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

<table>
<thead>
<tr>
<th>Subject</th>
<th>Awake Mean</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>001</td>
<td>148</td>
<td>133</td>
</tr>
<tr>
<td>002</td>
<td>134</td>
<td>128</td>
</tr>
<tr>
<td>004</td>
<td>134</td>
<td>129</td>
</tr>
<tr>
<td>005</td>
<td>158</td>
<td>139</td>
</tr>
<tr>
<td>006</td>
<td>120</td>
<td>122</td>
</tr>
<tr>
<td>007</td>
<td>128</td>
<td>125</td>
</tr>
<tr>
<td>010</td>
<td>161</td>
<td>162</td>
</tr>
<tr>
<td>014</td>
<td>148</td>
<td>129</td>
</tr>
<tr>
<td>016</td>
<td>171</td>
<td>151</td>
</tr>
<tr>
<td>018</td>
<td>153</td>
<td>128</td>
</tr>
<tr>
<td>020</td>
<td>140</td>
<td>128</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Subject</th>
<th>Mean change Awake</th>
<th>Awake Range</th>
<th>Mean change Asleep</th>
<th>Asleep Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>001</td>
<td>12.75</td>
<td>1.45</td>
<td>31.25</td>
<td>49.75</td>
</tr>
<tr>
<td>002</td>
<td>9.37</td>
<td>2.29</td>
<td>11.96</td>
<td>16.68</td>
</tr>
<tr>
<td>004</td>
<td>7.80</td>
<td>1.30</td>
<td>21.85</td>
<td>22.25</td>
</tr>
<tr>
<td>005</td>
<td>3.15</td>
<td>4.85</td>
<td>20.65</td>
<td>30.80</td>
</tr>
<tr>
<td>006</td>
<td>8.69</td>
<td>6.16</td>
<td>22.42</td>
<td>26.46</td>
</tr>
<tr>
<td>010</td>
<td>1.45</td>
<td>6.45</td>
<td>23.70</td>
<td>25.20</td>
</tr>
<tr>
<td>014</td>
<td>4.60</td>
<td>2.70</td>
<td>11.17</td>
<td>20.10</td>
</tr>
<tr>
<td>016</td>
<td>7.50</td>
<td>7.70</td>
<td>19.10</td>
<td>18.80</td>
</tr>
<tr>
<td>020</td>
<td>3.65</td>
<td>0.20</td>
<td>9.85</td>
<td>15.95</td>
</tr>
</tbody>
</table>

pared 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.

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 demonstrate 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.

MICHAEL LEWIS
BETTY BARTELS
SUSAN GOLDBERG
Fels Research Institute,
Yellow Springs, Ohio

References and Notes
9. Supported in part by grants HD-00868, FR-0022, 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.
10 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).
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.

CARL A. LARSON
Institute of Genetics,
University of Lund,
Lund, Sweden

References

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 basi-craniun 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 pinnipeds 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 pinnipeds, 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 procursids, 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.

SYLVIA F. GRAHAM
Department of Vertebrate Paleontology,
American Museum of Natural History,
New York, and Department of Biological Sciences, Queensborough Community College, New York

References and Notes
11. I thank Drs. M. C. McKenna and R. G. Van Gelder of the American Museum for use of collections and, along with Dr. G. T. McIntyre, for their stimulating discussions.

27 JANUARY 1967

Table 1. Number of skin anomalies ascribed to inheritance.

<table>
<thead>
<tr>
<th></th>
<th>Autosomal</th>
<th>Gonosomal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dominant</td>
<td>X</td>
<td>Y</td>
</tr>
<tr>
<td>Recessive</td>
<td>105</td>
<td>38</td>
</tr>
</tbody>
</table>

At the beginning of the page, there is a table showing the number of skin anomalies ascribed to inheritance, with the following numbers:

- Dominant: 105
- Recessive: 38
- Autosomal: 8
- Gonosomal: 1
The Skin: Problems of Inheritance
Carl A. Larson

Science 155 (3761), 488-489.
DOI: 10.1126/science.155.3761.488