were taken from reticular nuclei localized more rostrally and laterally, as shown by histological controls. As for the third hypothesis, CAJAL[13] followed primary vestibular fibres till the reticular formation without being able to trace, however, their sites of termination which were, later, described by BRODAL[3] and further confirmed by CARPENTER[14] and GERNANDT[15]. According to these researchers, these primary fibres would leave the 8th nerve trunk before entering the vestibular nuclei and reach the reticular neurons directly. Primary vestibular fibres have been also reported to project directly to the cerebellar cortex and the fastigial nucleus[13–16]. Another interpretation of these short-latencies responses would be an electrotonic coupling between primary afferent fibres and neurons located either in the medial vestibular nucleus or in the surrounding reticular formation. The existence of this type of gap-junction synapse has been hypothetized in the mammalian abducens motoneurons and in explaining also latencies of 0.58 msec recorded in the MLF following electrical stimulation of contralateral vestibular nerve[17].

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Effect of Prostaglandin E_1 on Rat Gastric Motility and Cyclic Nucleotide Content

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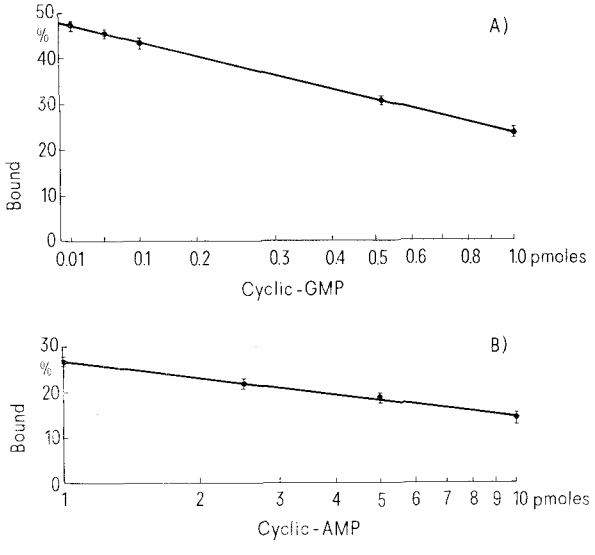
Summary. Administration of exogenous prostaglandin E_1 resulted in an increase in contractility of rat fundic muscle measured in vivo; a significant decrease in fundic tissue levels of cyclic- adenosine monophosphate and a significant increase in cyclic-guanosine monophosphate.

Prostaglandin (PG) interactions with adenosine 3¹,5¹-monophosphate (cAMP) have been studied in association with several different types of hormone systems[1,2] and research involving bi-directionally controlled mechanisms has implicated cyclic guanosine 3¹,5¹-monophosphate (cGMP) as an important regulator molecule which may act in opposition to cAMP[3]. By in vivo studying motility of the rat fundus, we have investigated the relationship between cAMP, cGMP, prostaglandin E_1 (PGE_1) and motility; PGE_1 should potentiate a cAMP decrease with an increase in motility and a decrease in cGMP should parallel a decrease in motility[4].



Standard curves for cyclic-GMP and cyclic-AMP. Each value represents duplicate trials with mean and SEM. A) cGMP; B) cAMP.

Male Holtzman rats 3–6 months of age, weighing 300–325 g were used in the experiment. They were maintained in one large cage with a common source of food (Purina Lab Chow) and water. The construction of the cage did not allow chewable roughage to accumulate. The rats were divided into 2 groups: control group, consisting of 5 rats which received daily i.p. injections of ethanol-Krebs solution solvent[5]; test group, consisting of 5 rats which received daily i.p. injections of PGE_1 (compliments of Dr. J. PIKE, The Upjohn Company, Kalamazoo, Mich., USA), 4.5 mg/kg[6]. Individual rat weight was checked daily to insure injections of proper dose.

A telemetry system[7] was used to monitor stomach motility. After a suitable plane of anaesthesia was induced with ether, a transverse incision accross the midline was made, and the transducer was firmly sutured to the outside fundic area. The body of the transmitter was placed in the peritoneal cavity and lightly sutured to the body wall to prevent displacement.

After implantation, recordings were made by connecting the discriminator output of an FM radio receiver directly to a strip-chart recorder. Three days were allowed for general recovery after which the telemetry system was tested for correct in vivo transmission. Recording was started 4 days after implantation, and gastric motility was monitored for a period of 14 days. The rats were

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given food on alternate days, and water was supplied on a continuous basis. Recordings were made only during periods when the rat was quiet.

At the end of the recording period, each rat was sacrificed with a blow to the head; the telemetry system was checked for proper placement of the transducers, the tissue reaction observed, the fundic area of the stomach immediately removed and rapidly frozen at $-70\,^{\circ}C$ until thawing for cyclic nucleotide assay. A double antibody radioimmunioassay kit procedure (Colabrative Research Inc., Waltham, Mass., USA) for cAMP and cGMP was performed on the fundic tissue[8]. Labelled nucleotide recovery was 72.3% on duplicate samples. Displacement of radioactive-labeled antigen by unlabeled cAMP or cGMP standards is linear when plotted as a semi-logarithmic function. The standard curves for cAMP and cGMP are reproduced in the Figure.

Ten recordings per rat were randomly selected, and a motility index[9] was calculated for each rat. Concentration of cyclic nucleotides was calculated using duplicate samples from each rat and converted into picomoles/g wet wt., tissue. Student's t-test was used to determine significance ($p < 0.05$). Significant differences were found ($p < 0.05$) between the two groups for all three factors: motility index, concentration of cAMP, concentration of cGMP (Table).

Previous investigators[10] had established a relationship between PGE_1, decrease in cAMP levels, increase in cGMP tissue levels and increased motility in smooth muscle studied in vivo. The increased motility observed in this study was attributed to the action of exogenous PGE_1 on the force and contractility of the circular and longitudinal fundic muscle as well as enhancing the effect of endogenous mucosal PGE_1.

The presence of exogenous PGE_1 administered by the i.p. route, served to influence total fundic tissue levels of the two cyclic nucleotides and served as a positive feedback on cholinergic activity. The increase or decrease in contractile activity was affected by the interaction between exogenous PGE_1, cAMP and cGMP and controlled by significant decreases in the tissue levels of these two cyclic nucleotides.

Motility index and cyclic Nucleotide values

	Motility index (Mean ± SEM)[b]	cAMP[a] (Mean ± SEM)[b]	cGMP[a] (Mean ± SEM)[b]
Control	61 ± 16.10	16 ± 1.40	0.30 ± 0.02
PGE_1 injected	84 ± 8.20	5 ± 1.20	0.45 ± 0.03

[a] Data are expressed as picomoles of cyclic nucleotide/g fundic tissue. wet. wt. [b] Each value is the mean (± SEM) of 5 different tissues.

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Effects of Three Synthetic Peptides Analogous to Neurohypophyseal Hormones on the Excitability of Giant Neurones of *Achatina fulica* Férussac

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Summary. Deamino-dicarba-(D-D-)oxytocin and D-D-Arg-vasotocin at 10^{-4} kg/l showed an excitatory effect on the periodically oxcillating neurone (PON) of *Achatina fulica* Férussac. D-D-Arg-vasopressin had no effect.

We obtained three synthetic peptides analogous to neurohypophyseal hormones, deamino-dicarba-oxytocin (D-D-oxytocin), deamino-dicarba-Arg-vasotocin (D-D-Arg-vasotocin) and deamino-dicarba-Arg-vasopressin (D-D-Arg-vasopressin) in the analytically pure state[2,3]. In the present study, we attempted to compare the effects of these peptides on the excitability of 2 spontaneously firing giant neurones (the PON, periodically oscillating neurone; and the TAN, tonically autoactive neurone)[4–6] identified in the subesophageal ganglia of an African giant snail, *Achatina fulica* Férussac.

A micropipette, implanted into one of the identifiable neurones, recorded its intracellular biopotential with a pen-writing galvanometer, and counted the number of its spike discharges per min by a spike counter. We applied these peptides dissolved in the snail's physiological solution[7] directly to the dissected ganglia (bath application). We also applied these peptides locally to an identifiable neurone (microdrop application)[8]. In this case, we made a microdrop in the open air (about 150 µm in diameter) of a peptide solution at the tip of a micropipette containing the peptide solution by oil pressure, and placed the microdrop on the surface of an identifiable neurone (its diameter is about 200 µm). As the electrical resistance of the neuromembrane, we measured its current-voltage relationships (I–V curve), using 2 microelectrodes implanted into the soma: one was to record the biopotential, the other to apply the transmembrane triangular current of long duration. We always recorded

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