

Actual (left) and simulated (right) ESR spectra of radical-anion from hermidin.

midin (1) for which there is analogy³. Of the two, (5) seems the more likely since (7) would have been expected to show splittings from 2 nitrogens, 2 methoxyls, etc.³ Further, when the water insoluble dimer (3) (written as the dianion) was dissolved in methanol-aqueous buffer the resulting solution did not give an ESR spectrum until alkali was added. Then, a weak spectrum of the radical-anion (2) was detected. This presumably arises from partial dissociation² of the dimer (3). The transient blue color produced on exposure of solutions of hermidin (1) (and possibly of bruised or cut stems of the plant from which it is derived) to air is due therefore to the formation of the radical-anion (2) "cyanohermidin".

- Acknowledgment. I thank Professor R. H. Thomson for helpful discussion.
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- 3 Ashworth, P., Tetrahedron 32 (1976) 261.
 - 0014-4754/84/070688-02\$1.50 + 0.20/0 © Birkhäuser Verlag Basel, 1984

Viscero-somatic reflexes following distension of urinary bladder in cats: Role of supraspinal neuraxis1

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Summary. Viscero-somatic reflexes have been studied by recording monosynaptic reflexes following distension of the urinary bladder in intact, decerebrate and spinal animals. It was observed that the viscero-somatic responses following bladder distension are inhibitory in nature and this inhibition was highest in decerebrates and least in spinal animals. The site of viscero-somatic interaction probably lies in the bulbar area (supraspinal) and spinal cord.

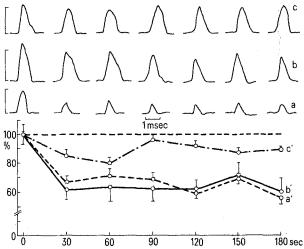
It is well known that dynamic behavior of the somatic muscle is altered with the distension of hollow viscera like the intestine, urinary bladder, uterine horn etc. Downman and McSwiney² showed that pinching or gently squeezing the intestine, head of the pancreas or uterine horn in spinal and decerebrate cats, produced movement of hind limbs. Evans and McPherson^{3,4} have also reported the effects of bladder distension on the monosynaptic reflexes in different experimental conditions and preparations of the animals. The aim of the present investigation is to study the spinal and supraspinal control on

the viscero-somatic reflexes following distension of the bladder in cats.

Materials and methods. The experiments were carried out in cats of either sex, weighing 2–3 kg. Animals were anesthetized with sodium pentobarbitone (Nembutal, Abott) at a dose of 30 mg/kg b.wt i.p. and maintained with a dose of 10 mg/kg b.wt i.v. The urethra was exposed through a midline suprapubic incision. The bladder was cannulated with a polythene catheter via the urethra in order to change bladder volume and monitor intravesicular pressure. The catheter was connected to a

T-tube, one end of which was connected with the pressure transducer, while the other end was used for distension of the bladder with normal saline. Blood pressure and body temperature were monitored and maintained within the normal range. Some cats were decerebrated under ether anesthesia at midcollicular level⁵ and spinal transection was made at the C₇-C₈ level⁶. Laminectomy was performed between segments L₄ and S₂ and the animals were fixed on a cat stand. Ipsilateral dorsal and ventral nerve roots were isolated and sectioned at the level of segment L₂. To study the monosynaptic reflex, the whole bundle of the dorsal nerve root was stimulated with a monophasic square wave pulse (3-5 V, 0.8-1 msec) from a Grass S-48 stimulator delivered through a stimulus isolation unit (SIU 5). Action potentials were picked up from the corresponding ventral nerve root using a silver-silver chloride electrode and recorded on a Tektronix 5113 oscilloscope and photographed with a Philips oscilloscope camera. The resultant effect of bladder distension on the monosynaptic reflex was observed for 180 sec. Collection and analysis of the data were performed as described by Deshpande and Devanandan⁷. In all cases 15 controls were taken into account. The height of the monosynaptic reflexes were measured and expressed as a percentage of the height of the average control reflex. Statistical analysis was performed by Student's t-test and Anova.

Results and discussion. In 15 cats the effects of bladder distension on monosynaptic reflexes were studied. In all cases the bladder was distended with saline with a volume ranging from 30-50 ml. During the filling phase of the bladder, the intravesicular pressure rose sharply to attain a height of 130-150 mm of Hg, but this was immediately followed by a fall to a level of 25-30 mm of Hg at the completion of filling, and it remained at that level as long as the bladder was kept distended. It was observed in intact anesthetized animals that with the distension of the bladder at a pressure head of 25-30 mm of Hg there was significant inhibition of monosynaptic reflexes (p < 0.01) as long as the distension was maintained (fig., a, a'). In midcollicular decerebration, rapid distension of the bladder with saline caused a significant inhibition of monosynaptic reflexes (p < 0.01) which were more pronounced than those observed



Inhibited monosynaptic reflexes recorded at L₇ ventral root of cat. The average height of monosynaptic reflex is expressed as percentage of average control reflex (n = 15) plotted against time at different time intervals during distension of the bladder in anesthetized intact (a'), decerebrate (b') and spinal (c') animals. The results are expressed as mean \pm S.E. (n = 5). The top 3 rows of monosynaptic reflexes are sample sets for intact (a), decerebrate (b) and spinal (c) animals at corresponding time intervals. The control sample is at the O-time. Zero sec. of the time base indicates the time of onset of distension. Vertical calibrations on the left hand side of rows a, b and c represent 4 mV.

in intact animals (fig., b, b'). In acute spinal animals with the cord sectioned at the C₇-C₈ level, rapid distension of the bladder with the same volume of saline caused depression of monosynaptic reflexes (p < 0.05) though to a lesser extent than in intact and decerebrate preparations (fig., c, c').

The present observations indicate that the viscero-somatic responses are inhibitory in nature, and these inhibitions are statistically significant in all cases (intact, decerebrate and spinal animals). Such inhibitions are significantly higher in the case of intact and decerebrate as compared to spinal animals. Afferent pathways for this reflex probably lie in both pelvic and hypogastric nerves⁸⁻¹¹. As regards the centre involved in such processes, the work of de Groat and Ryali¹², de Groat^{13,14}, Morrison⁸ and McMohan and Morrison^{9,10} may be recalled. De Groat and Ryall¹² have suggested that 2 pathways may operate in the micturition process. One is the long loop involving the spinobulbospinal pathways and the other is the short loop involving spinal cord motor neurons. The long loop also involves the pontine micturition center^{13–15}. Existence of such a long loop has also been described by Morrison⁸, McMohan and Morrison^{9,10} in animals where the spinal cord is intact. They further argued that the short loop for micturition is functionally non-existent in the intact animal, however, such a loop is functionally existent in chronic spinal animals^{8-10,12-14}.

From the pathways of the micturition reflex it may be opined that in intact and decerebrate animals, sensations of bladder distension normally reach the bulbar center through spinobulbar pathways. Monosynaptic inhibition that has been observed in such animals may happen through the spinobulbospinal pathways, involving the spinal cord motor neurons as well as bulbar neurons. In the acute spinal animal (C₇-C₈) monosynaptic reflex inhibition is still present, though to a lesser extent, and this finding may suggest that the reflex pathways for such inhibition are mediated through the spinal cord, involving bladder afferents and spinal cord motor neurons.

Thus on the basis of our present observations it may be suggested that both the bulbar area (supraspinal) and the spinal cord are involved in the inhibition of monosynaptic reflexes induced by bladder distension, and probably the contribution of supraspinal components to such reflexes is greater than that of the spinal component.

- Acknowledgment. The work was carried out with the financial assistance of the Indian Council of Medical Research, Govt of India. We are thankful to A. T. Pradhan, Abott Laboratories (India) Pvt. Ltd. for a generous supply of Nembutal.
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