anemia. However, it is apparent that these 2 aforementioned studies are in agreement with the present report that endogenous hypergastrinemia in pernicious anemia does not directly control endogenous calcitonin levels. Nevertheless, the interrelationship may be complex since calcitonin is known to decrease both gastric acid and serum gastrin¹³⁻¹⁵. It is apparent that calcitonin is not under the unique influence of gastrin. Is the main regulator of calcitonin secretion some other gastrointestinal factor, serum calcium, some as yet undescribed chemical intermediary or a combination of these factors? As yet, the in vivo physiology of calcitonin secretion remains to be elucidated.

- H.P. Selawry, K.L. Becker, L.E. Bivins, R.H. Snider and O.L. Silva, Horm. Metab. Res. 7, 432 (1975).
- C.W. Cooper, W.H. Schwesinger, A.M. Mahgoub and D.A. Ontjes, Science 172, 1238 (1971).
- O.L. Silva, R.H. Snider, Jr, C.F. Moore and K.L. Becker, Ann. Surg. 189, 269 (1979).

- C.W. Cooper, J.E. McGuigan, W.H. Schwesinger, R.L. Brubaker and P. L. Munson, Endocrinology 95, 302 (1974)
- J.E. McGuigan and W.L. Trudeau, N. Engl. J. Med. 282, 358 (1970).
- J. Fahrenkrug, O.B. Schaffalitzky de Muckadell and J.F. Rehfeld, Acta endocr. 86, 140 (1977)
- P. Franchimont and G. Heynen, in: Parathormone and Calcitonin Radioimmunoassay in Various Medical and Osteoarticular Disorders, p. 115. Masson Inc., Paris 1976.
- O.L. Silva, R.H. Snider and K.L. Becker, Clin. Chem. 20, 337
- R.H. Snider, O.L. Silva, C.F. Moore and K.L. Becker, Clin. chim. Acta 76, 1 (1977).
- J.E. McGuigan, Gastroenterology 54, 1005 (1968)
- T.K. Li and J.T. Piechocki, Clin. Chem. 17, 411 (1971).
- G.W. Sizemore, V.L.W. Go, E.L. Kaplan, L.J. Sanzenbacher, K.L. Holtermuller and C.D. Arnaud, N. Engl. J. Med. 288,
- R.D. Hesch, M. Hufner, B. Hasenjager and W. Creutzfeld, Horm. Metab. Res. 3, 140, 1971. R.M. Bolman III, C.W. Cooper, S.C. Garner, P.L. Munson
- and S.A. Wells, Jr, Endocrinology 100, 1014 (1977). H.D. Becker, D.D. Reeder, M.T. Scurry and J.C. Thompson, Am. J. Surg. 127, 71 (1974).

Visual input to rat pineal

Nachum Dafny¹

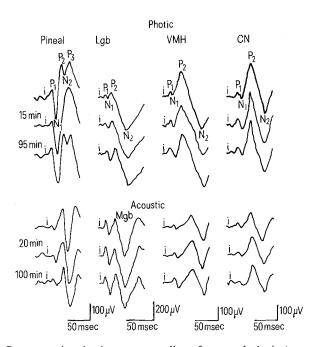
Department of Neurobiology and Anatomy, The University of Texas Medical School at Houston, Houston (Texas 77025, *USA*), 5 February 1980

Summary. Electrophysiological recordings from freely behaving rats, previously implanted stereotaxically with permanent electrodes in the pineal, ventromedial hypothalamus, caudate nucleus, lateral geniculate body and medial geniculate body were obtained. The pineal photic responses revealed 5 sequential components. Injection of a neuronal blocker at the level of the superior cervical ganglion did not alter the earlier photic responses, but did eliminate the late components (N₂-P₃) for 60-90 min after the injection. All of the other responses were unchanged during the experiment. The present experiments demonstrated that photic input travels to the pineal through two pathways.

In previous studies reported from this laboratory²⁻⁹, single cell activity as well as bipolar and monopolar evoked responses following photic, acoustic, olfactory bulb, ventromedial hypothalamus (VMH) and amygdaloid complex (Amyg) stimulation were recorded from the pineal and other structures simultaneously. The monopolar and bipolar recordings following photic stimulation were identical⁴; moreover, the initial photic response obtained demonstrated short latency. This short latency response could not be easily explained by the commonly accepted neuronal pathway i.e., it originates in the retina, which projects via the retinohypothalamic tract to suprachiasmatic nucleus, medial forebrain bundle, reticular formation, the upper thoracic intermediolateral cell column (the origin of preganglionic sympathetic fibres) and superior cervical ganglion (scg). The post synaptic sympathetic process, i.e. nervi conarii, from the scg travels along the tentorium cerebelli and enters the pineal with blood vessels 10-14. The present study was initiated to examine the possibility that photic inputs also reach the pineal via the central nervous system. 6 Sprague-Dawley male rats, weighing 250-350 g, were anesthetized with pentobarbital (50 mg/kg i.p.) and 5 nichrome electrodes (60 µm in diameter) were implanted stereotaxically in the pineal, ventromedial hypothalamus (VMH), caudate nucleus (CN), lateral geniculate body (Lgb) and medial geniculate body (Mgb) using the stereotaxic atlas of Konig and Klippel¹⁵ for coordinates. 4-6 days after electrode implantation, the animals were placed in a plastic cage within an electrophysiological testing chamber.

The electrodes were connected to conventional electrophysiological instruments⁵. 32 (1 set) photic or acoustic evoked responses (1/2.5 Hz) were averaged on-line using the NIC 1070 signal averaging computer. 4 sets (each set consisting of the average of 32 consecutive responses) following each modality (photic or acoustic) were recorded prior to bilateral injection of local anesthesia (0.3 ml of 1% xylocaine) in the region of the scg. Recordings were resumed every 5 min until recovery from the xylocaine effects was observed. At the conclusion of each experiment, the animals were sacrificed for histological verification of electrode placement²⁻⁶. The average photic and acoustic evoked responses recorded from the pineal, VMH, CN, Lgb and Mgb exhibited in general 5 components. The components are: initial positive peak (P₁) followed by negative (N₁) and positive-negativepositive (P₂-N₂ and P₃) deflections. Similar observations were observed previously in monopolar and bipolar recording^{4,16}. However, differences in amplitudes and latencies to the different peaks were apparent (figure). Bilateral xylocaine (1%, 0.3 ml in each side) injection eliminated only the N₂ and P₃ deflection of the pineal averaged photic evoked responses for 70-90 min without affecting the evoked responses (photic) recorded from the VMH, CN and Lgb (figure). Moreover, the acoustic responses in all the four structures (pineal, VMH, CN and Mgb) were not altered during the experimental period (figure). All 6 animals exhibited similarity in the control recordings as well as after the xylocaine injection, as demonstrated in the figure, which represents 1 animal.

Several studies^{4,8,9,14,17} indicate that the central nervous system makes neuronal connection to the pineal through the habenular and/or the posterior commissure complex. The finding of the present study demonstrated that local anesthesia, which is known to block electrical transmission¹⁸ eliminated only the 2 late components (N₂-P₃) of the photic evoked responses recorded from the pineal. The photic responses recorded from the VMH, CN and Lgb were unchanged as were the acoustic responses in all



Representative simultaneous recording of averaged photic (upper traces) and acoustic (lower traces) evoked responses from pineal, lateral geniculate body (Lgb), medial geniculate body (Mgb), ventromedial hypothalamus (VMH), and caudate nucleus (CN) before (upper traces) and after local anesthesia of both superior cervical ganglions. Time in min indicates the recording time after injections; i indicates onset of stimulation; P1 indicates the 1st positive component of the evoked response; N1, the 1st negative deflection; P2, N2 and P3 the 2nd positive, negative and 3rd positive deflection, respectively.

4 structures. The use of local anesthesia instead of surgical intervention. (i.e. scg-gangliectomy) in such experiments is advantageous because it provides recordings from the same animal at short time intervals both before and after interference of activity mediated through the scg. Moreover, in 4 animals, scg-gangliectomy was performed and similar observations obtained. Simultaneous recordings from 4 different brain locations allowed for more reliable conclusions regarding the effects of drugs on specific brain sites. Since only the late photic responses recorded from the pineal were abolished, it is possible to conclude that the early photic response reaches the pineal presumably via the stria medullaris habenular connection and therefore was not affected by the neuronal blockage at the level of the scg. The late responses were eliminated by the neuronal blockade and therefore it is possible to assume that they are transmitted via the classical pathway through the superior cervical ganglion.

- The author is grateful to Dr C.M. Prashad and Ms. Marjorie Brown for technical assistance. Supported in part by grant
- T.F. Burks and N. Dafny, Neuropharmacology 16, 681 (1977).

- T.F. Burks and N. Dafny, Exp. Neurol. 55, 458 (1977). N. Dafny, Exp. Neurol. 55, 449 (1977). N. Dafny and T.F. Burks, Neuroendocrinology 22, 72 (1976).
- N. Dafny and T.F. Burks, Exp. Neurol. 53, 633 (1976). N. Dafny and R. McClung, Experientia 31, 321 (1975).
- N. Dafny, R. McClung and S.J. Strada, Life Sci. 16, 611 (1975)
- R.E. McClung and N. Dafny, Life Sci. 16, 621 (1975).
- 10
- J.A. Kappers, Z. Zellforsch. 52, 163 (1960). D.C. Klein, in: The Hypothalamus, p.303. Ed. S. Reichlin, 11 R.J. Baldessarini and J.B. Martin. Raven Press, New York 1978.
- R.Y. Moore, Brain Res. 49, 403 (1973).
- 13 R. Y. Moore, Prog. Repro Biol. 4, 1 (1978).
- K.M. Shaw, in: Advances in Drug Research, vol.2, p.75. Ed. N.J. Harper and Alma B. Simmonds. Academic Press, London, New York, San Francisco 1977.
- J.F.R. Konig and R.A. Klippel, The Rat Brain. A Stereotaxic Atlas of the Forebrain and Lower Parts of the Brain Stem. R. E. Krieger Publishing Co., Inc., 1970.
- N. Dafny and B. M. Rigor, Neuropharmacology 17, 851 (1978).
- R.J. Reiter, A. Rev. Physiol. 35, 305 (1973).
- J.M. Ritchie and P.J. Cohen, in: Pharmacological Basis of Therapeutics, p. 379. Ed. Goodman and Gilman. Macmillan, New Ŷork 1975.

The effect of a juvenile hormone analogue on Eucelatoria sp. (Diptera: Tachinidae) through its host, Heliothis armigera (Hubn.) (Lepidoptera: Noctuidae)

B.J. Divakar

Central Biological Control Station, Directorate of Plant Protection, Quarantine and Storage, Bangalore - 560 032 (India), 28 December 1979

Summary. The juvenile hormone analogue, ZR-515 Methoprene, when applied topically to Heliothis armigera, adversely affected its parasite, Eucelatoria sp.

In the attempts to use synthetic hormones and pheromones to control insect pests, trials with several juvenile hormone (JH) analogues were made on several species of Heliothis, a serious polyphagous pest²⁻⁵. In biological control programmes, it is of the utmost importance to evaluate the effect of these synthetic compounds on the parasite complex as well. The effect of JH analogues on Apanteles rubecula Marshall⁶, spruce budworm parasitoids⁷, Pales

pavida Meig.⁸, and on the endoparasites of *Liriomyza* sativae Blanch.⁹ have been studied earlier. The following is an account of the effects of a JH analogue, ZR-515 Methoprene (isopropyl-11-methoxy-3,7,11-trimethyl-2,4-dodecadienoate) on Eucelatoria sp., a larval endoparasite of Heliothis armigera, when applied to the host.

H. armigera has been bred on an artificial diet10 in the laboratory for a number of generations under aseptic and