work without being punished at all, and some work without a chain. Apparently social reward and perhaps two types of activity rewards are involved. In many primate societies a high-ranking dominant is not only feared, he is also a focus of attraction: other members try to stay near him and to interact with him. The coconut monkey probably derives social reward from the interaction with his master, at least if the master is a "friendly" dominant. In a macaque group, there are two types of dominants: the "nonassertive" ones (*4*), who elicit more attraction than avoidance, and the "assertive" or aggressive ones, who elicit the opposite response. Monkey owners can also be divided into these two categories, and a coconut monkey will perform well with one man and poorly with another. Whether or not the master feeds the monkey seems irrelevant to their relationship. Indeed the women and children who generally feed the monkeys are often threatened and sometimes bitten by them, probably because they show fear. Corner has suggested that a monkey works partly because he is afraid of being beaten, and partly because he enjoys breaking, smashing, and knocking things down. Such activities are a basic component of the behavior of male macaques. Finally, monkeys may prefer working in trees to being tied to a pole all day. When not working, they are kept chained, and some show definitely stereotyped motions, such as clutching their head, biting their feet and hands, and rocking back and forth.

Thus fear, the motive on which training is established, may cease to be the primary determinant of performance. It is questionable if reward training would give as good or even better results than punishment training. Methods using the presentation and omission of negative reinforcers have two obvious weaknesses: they indicate to the monkey what must not be done, rather than what should be done, and they arouse escape and "freezing" reactions which interfere with learning. Perhaps the monkey would learn to make the appropriate movement sooner if he were rewarded as soon as he approximated it, rather than if he were punished for not making it. An animal that cannot be trained with the punitive method could prove amenable to reward.

There are two possible explanations for the absence of reward training in Malaya and Thailand. First, the idea of rewarding the monkeys during training and work had not occurred to the Thai villagers I interviewed. They said that a monkey should work because he is told to do so, and should be punished if he rebels, as were children in the traditional Thai schools. Furthermore, generations of trainers found that punishment is necessary to establish a dominance relationship without which the monkey will not obey. It is not surprising that the use of punishment was then extended to other aspects of training. The best training method might be to use punishment at the beginning of training, to establish a dominance relationship, and then switch to reward, to establish a system of communication.

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References and Notes

1. E. J. H. Corner, *Malayan Nature J.* **2**, 11 (1941); *Zoo Life* **1**, 89 (1946).
2. J. Bastian, in *Primate Behavior*, I. DeVore, Ed. (Holt, Rinehart and Winston, New York, 1962), p. 588.
3. E. J. H. Corner, *Proc. Roy. Inst. Gt. Brit.* **36**, 1 (1955).
4. S. A. Altmann, *Ann. N.Y. Acad. Sci.* **102** (2), 338 (1962).
5. I thank Dr. B. Lekagul, Mrs. P. Phinpraphat, Professor F. Bourlière, Professor E. J. H. Corner, and Dr. J. S. Myer for assistance. Research was supported by PHS grant 9-Ro7-TW00141-o5-CIC.

15 December 1966

State as a Determinant of Infants' Heart Rate Response to Stimulation

Abstract. *With each infant serving as his own control, the data indicate that waking or sleeping states, independent of the prestimulation heart rate, can significantly affect the heart rate response to tactile stimulation.*

The role of state in determining a psychological or physiological response is not disputed. However, few studies of neonates and young infants have paid much attention to this variable. This is surprising in view of the fact that infants are more likely to show dramatic changes in state over a relatively short testing period than are adults, who maintain relatively consistent states and long periods of alertness. Recent studies have attempted to relate behavioral and autonomic indices of state (*1*), as well as to determine the effect of prestimulation level of behavioral and physiological variables on subsequent performance (*2*). These studies, as well as others using adults (*3*), have all shown the effect of prestimulation level and the necessity of controlling for this effect.

However, no studies of young infants have tested the same subject under different states or observed whether there were any differences in response to stimulation as a function of state independent of the initial level. The present study is a first attempt to investigate these problems.

Eleven subjects between 2 and 8 weeks of age were tested individually under two state conditions, asleep and awake. These states were defined by two observers who rated an infant asleep if (i) the infant's eyes were closed, (ii) he exhibited no vocalization, (iii) no gross activity or sucking, and (iv) an even respiration record (Fels Respirometer) (*4*). The infant was judged awake only if all four of the following conditions were met: (i) his eyes were open, (ii) he exhibited uneven respiration record, (iii) he showed slight body activity or sucking behavior, or both, and (iv) he was relatively alert (rated subjectively according to the amount and nature of the eye movement and scanning). Infants judged to be awake but irritable were not tested until they were quieted. High inter-observer reliability has been reported when similar measures were used (*2*). Interobserver reliability was recorded: eyes open, .94; vocalization, .84; activity and sucking, .73; respiration, .79; alertness, .71.

The tactile stimulation consisted of a calibrated series of 20 nylon filaments (*4*) which were presented to the corner of the infant's mouth and run back toward the upper angle of the ear. The stimuli were presented in a monotonically increasing order which was constant for each infant and for each state. The duration of the tactile stimulation was approximately 1 second with a variable intertrial interval of 15 to 25 seconds. The subject was placed either in a supine or prone position, depending upon his comfort, and the experimenter always approached the subject so that the infant was unable to see the experimenter. At the same time that the subject was stimulated, the experimenter depressed a key to record the stimulus duration on a

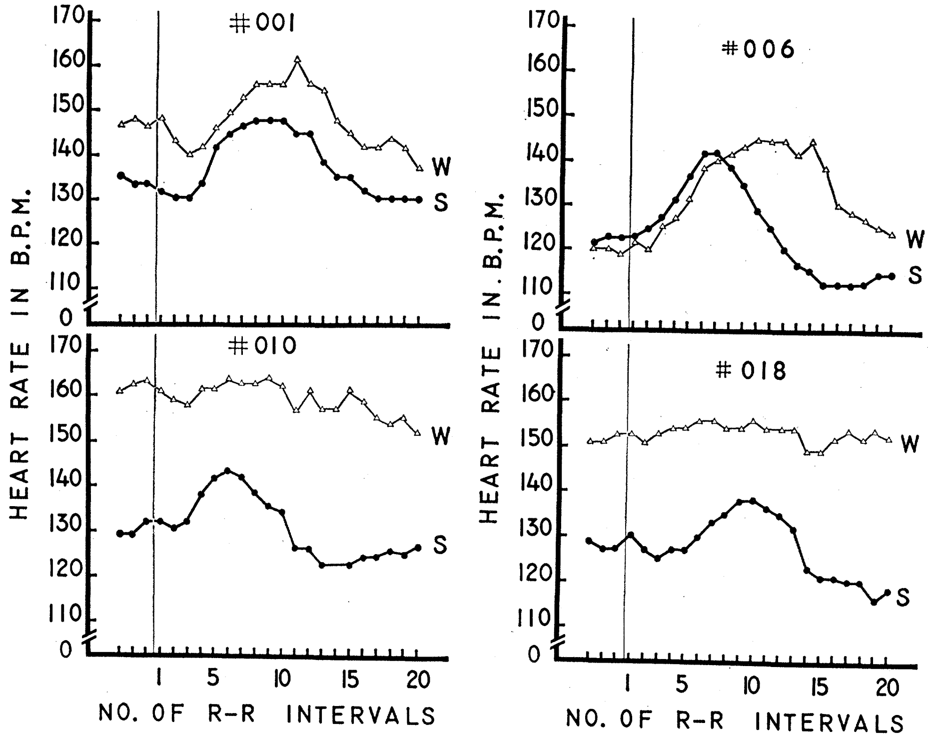

Fig. 1. Four individual records of the cardiac response to tactile stimuli in the waking and sleeping conditions. *B.P.M.*, beats per minute.

polygraph which was continuously recording the infant's heart rate and respiration.

Each R-R (cycle to cycle) interval was converted to a rate-per-minute score by use of a Fels cardiotachometer (*4*). The continuously monitored beat-by-beat cardiac rate appeared on a standard polygraph output. At the same time, the infant's R-R intervals were recorded on punch paper tape. Also automatically recorded on both the polygraph and punch paper tape was the onset and duration of each stimulation period.

By these recording procedures it was possible to obtain for each subject, for each trial, a beat-by-beat cardiac rate anchored from the onset of stimulation. The beat immediately prior to stimulation was $S-1$, which, in turn, was preceded by $S-2$, and so forth. Similarly, the beat subsequent to stimulation was $S+1$, followed by $S+2$, and so forth. For the 20 trials, for each state, medians were computed separately for the $S-1$ beats, for $S-2$, and so forth. In this way, each subject had a median beat-by-beat curve for stimulation during the waking and sleeping states. A beat-by-beat measure was used, rather than averaging over real time, in order to observe small changes in the cardiac response which would be lost in any averaging procedure.

Figure 1 presents four of the 11 records which were thought to represent the sample as a whole. For each subject, curves for cardiac response to stimulation when awake and asleep are presented for the first 20 R-R intervals, or approximately 8 seconds. The x-axis represents the number of R-R intervals from the onset of stimulation, while the y-axis represents the rate in beats per minute of the particular R-R interval. The vertical line indicates the onset of stimulation. A decrease in the beat-by-beat rate indicates cardiac deceleration.

These data, as well as the data collected from the seven other subjects, indicate a marked difference in the cardiac response to tactile stimulation during the sleeping and waking states. First, the records of all 11 subjects while asleep showed a clear pattern of cardiac acceleration, with the peak acceleration occurring at approximately 6 to 9 beats (approximately 3 to 5 seconds with an average heart rate of 150 beats per minute) after the onset of stimulation. This acceleration response was quite symmetrical, with a return to base level or below about 13 beats after the onset of stimulation. Moreover, this response resembled an inverted V rather than an inverted U curve. While all of the subjects showed an acceleration response, subject No. 001 showed an initial one- or two-beat deceleration before accelerating. This was the only case of such a biphasic response, that is, deceleration before acceleration. The remaining subjects showed acceleration followed by a deceleration component.

The curves of the same subjects when awake fail to show a consistent pattern. Subjects Nos. 010 and 018 were typical of the four subjects who showed no cardiac response. Three subjects showed initial deceleration before accelerating (see No. 001), while three subjects showed initial acceleration to stimulation which had a slow rise to peak and showed no recovery to a base level for at least 30 to 50 R-R intervals after stimulation. The most impressive result of stimulation during the waking state was the lack of consistent intersubject responses.

In order to observe whether there were statistical differences between the curves, several specific measures were taken: (i) latency to peak heart rate (latency to shortest R-R interval); (ii) the initial response to stimulation (the difference between the rate of the last three beats prior to stimulation and that of the five beats immediately following the onset of stimulation); (iii) the range of cardiac responsivity (the difference between the mean heart rates based on the three highest and the three lowest beats during stimulation). While other measures of cardiac responsivity are possible, these were chosen because they have been shown to reflect important stimulus and individual differences (*5*).

The data reveal that the latency to the peak heart rate was significantly longer in the waking than in the sleeping condition in 9 out of the 11 comparisons (Sign test, $p = .033$, one tail). In order to compare the other parameters, the prestimulation heart rate based on the mean of the last three beats prior to stimulation was observed for each subject, awake and asleep. The data revealed significant differences in the two states (see Table 1), with the prestimulation rate higher in the waking than the sleeping condition for 10 out of the 11 subjects (Sign test, $p = .011$, one tail). This result is consistent with the previously cited work and indicates that one difference between the waking and sleeping states is the level of the prestimulation heart rate.

Control of this variable would enable investigation of state differences

Table 1. Individual mean prestimulation heart rates (beats per minute) for the awake and asleep states.

Subject	Awake	Asleep	
		Mean	S.D.
0.01	148	133	7.28
002	134	128	6.43
004	134	129	5.62
005	158	139	7.06
006	120	122	5.96
008	128	125	2.63
010	161	129	4.63
014	148	129	6.11
016	171	151	2.83
018	153	128	4.76
020	140	128	2.46

independent of the prestimulation heart rate. Because of the small number of trials, it was not possible to control mathematically for the prestimulation rate (*3*). Instead, the procedure used was as follows. For the 20 trials asleep, a mean heart rate and a standard deviation were obtained for each subject (see Table 1). Next, each trial when the subject was asleep was compared to its paired trial when he was awake and any trial during the waking condition for which the prestimulation heart rate was one standard deviation or less away from the heart rate during sleep was considered to have an equal prestimulation heart rate. Each subject could have a maximum of 20 matched pairs; however, two subjects had no matched pairs, while the remaining nine subjects had from two to nine pairs. For these nine subjects a mean curve was generated for the waking and sleeping states so that each subject would contribute only one value to the following analyses.

Table 2 presents the data for the nine subjects who could be equated on prestimulus heart rate. In order to observe the initial cardiac response to stimulation, the difference between the prestimulation rate and the first five beats after stimulation were compared for each of the states. The data indicate no significant differences between the states, although three of the nine subjects showed cardiac deceleration during the waking state and none showed this response while asleep. Second, the range data indicated that there is significantly greater intrasubject cardiac variability in response to stimulation when asleep (Sign test, $p < .02$, one tail) than when awake. The data, therefore, indicate that there are significant differences between states which are independent of the prestimulation heart rate.

Table 2. Individual subjects' cardiac responses after experimenter controls for prestimulation level.

Subject	Mean change		Range	
	Awake	Asleep	Awake	Asleep
001	12.75	1.45	31.25	49.75
002	9.37	2.29	11.96	16.68
004	−7.80	1.30	21.85	22.25
005	3.15	4.85	20.65	30.80
006	8.69	6.16	22.42	26.46
008	−1.45	6.45	23.70	25.20
014	−4.60	2.70	11.17	20.10
016	7.50	7.70	19.10	18.80
020	3.65	0.20	9.85	15.95

One difference between states, other than the prestimulation heart rate, might be different rates of habituation (*6*). One way to check this possibility would be to observe state differences on trial 1. Observation of the sleeping and waking data for this trial indicated that prestimulation heart rate could not be matched. The only parameter available to test state differences was the latency measure, which indicated that even on trial 1, subjects showed shorter latency to peak heart rate during sleep than when awake ($p < .08$).

Although the behavioral criteria which determined our definition of state lacked the rigor necessary to provide a clear picture of the state of the infant, and could not differentiate depths of sleep, the results of the present study are in agreement with a recent study using electroencephalographic measures as the criterion of sleep (*6*). The data for five adult males indicated greater cardiac variability in response to stimulation in the sleeping than in the waking state.

The present data do serve to demonstate that there are important differences in infants' cardiac response to tactile stimulation which are dependent on the state of the organism. First, there are significant prestimulation differences in the heart rate. It is clear from this experiment, as well as from the work of Birns *et al.* (*2*), that one of the differences in state is the level of arousal prior to stimulation. However, even when the prestimulation heart rate is controlled, or when it would not be expected to influence the data (as in the latency to peak rate), state differences are still found. Thus, the present results raise the important issue of state differences which are independent of prestimulation level. Furthermore, the results point up the necessity for investigators to specify and control state differences as well as prestimulation levels. This is especially true for any study exploring the developmental changes in cardiac responsivity using the very young infant (*7*). Since neonates are asleep 70 percent or more of the time (*8*), it is more than likely that they will be stimulated when asleep while the older infants may be awake. The differences observed might not reflect a maturational change in the functioning of the autonomic nervous system so much as a difference in state. The present study underscores the importance of careful observation and control of state as well as initial physiological levels.

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References and Notes

1. W. H. Bridger, B. M. Birns, M. Blank, *Psychosomat. Med.* **27**, 123 (1965).
2. B. M. Birns, M. Blank, W. H. Bridger, S. Escalona, paper presented at the Eastern Psychological Association Meeting, 1964, Philadelphia, Pa.
3. J. I. Lacey and B. C. Lacey, *Ann. N.Y. Acad. Sci.* **98**, 1257–1326 (1962); J. I. Lacey, *ibid.* **67**, 123–164 (1956).
4. Semmes-Weinstein Pressure Aesthesiometer, Shaw Laboratories, Syasset, L.I., N.Y.; Fels Respirometer and Fels Cardiotachometer, Yellow Springs Instrument Co., Yellow Springs, Ohio.
5. M. Lewis, paper presented at the American Psychological Association Convention, Chicago, September 1965; ——, J. Kagan, H. Campbell, J. Kalafat, *Child Develop.* **37**, 63 (1966); M. Lewis and S. J. Spaulding, *Psychophysiology*, in press; E. L. Lipton, A. Steinschneider, J. B. Richmond, *Psychosomat. Med.* **23**, 461 (1961).
6. D. J. Hord, A. Lubin, L. C. Johnson, *Psychophysiology* **3**, 46 (1966).
7. E. L. Lipton, A. Steinschneider, J. B. Richmond, *Child Develop.* **37**, 1 (1966).
8. A. H. Parmelee, W. H. Wenner, R. Schultz, *J. Pediat.* **65**, 576 (1964).
9. Supported in part by grants HD-00868, FR-00222, and FR-05537 from the National Institute of Mental Health. We thank Marilyn Rausch and Helen Campbell for the collection and analysis of the data.

7 November 1966

The Skin: Problems of Inheritance

In a recent article in *Science* R. F. Rushmer *et al.* point out the opportunities for interdisciplinary research focused on the skin (*1*). Such research would meet a number of fascinating problems related to inheritance. More than 150 anomalies of the skin and its appendages have been described as being caused by different mutant genes (Table 1). The chain of events between gene mutation and skin anomaly is, in most instances, virtually unknown (*2*).