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BRIEF COMMUNICATION

The Functional Relevance of the Lateral Parabrachial Nucleus in Lithium Chloride-Induced Aversion Learning

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AGÜERO, A., M. ARNEDO, M. GALLO AND A. PUERTO. *The functional relevance of the lateral parabrachial nucleus in lithium chloride-induced aversion learning.* PHARMACOL BIOCHEM BEHAV 45(4) 973–978, 1993. – Lesions to the lateral parabrachial nucleus (PBN), one of the subnuclei that make up the pontine parabrachial complex, impairs the acquisition of taste aversion learning (TAL) with LiCl as the toxic stimulus. In this experiment, PBNl-lesioned and control rats were trained to learn a delayed task with a 15-min interval between presentation of the gustatory and the aversive stimulus. The impairment in learning observed after lesions of the PBNl is discussed in terms of disruption of the transmission of toxic stimuli (LiCl) processed by the humoral pathway and the area postrema (AP).

Lateral parabrachial Lithium chloride Taste aversion learning Interstimulus interval Rats

THE pontine parabrachial nucleus (PBN) is composed of two large subdivisions distinguishable by their cytological features: the medial and the lateral portion (14,21,22). These subdivisions apparently differ in their afferent and efferent connections (1,31,35,50,51,53,54) as well as in their specific functional significance (18,19,22,38).

Previous experiments (3–6) have documented the participation of the medial PBN (PBNm) in interoceptive learning (IL), and have shown that this nucleus plays an essential role in the acquisition of short-term taste aversion learning, in which the animal must discriminate, in each session, between two gustatory stimuli presented at the same time, only one of which is associated with simultaneous intragastric administration of an aversive product (10,11).

The lateral PBN (PBNl) receives information of visceral origin from the nucleus tractus solitarius (NTS) and the area postrema (AP) (20,30,34,36,49). On the basis of this visceral nature, and on the basis of anatomical evidence that the PBNl is the next known relay from the AP toward rostral structures of the central nervous system (CNS) (33–35,52,55), it can be hypothesized that blockage of this parabrachial subnucleus may disrupt the transmission of toxic products such as LiCl, processed through the AP (13, 24,47).

In this connection, Borison and Wong (15) discovered that the AP, located in the floor of the IVth ventricle, is an important chemoreceptive zone involved in emetic reflexes (17, 27,32). Moreover, the location of the AP outside the blood-brain barrier, and its consequent susceptibility to a wide range of substances borne by the circulatory system, suggest that the AP is a region that process certain aversive stimuli used in the IL paradigm. This is supported by the finding that lesions to the AP eliminate or attenuate gustatory aversion learned with LiCl, apomorphine, scopolamine, or body rotation as the aversive stimulus (13,24,25,45–48). These findings, in turn, suggest that LiCl acts primarily on the AP, or on nuclei upon which it projects (PBNl). Because the AP projects to the PBNl both directly and indirectly, through the caudal NTS (33, 36,52,55,), we designed the present experiment to test the hypothesis that interruption of the pontine PBNl will block the processing of visceral information encoded by the AP and involved in TAL.

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METHOD

Subjects

Twenty-one male Wistar rats weighing 280–380 g were used. All animals were housed individually in 30 × 15 × 30 cm cages that also served as training chambers during the experiment. A 12L : 12D cycle was used with lights on at 0800 h. Eleven animals were assigned to the control group (sham lesion), and the remaining 10 were assigned to the experimental group (electrolytic lesion of the PBNl).

Surgical Procedure

The animals were operated on a David Kopf stereotaxic instrument for rats (David Kopf Instruments, Tujunga, CA). Electrolytic lesions were made with a DCML-5 lesion generator (Grass Instruments Corp., Quincy, MA) which supplied direct negative current through a monopolar electrode approximately 200 μ in diameter, insulated throughout its length except for the last 0.5 mm.

The stereotaxic coordinates of the PBNl (A-P: −1.4; L: ±2.3; V: +2.6, from the interaural point of reference) were obtained from the stereotaxic atlas of Pellegrino (42).

Surgery was performed under general anesthesia with sodium thiobarbital (50 mg/kg, Lab. Abbot, Spain). The animals were placed in the head holder, and immediately there, two microtrepanations of 1 mm in diameter were made in order to place a electrode over PBNl nuclei. A 1 mA direct negative current was passed through the electrode, over PBNl, for 25 s in the lesioned group. In the sham operated group (control group) the electrode was lowered to the surface of the PBNl nucleus but no current was passed. At the end of the stereotaxic surgical procedure the electrode was removed and the wound sutured. A 0.1 cc dose of penicillin was injected IM (Hoeschst Ibérica, Madrid).

At the conclusion of the experiment the subjects were perfused intracardially and the brains were removed and stored in 10% formaldehyde for a minimum of 48 h. Electrolytic lesions were located in slides prepared with a freezing microtome, and examined under a light microscope (Fig. 1).

Behavioral Procedure

After intracerebral brain surgery the subjects were allowed 2 weeks for recovery, after which an intragastric fistula was implanted (Silastic Medical Grade Tubing, Dow Corning Co., MI). One week was allowed for postoperative recovery. After this period, the animals were given 5 days of adaptation. Rats were placed on a 23 h 53 min water deprivation schedule and allowed to drink tap water for 7 min only from two graduated burets presented simultaneously to avoid the development of positional preferences. After 5 days, the experimental phase was begun according to the procedure shown in Fig. 2 for training and testing, consisting of one daily session during 4 days. Two different gustatory stimuli were available on alternate days. Strawberry (S) and coconut (C) extracts (McCormick and Co. Inc., Baltimore, MD) were diluted in tap water to a concentration of 0.5%. The general procedure consisted of presentation of one gustatory stimulus for 7 min, withdrawal of the stimulus, and intragastric administration of 0.15 M LiCl (Gruppo Montedison, Carlo Erba, Milan, Italy) 15 min thereafter. The rate of LiCl administration was 0.5 ml/l ml of the amount of the gustatory stimulus consumed.

In the second session, 24 h later, another stimulus was presented and the same procedure repeated except that physiological saline (PS) was administered at the end of the 15-min delay. The position of the buret and sequence of the experimental sessions were balanced (Fig. 2).

After two learning trials (four sessions), a choice test was presented: the animals had access to both stimuli simultaneously from burets placed in the same position as in the previous sessions.

RESULTS

The mean amounts of gustatory stimuli (associated with LiCl or PS) consumed during the training phase for both groups are shown in Table 1.

After analyzing the data with Student's *t*-test for paired samples no significant differences were found between the amounts ingested from each stimulus consumed during the choice test by the experimental group (t: 0.97, $p < 0.4$; Fig. 3a). In the control group (sham PBNl lesion), reliable differences were found (t: 5.7, $p < 0.001$; Fig. 3b).

The mean amounts ingested by the lesioned group during choice test were 4.5 ± 3.2 in the aversion condition, and 5.8 ± 3.1 in the nonaversive condition (Fig. 3a). The mean consumption for the control group was 0.9 ± 1.4 in the aversive condition and was 5.5 ± 2 in the nonaversive condition (Fig. 3b).

DISCUSSION

These results show that lesions to the PBNl impair the capacity to perform delayed viscero-gustatory associations in an IL paradigm when LiCl is used as the aversive stimulus. The inability of lesioned animals to learn a viscero-gustatory association is due exclusively to the PBNl lesion, as shown by the finding that control animals subjected to an identical experimental procedure readily learned the task, as several studies in the literature have previously reported (24,25, 45,48).

The impairment in learning may be due to disruption of the transmission of LiCl processed by the humoral pathway through the AP, and subsequently by the PBNl. This impairment caused by lesions to the PBNl appear to be specific to this modality of TAL, as they cause no disruption in other modalities of IL, such as the short-term paradigm (3,6). Shortly this procedure consists of the following: In short-term procedure two gustatory stimuli are presented simultaneously and separately, one of which (properly balanced) is associated with the intragastric administration of an aversive substance, while the other is associated with the administration of physiological saline (PS), or with no injection. Thus, in each experimental trial, the rats are given a choice of two gustatory stimuli presented at the same time. This type of learning short-term requires rapid visceral processing in order to associate the aversive stimulus with the gustatory stimulus being ingested, rather than with the other stimulus available for consumption during the same session. Under these conditions, learning is disrupted only by lesions to the vagus nerve and PBNm system (6,11), but not by lesioning the AP or PBNl (3,6,10).

However, LiCl appears to act through the humoral pathway, another route of visceral information processing, which may act upon structures outside the blood-brain barrier, such as the AP (2,8,13,24,37,47). Sectioning the vagus nerve or PBNm lesions do not disrupt LiCl-induced IL (3,5,8,9). The effect of lesioning the PBNl on LiCl-induced IL is, therefore, unlikely to be due to the interruption of vagal afferent connections from the caudal NTS, i.e., vagal information that may be necessary for LiCl processing. Moreover, the PBNm, con-

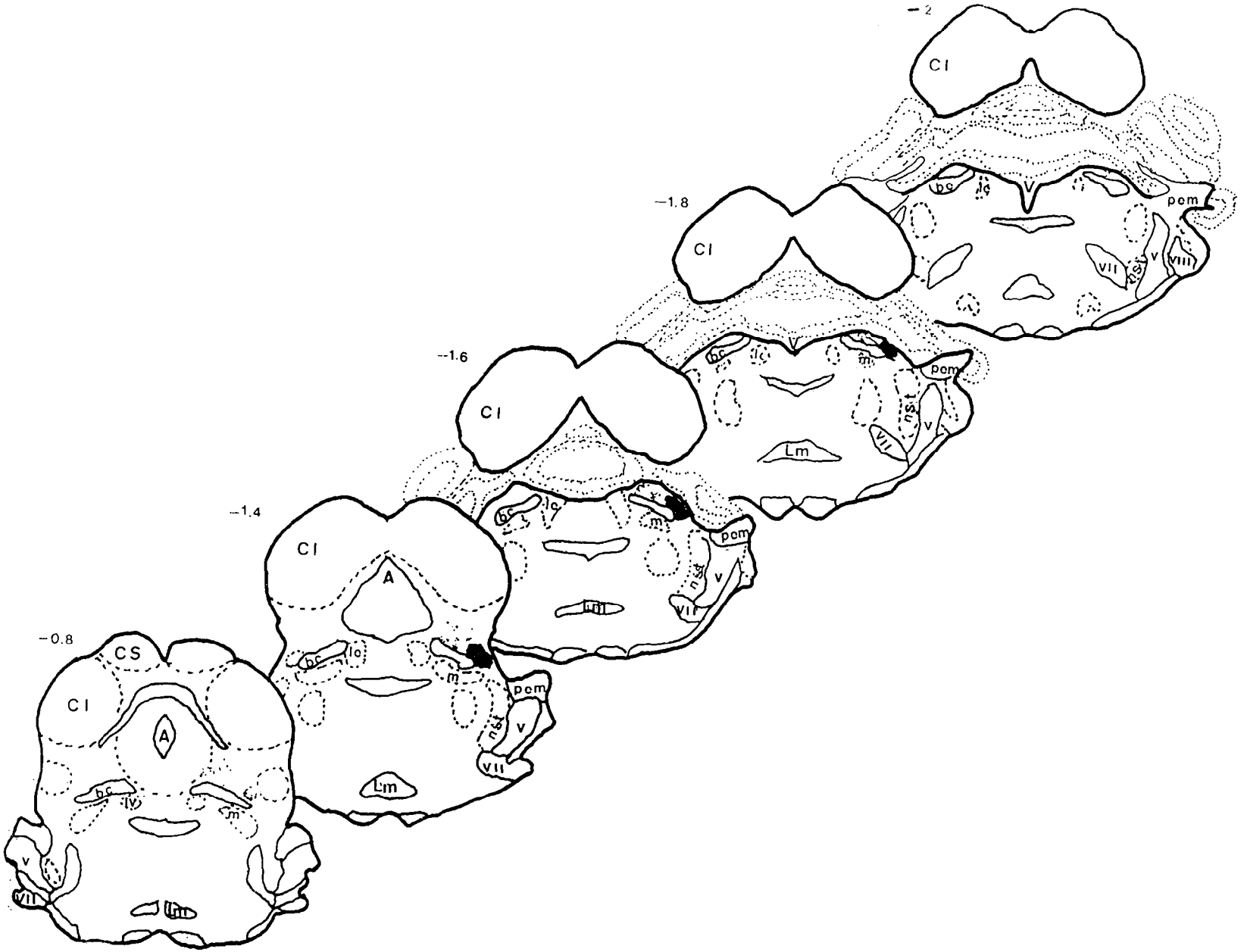

FIG. 1. Charting of representative sections that illustrate relative size of lateral parabrachial nucleus lesions (PBNl). Drawing modified from Pellegrino et al. (42). Abbreviations: A = aqueduct; bc = brachium conjuntivum; CI = inferior colliculus; CS = superior colliculus; lc = locus coeruleus; Lm = medial lemniscus; m = medial parabrachial nucleus; nst = nucleus of solitarius tract; pcm = medial cerebellar peduncle; V = IV ventricle; v = nucleus of cranial nerve V; vii = nucleus of cranial nerve VII; viii = nucleus of cranial nerve VIII.

	SESSIONS			
	1	2	3	4
HALF Animals	S_r 15′ DEL LiCl	C_l 15′ DEL PS	= 1	= 2
HALF Animals	S_r 15′ DEL PS	C_l 15′ DEL LiCl	= 1	= 2

	CHOICE TEST	
HALF Animals	S_r	C_l
HALF Animals	S_r	C_l

FIG. 2. Taste aversion learning paradigm used in experiment. Del, delay; PS, physiological saline; S_r, strawberry on the right; C_l, coconut on the left.

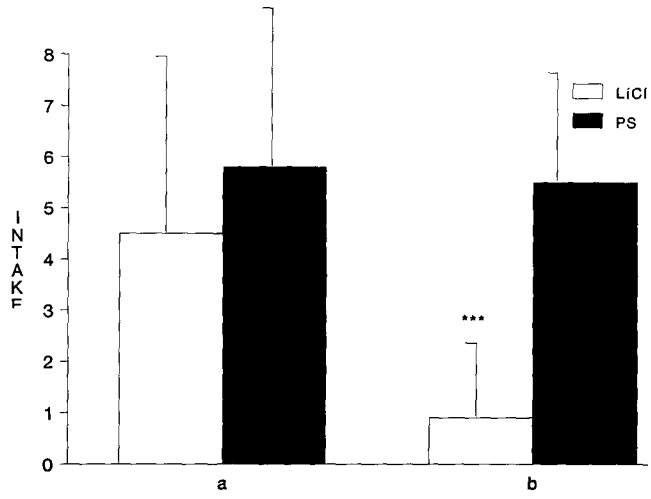

FIG. 3. Mean intake of gustatory stimulus followed by intragastric administration of LiCl (open bars) and physiological saline (PS) (filled bars) during choice test in the control group (b) and PBNl-lesioned (a). ***$p < 0.001$.

stituting the other system available for processing pontine vagal information (29,40), is left intact and is not used in the detection of LiCl. These observations do not rule out the participation of some other peripheral nervous system (e.g., the sympathetic nervous system), which may be involved in LiCl detection. However, even if such a system does participate, it does not come into play in the associative delayed learning task used in the present experiment. As the PBNl is the next relay for information from the AP, it is unsurprising that lesioning this structure disrupted this pathway of visceral information processing.

In summary, on the basis of evidence reported in this study with LiCl as aversive stimulus, the AP and PBNl seems to be the key structures in paradigms similar to that used in the present experiment, in which the slower humoral processing may associate visceral information in the brain with the single novel gustatory stimulus presented, regardless of interstimulus delay (12,26). This procedure seems to involve a specific neurobiological system, and each paradigm requires the use of one or the other route of processing (44).

Therefore, a likely alternative explanation for the blockage of learning seen in the present experiment is disruption of the visceral processing of LiCl, encoded by the humoral pathway, which uses the AP and its next relay, the PBNl, as the mechanism of visceral transmission.

Moreover, because the gustatory pathways use the rostral pole of the NTS and PBNm (39,41,43,56), which in the present experiment were left intact, it is unlikely that processing of the gustatory component was affected.

The impaired coding of visceral information from the AP to the PBNl was recently confirmed in our laboratory (7). Lesions to the PBNl disrupted the effects of electrical stimulation of the AP, shown in recent studies to act as an effective substitute for the aversive stimulus of visceral origin (23).

However, these results do not rule out the participation of other CNS structures in the humoral processing of visceral information. A number of studies have questioned whether the AP alone is the factor exclusively responsible for encoding aversive stimuli. For example, some experiments have shown that animals subjected to intensive learning, by increasing either the dose of product or the number of trials, display learning even after the AP has been lesioned (16,24,45).

Finally, the results described in the present study can be examined in the light of experiments performed by Grill and Norgren (1978) (28) in decerebrate animals. Despite the fact that the lesion in these animals was made at the supracollicular level, and thus left the PBN intact, they failed, even after numerous trials, to perform the TAL task with LiCl as the toxic stimulus. A reasonable interpretation of these findings suggests that, although integrity of the PBNl is required for the integration of viscero-gustatory information, a further requirement is the participation of more rostral structures of the CNS, whose input was disrupted by decerebration. In other words, the relevance of a given structure (in this case the brain stem PBN), does not mean that its participation is

TABLE 1

MEAN INTAKE OF GUSTATORY STIMULI ASSOCIATED WITH LiCl OR PS DURING THE TRAINING

	Trial 1		Trial 2	
Subjects	LiCl	PS	LiCl	PS
Experimental group				
Mean	9.65	9.3	10.1	9.6
SE	4.2	3.83	3.74	4.64
Control group				
Mean	10.09	9.27	7.8	7.2
SE	1.35	2.8	2.2	2.47

independent of other constitutive areas of the neurobiological system of which it forms part, although not necessarily as a fundamental center of convergence. This issue remains to be examined in depth by researchers interested in IL. In this connection, some studies have shown neural activity in the PBN to be influenced by areas of the hypothalamus and cortex (20,57); such centrifugal regulation may prove to be necessary for learning.

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