effect of insufficient cold treatment. After 6 weeks' storage, at least 10 days of continuous light were needed to effect emergence of larvae which would not emerge in more normal photoperiods. The minimum treatment required was less for families that had been stored an additional 8 weeks. There should be no question, however, about the nature of the diapause in this budworm, since even exposure to continuous light failed to produce appreciable emergence after storage for less than from 4 to 6 weeks.

It is interesting to recall at this point that in budworm stocks selected over several generations for nondiapause, the best response was achieved only when the larvae were exposed to continuous light (*1*). The nature of the effects of continuous light and the relation to the humoral control of diapause merits further investigation. The action of continuous light may normally be deleterious and may possibly be similar to the effects, in *Cecropia* silkworm, of surgical manipulation, during the last few weeks of the chilling period, in hastening the restoration of the brain's endocrine activity (*4, 5*).

GEORGE T. HARVEY
Forest Insect Laboratory,
Sault Ste. Marie, Ontario, Canada

References and Notes

1. G. T. Harvey, *Can. J. Zool.* 35, 549 (1957).
2. This paper is contribution No. 481 of the Forest Biology Division, Science Service, Department of Agriculture, Ottawa, Canada.
3. G. Stehr, *Can. Entomologist* 86, 423 (1954).
4. C. M. Williams, *Biol. Bull.* 110, 210 (1956).
5. A paper giving further details of the results and of the effects of various overwintering treatments on subsequent growth and development is in preparation.

16 June 1958

Respiratory Discharge of the Pons

Central respiratory discharges have so far been recorded mainly in the medulla oblongata (*1*), which was known to cause maximal inspiratory or expiratory response by electric stimulation. On the other hand, an involvement of the pons in the neurogenesis of respiration has been widely accepted. A region which inhibits the activity of the apneustic center and so produces normal respiratory rhythm has been called the pneumotaxic center and was located in the anterior pons (*2*).

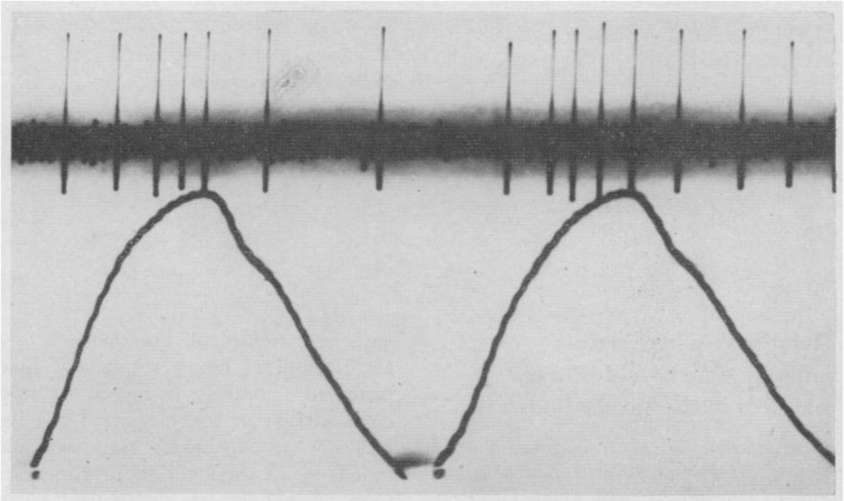

Fig. 1. (Top) Respiratory spike discharges obtained in the pons (9 mm rostral from the obex, 2.5 mm lateral from the mid-line, and at a depth of 2 mm from the surface), and (bottom) respiratory movement (peaks are inspiratory phase).

To localize this area more precisely, electrolytic lesions (*3*) and electrical stimulations (*4*) were used, and it was concluded that the pneumotaxic center lies in the extreme dorsolateral portion of the anterior pontine tegmentum or in the dorsolateral reticular formation of the isthmus of the pons. Rhythmicity of respiration was attributed to two mechanisms which are mutually replaceable—the vagal reflex and the pneumotaxic center—although the mode of firing of the latter remains obscure. Electric sounding of the pons, therefore, may offer a possibility for a more precise localization and may clarify the function of the pneumotaxic center.

After anesthetization with urethane (1.2 g/kg), rabbits were tracheotomized, and both carotid arteries were ligatured. Occipital craniotomy was performed, but the cerebellum was left intact. Care was taken to minimize bleeding. The pons and medulla oblongata were explored by means of stereotaxic apparatus carrying a monopolar steel electrode (of about 10 μ diameter), insulated except at the tip. Changes of potential and the respiratory movements of the chest were simultaneously photographed on the screen of a two-beam cathode-ray oscillograph.

Inspiratory and expiratory discharges were obtained in the medulla; bursts or increase in frequency of continuous discharges occurred coincidentally with the inspiratory or expiratory movements. The respiratory discharges were relatively sparse and failed to separate distinctly the inspiratory and expiratory region. Periodic bursts of impulses corresponding with respiratory rhythm, however, were also obtained in the pons at a depth of from 1 to 3 mm from the dorsal surface. The active loci were restricted to a relatively small region—from 2 to 2.5 mm lateral from the mid-line and from 7 to 11 mm rostral from the obex—approximately corresponding to the nucleus reticularis pontis caudalis of Meessen and Olszewski. Pontine respiratory discharges, by contrast with respiratory discharges obtained in the medulla, did not correspond either to the inspiratory or to the expiratory phase but appeared as shown in Fig. 1 in the course of inspiration and disappeared in the course of expiration. The frequency of spikes increased with the progress of inspiration and declined with the onset of expiration. These figures resemble vagal afferent discharge, the frequency of which varied with the degree of lung inflation (*5*). The vagus-like discharges obtained from the pons were composed of positive spikes, while inspiratory and expiratory discharges of the medulla were composed of negative ones. The meaning of this, however, remains, for the present, a matter for speculation.

In the vagotomized rabbit, transection of the mid-pons at the upper end of the acoustic tubercles failed to affect the respiratory rhythm so long as the section was limited to the medial part. Apneustic breathing resulted, however, when the section was made to extend 3.5 mm lateral from the mid-line. The functional similarity of vagal afferent and pneumotaxic centers is well known; hence, the discharge here observed in the pons may be a representation of pneumotaxic activity and function in converting the inspiratory to the expiratory act (*6*).

KENTARO TAKAGI
TERUO NAKAYAMA
Department of Physiology, Nagoya
University School of Medicine,
Nagoya, Japan

References and Notes

1. R. Gesell, J. Bricker, C. Magee, *Am. J. Physiol.* 117, 423 (1936); E. C. Amoroso, J. G. Bainbridge, F. R. Bell, A. M. Lawn, H. Rosenberg, *Nature* 167, 603 (1951); M. N. J. Dirken and S. Woldring, *J. Neurophysiol.* 14, 211 (1951); E. Haber, K. W. Kohn, S. H. Ngai, D. A. Holaday, S. C. Wang, *Am. J. Physiol.* 190, 350 (1957).
2. T. Lumsden, *J. Physiol.* (*London*) 57, 153 (1923); G. Stella, *ibid.* 93, 10 (1938); R. F. Pitts, H. W. Magoun, S. W. Ranson, *Am. J. Physiol.* 127, 654 (1939).
3. P. C. Tang, *Am. J. Physiol.* 172, 645 (1953).
4. S. H. Ngai, S. C. Wang, *ibid.* 190, 343 (1957).
5. E. D. Adrian, *J. Physiol.* (*London*) 79, 332 (1933).
6. This work was reported at the 35th general meeting of the Physiological Society of Japan, held from 3 to 5 May 1958.

5 June 1958