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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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*).

Table 1. Number of skin anomalies ascribed to inheritance.

Autosomal		Gonosomal	
Dominant	Recessive	X	Y
105	38	8	1

New tools for diagnosis could improve not only the scarce knowledge about genic action, mutant and normal, but also the accuracy of family prognosis by tracing micromanifestations in heterozygous carriers of mutant genes.

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Seal Ears

The possible mechanism, recently offered by Odend'hal and Poulter (*1*), for pressure regulation in the cavity of the middle ear of sea lions is of interest to evolutionary biologists as well as to physiologists. The use of distensible venous sinuses to maintain the auditory ossicles in an air-filled space with a pressure equal to extratympanum pressure has been reported for members of two widely separated mammalian cohorts, the pinnipeds (*1*) and the cetaceans (*2*), although this physiological convergence varies in detail. Since the opinion that the seals are biphyletic (*3*, *4*) is finally gaining favor, physiological investigation of the middle ear of "true seals" (not covered by Odend'hal and Poulter) would be enthusiastically received.

Studies of the osteology of the basicranium and ear region in both groups of seals indicates that they differ considerably with respect to detailed structure of this region (*5*). The Phocidae ("true seals") and Otariidae (sea lions) are less like each other than each is like some other group within the Carnivora. The phocids are closer to mustelids; the otariids are closer to the bears. Among many observable differences is that the epitympanic sinus and recess is larger than the tympanic cavity in the "true seals" but not in the sea lions; also, the "true seals" have a well-developed posttympanic sinus and a greatly expanded hypotympanic sinus, all combining to form a relatively larger middle-ear cavity than that of the sea lions.

The volume of the middle ear at surface pressure would determine the final volume of air available in the middle ear at depth, when it is in equilibrium with the increased environmental pressure. Since the auditory ossicles are most effectively operative in such an equilibrated air-filled space, the final depth to which a seal can dive and still receive effective transossicular vibrations would be predetermined by the relative size of the ossicles (reflected in the size of the tympanic cavity) and the rest of the middle-ear cavity. If, as seems likely, there is a venous complex within the bulla to equalize pressure in the "true seals," and if we use Odend'hal and Poulter's line of reasoning, the "true seals" should be able to reach greater depths than the otariids and still be able to use sonar.

To my knowledge, sonar has been reported for sea lions (*6*) but not yet for phocids, although some are known to feed at depth [for example, the Northern elephant seal eats ratfish, which are always found below 50 fathoms (*4*)]. Since light is extinguished with increasing depth, the "true seals" probably also use sonar. Moreover, their ancestors were apparently more highly preadapted to this mode of life than the otariid antecedents. Some of the trends discussed above were already being expressed in early lutrines (*7*) and are in process of still greater refinement in the phocids, which are derivable from the mustelid stock. By contrast, the ancestors of the sea lions were probably primarily terrestrial proursids, and the anatomical modification of their middle-ear cavity is not as extreme. Some degree of preadaptation is implicit though, since they have effectively shifted into this adaptive zone.

This discussion is intended to illustrate how imperative information from physiological ecology can be to morphologists and systematists. How else can we explain hypertrophication of the middle-ear cavity, which in some aquatic mammals is for high-frequency reception, when this same hypertrophication in a terrestrial mammal, *Dipodomys*, is for low-frequency reception (*8*)?

I suggest that in addition to the suction-pump effect for decreasing intrabullar pressure and thus for filling these sinuses during diving, there may be a positive force as well. Since there are massive cardiovascular adjustments in diving mammals, such as the marked decrease in peripheral circulation (*9*), and since there are numerous reports that apnea and bradycardia are correlated with increased central blood pressure in mammals generally (*10*), the filling of the bullar sinuses may be facilitated by the physiological adaptation to the anoxia that accompanies diving. This would seem to be a classic example of correlated adaptation in two physiological systems.

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