averaged 8.7 dB higher than the behavioral thresholds.

The positive correlations between reaction time and IBI in Table 1 suggest that threshold estimates based on the IBI functions may be biased, since, by terminating the trial, short reaction times preclude the occurrence of longer IBIs. Accordingly, to correct the IBI functions of Figure 1 for this forced correlation, we eliminated the data where significant correlations of IBI and reaction time existed. This was accomplished by selectively eliminating the shortest reaction times and their associated IBIs until nonsignificant regressions were obtained for IBI upon reaction time. Cardiovascular thresholds reestimated from these corrected functions increased by an average of only 1.6 dB for the critical difference estimation procedure, and only 0.4 dB for the 50% threshold estimation procedure. Corrected IBI functions showed only occasional, minor deviations from the functions depicted in Figure 1.

Significant changes in diastolic blood pressure as a function of tone intensity were evident only for Subject MA. The two threshold estimation procedures described above were also employed with this subject's DBP data, with the critical difference threshold estimate at 14.3 dB SPL and the 50% method yielding a threshold estimate of 15.1 dB SPL. The diastolic blood pressure threshold estimates were 1-1.5 dB higher than the IBI threshold estimates, and about 10 dB higher than the behavioral threshold estimates.

DISCUSSION

The present study found accelerative heart rate changes to near-threshold tones which were function-

ally related to tone intensity. Also, auditory thresholds based on cardiovascular changes were found to be about 7-9 dB higher than those based upon the standard behavioral method employed.

Although trends in blood pressure responses were not significant, both systolic and diastolic blood pressures generally increased to a small degree in response to tones (see also Lacey & Lacey, 1970), with one subject demonstrating a significant relationship between diastolic blood pressure increase and tone intensity. A hearing threshold calculated from the diastolic pressure change function for this subject yielded a threshold estimate close to that obtained from the IBI change function. Both increases and decreases in blood pressure in response to auditory stimuli in a classical conditioning paradigm have been observed (Katcher, Solomon, Turner, Lolordo, Overmier, & Rescorla, 1969; Newton & Perez-Cruet, 1967). One study, in particular, obtained an increase in DBP but not in SBP for "clearly audible" tones (Obrist, Howard, Lawler, Sutterer, Smithson, & Martin, 1972).

The direction of heart rate change to signal and nonsignal tones has been the focus of a great deal of research and theory (e.g., Graham & Clifton, 1966; Lacey & Lacey, 1970, 1974; Sokolov, 1963). The majority of studies measuring the heart rate response to tone stimuli in both humans and animals have found heart rate deceleration in the first heart beat of the tone (e.g., Barry, 1977; Galin & Lacey, 1972; Gautier, 1972; Gerber et al., 1977; Jackson, 1974; Lacey & Lacey, 1970; Newton & Perez-Cruet, 1967; Walter & Porges, 1976), whereas the present study found heart rate acceleration at tone onset. The direction of heart rate responses to tones, however,

may be determined by several variables. For example, heart rate of humans has been found to accelerate to tones during early trials of a study, and to decelerate during later trials (Friedman, Tursky, & Erlenmeyer-Kimling, 1980). Coles and Duncan-Johnson (1977) found heart rate acceleration during an easy tone-frequency-discrimination task, and deceleration at tone onset when the discrimination was difficult. Jennings and Wood (1977) noted that the direction of heart rate change at tone onset in a reaction time task depended upon whether the tone was synchronized with the R-wave of the EKG, resulting in acceleration in the first heart beat, versus when the tone occurred 350 msec after the R-wave, resulting in heart rate deceleration. Direction of heart rate response can depend not only upon the above-mentioned factors specifically relating to near-threshold level tones, but also upon CS-US interval (in conditioning paradigms), species of subject, and importantly, the prestimulus level of heart rate (see Cohen, 1974, and Turkkan, Brady, & Harris, 1982, for reviews). The present findings of heart rate accelerations to tones may stem from two sources. First, as suggested by Jennings and Wood (1977), tone onset at the R-wave of the EKG may have coincided with a period of minimal inhibitory vagal activity, allowing a heart rate acceleration within the same cardiac cycle. Second, short reaction times at the higher intensity tones precluded long interbeat intervals, thereby increasing the relative frequency of short, acceleratory IBIs, although this factor resulted in only minor changes in the cardiovascular functions after corrections were made for regression of IBI on latency.

Several aspects of these data differ from previous studies which have also measured the cardiovascular response to pure tones. First, diastolic blood pressure and heart rate changes were observed at intensities lower than generally reported in humans. For example, T. Suzuki (1978) found detectable heart rate responses in sleeping infants at intensities greater than or equal to 40 dB above subjective threshold for 1-kHz tones. Schulman and Wade (1970) obtained heart rate responses to 1-kHz tones at 34 dB in 77% of normal infants. Gerber et al. (1977) obtained heart rate decelerative responses to 1-kHz tones at 20 dB SPL in adults, but did not attempt to measure hearing thresholds. The present data estimated threshold for changes in IBIs and DBPs at an average of 13-15 dB SPL. It seems unlikely that the increased cardiovascular sensitivity to tones in this study might have stemmed from the use of baboon subjects, since behaviorally determined auditory thresholds have been found to be virtually identical for the two species at tone frequencies between .250 and 4 kHz (Hienz et al., 1982).

Some difficulties in comparing the present findings with those of others lie in the lack of standard pro-

cedures for determining auditory thresholds based on cardiovascular responses. Investigators have occasionally failed to report a criterion for the presence or absence of cardiovascular response (e.g., Schulman, Smith, Weisinger, & Fay, 1970); others have stated the cardiovascular response to be "all or none" (Gerber et al., 1977) or graded with tone intensity (T. Suzuki, 1978; Zeaman & Wegner, 1956). Often, alternate methods of audiological assessment have not been used for comparison of cardiovascular and conventional behavioral thresholds. In the present study, behavioral thresholds were defined by standard animal psychophysical techniques (Pfingst, Hienz, & Miller, 1975), whereas cardiovascular thresholds were defined as critical differences from the no-tone (catch trial) cardiovascular response, or as a 50% change from no-tone levels to the peak cardiovascular changes observed. Differences between cardiovascular and behavioral thresholds averaged about 7-9 dB, with behavioral threshold estimates being lower.

In considering the greater sensitivity of thresholds based upon motor, as opposed to cardiovascular, responses, there are at least two possible explanations. First, reinforcement contingencies were applied to specific motor responses, and not to cardiovascular responses. While one might expect other concomitant responses of an organism (including cardiovascular responses) to be strengthened via the reinforcement process, the greatest strengthening would be expected for that response explicitly operated on by the prevailing contingencies (similar to human psychophysical models in which the response that yields the largest payoff for "hits" will yield the lowest threshold value, e.g., Galanter, 1966, chap. 6). This interpretation agrees with the observation that no cardiovascular changes were observed when contingencies were eliminated during the probe session.

A second explanation for the lower motor thresholds may be that there is a direct relationship between behavioral and cardiovascular change. The probe session manipulation eliminated not only the reinforcement contingencies but also the animal's ability to execute the response. Additionally, no cardiovascular changes were observed during normal sessions when animals failed to release the lever during tone trials, regardless of tone intensity. Thus, in all cases, cardiovascular changes occurred only when lever-release responses also occurred. Although it is tempting to assume that the cardiovascular changes observed were due to their being coupled with the skeletal motor activity of leverpress ("cardiac-somatic coupling," Obrist, Howard, Lawler, Galosy, Meyers, & Gaebelain, 1974), more recent data have demonstrated the limits of direct coupling of cardiac and somatic events (e.g., Cohen, Johnson, & McArthur, 1980). Moreover, as suggested by Obrist and his colleagues (1981, chap. 4), the degree of covariation in

heart rate and somatic activity may depend on a number of factors, including the extent of sympathetic nervous system influences in the behavioral paradigm employed. Thus, the issue of cardiac-somatic interaction in this paradigm remains an open question.

The data reported here raise the issue of adequately defining what is meant by terms such as "hearing" or "hearing threshold." Certainly, the assessment of an organism's hearing sensitivity will depend upon the type of response used as an indicator of stimulus detection (e.g., behavioral, cardiovascular, neural). An overriding consideration, however, is whether or not explicit consequences exist that will establish and maintain the chosen response as such an indicator. If one simply presents tones to an organism and measures an arbitrary response (e.g., heart rate, eye blink, body temperature), one may not obtain an accurate assessment of that organism's hearing function. On the other hand, when contingencies are employed that require an organism to respond differentially to the presence or absence of acoustic stimuli (i.e., when we say an organism is "attending" to the stimuli), considerably more accurate assessments may be obtained. In the last analysis, sounds can only be judged as heard when an organism's behavior changes in some significant manner in relation to the occurrence of the sounds, and such changes are considerably more likely when explicit consequences are contingent upon behavior.

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