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IMPACT OF ANTRAL MECHANORECEPTOR ACTIVATION ON THE VAGO-VAGAL REFLEX IN THE RAT: FUNCTIONAL ZONATION OF RESPONSES

BY MONICA J. MCCANN AND RICHARD C. ROGERS

From the Department of Physiology, The Ohio State University College of Medicine, 333 West Tenth Avenue, Columbus, OH 43210, USA

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SUMMARY

1. Activation of gastric sensory afferents alters gastric motor and secretory function via the gastric vago-vagal reflex. In this report, we investigated in the rat the impact of gastric mechanoreceptor activation on the brain stem components of the reflex, which are located in the dorsal vagal complex (DVC), i.e. the nucleus of the solitary tract (NTS) and the subjacent dorsal motor nucleus (DMN).

2. In our extracellular recordings of single-cell activity in the DVC, we observed a relation between the response to antral distention and the location of the cell in the DVC. Specifically, cells that were excited by antral distention (ON cells) were located dorsal to those that were inhibited (OFF cells) by the same stimulus (mean depth = 536 ± 15 and 627 ± 14 μm for ON and OFF cells, respectively).

3. For a subset of DVC cells, the location was marked by ionophoretic ejection of Pontamine Blue from the recording barrel. Histological analysis indicated that ON cells were located in the NTS, and OFF cells were located in the ventral NTS or within the boundaries of the DMN. Together, these data led to the hypothesis that ON and OFF cells are functionally different groups of neurones, i.e. ON cells may be NTS neurones, and OFF cells may be DMN neurones. We tested this directly by employing both an intragastric balloon and a non-traumatic vagal stimulating electrode to determine whether inflation-related cells were NTS or DMN cells via orthodromic and antidromic activation, respectively.

4. Almost all ON cells (12/13) were orthodromically activated by vagal stimulation, i.e. they were NTS neurones. One ON cell was antidromically activated, and therefore was a DMN neurone. Of the twenty-eight OFF cells that were encountered, ten were classified as NTS neurones because they were orthodromically inhibited by vagal stimulation. The remaining eighteen OFF cells were orthodromically inhibited *and* antidromically activated (i.e. DMN neurones). Thus, our results support the hypothesis that ON and OFF cells can be functionally distinct populations of neurones, in that almost all ON cells are NTS cells and approximately 2/3 of the OFF cells are DMN neurones.

5. The response to mechanoreceptor activation was different for NTS and DMN neurones. NTS cells were activated (55%) or inhibited (45%) by balloon distention of the stomach, whereas DMN cells were almost exclusively inhibited (95%) by this

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stimulus. This information provides insight into the organization of excitatory and inhibitory connections of the brain stem components that mediate gastric vago-vagal reflexes.

INTRODUCTION

The reflex control of gastric smooth muscle and secretory epithelium depends on complex hierarchial interactions between local reflexes, involving the myenteric and submucosal plexuses in the enteric nervous system, and long-loop reflexes, involving the parasympathetic and sympathetic branches of the autonomic nervous system (Diamant, Hall, Mui & El Sharkawy, 1980; Wood, 1986). Of the long-loop reflexes, the parasympathetic or 'vago-vagal' reflex exerts a dominant influence over sensory and motor functions of the proximal gut. The sensory limb of the vago-vagal reflex originates in the stomach and terminates in the nucleus tractus solitarius (NTS) in the caudal brain stem (Kalia & Mesulam, 1980; Shapiro & Miselis, 1983). The NTS neurones that receive afferent input from the stomach can influence the activity of the vagal preganglionic neurones in the dorsal motor nucleus (DMN), which lies just ventral to the NTS. These vagal efferents project to enteric motor or secretory neurones in the stomach, thereby completing the reflex loop (Takayama, Ishikawa & Miura, 1982; Fox & Powley, 1985; Schemann & Grundy, 1991). There is also anatomical evidence for a reflex loop in which primary vagal afferents communicate directly with DMN neurones (Shapiro & Miselis, 1985; Rinaman, Card, Schwaber, & Miselis, 1989).

Via these reflex pathways, vagal sensory neurones can evoke changes in gastric function. For example, stimulation of vagal afferents increases acid secretion from the parietal cells, and decreases gastric pressure (Harper, Kidd & Scratcherd, 1959; Grossman, 1962; Jansson, 1969). Because these effects can be elicited by electrical stimulation of the central cut end of the abdominal vagus and are attenuated by abdominal vagotomy, these physiological responses are mediated, in part, by gastric vago-vagal reflexes (Harper *et al.* 1959).

Activation of gastric mechanoreceptors by distending the antrum has been shown to activate and inhibit the firing rate of cells in the DVC (Harding & Leek, 1973; Barber & Burks, 1983; Ewart & Wingate, 1983) and influence gastric function (Abrahamsson, 1973). In the present report, we observed a dorsal/ventral pattern in the location of cells that had increased (ON) or decreased (OFF) activity in response to gastric antral inflation. This led to the hypothesis that ON cells were NTS cells, whereas OFF cells could be DMN neurones. Although the histological and location data are supportive of this hypothesis, they cannot be used as conclusive evidence because it has been demonstrated that dendrites of DMN neurones extend dorsally into the NTS region (Shapiro & Miselis, 1985). Thus, based on histological evidence alone, a DMN cell could be falsely classified as a NTS neurone. Therefore, we unambiguously identified inflation-related cells as DMN or NTS neurones using electrophysiological criteria (Fuller & Schlag, 1976; McCann & Rogers, 1990), in order to determine the impact of antral mechanoreceptor activation on identified components of the vago-vagal reflex.

METHODS

Subjects

Male Long–Evans rats ($n = 38$) weighing 300–450 g were anaesthetized with urethane (1·5 g/kg I.P.). A cannula was inserted into the trachea of each animal to maintain an open airway. Also, all animals were pretreated with dexamethasone (0·8 mg, S.C.) to minimize cerebral swelling.

Surgery

Gastric surgery. An intragastric balloon, created from the thumb of a latex glove and a piece of Silastic tubing, was inserted into the stomach through the pylorus. The balloon was positioned in the antrum and secured with sutures around the pylorus. The abdominal wall was closed with the distal end of the tubing exiting the incision. The tubing was connected to a Stratham P23 pressure transducer to measure inflation-induced pressures. The antrum was inflated by introducing 2–3 ml of air into the balloon to achieve a pressure of 10 cmH_2O.

In another group of six rats, gastric distention was achieved by introducing air into the stomach. To accomplish this, a tube was inserted via the pylorus and the pylorus was ligated around the tube. The stomach was inflated by introducing 3–5 ml of air via a syringe attached to the tube. This volume of air produced an intragastric pressure comparable to that observed with balloon inflation of the antrum alone (8–10 cmH_2O).

Vagal stimulating electrode placement. In studies involving the identification of brain stem vagal neurones using electrophysiological criteria, a non-traumatic stimulating electrode cuff was placed on the cervical vagal trunk of fifteen animals. Vagal stimulating electrodes were constructed from two 30 gauge wires inserted into a 0·5 cm piece of silicon tubing which was slit lengthwise to resemble a cuff. The cervical vagus was carefully isolated from the carotid, and the cuff was slipped around the nerve trunk. A suture was placed around the cuff to insure contact of the nerve with the wire, with care taken not to damage the vagal trunk. Additional sutures anchored the wires exiting the cuff to surrounding muscle. The incision was closed, and the leads were connected to a constant-current isolation unit (WPI) that was driven by a conventional stimulator (Grass S88). Stimulation pulses were typically 700–1000 μA, 0·5 Hz, 0·5 ms pulse duration.

Brain stem exposure. After abdominal surgery, the animal was placed in a stereotaxic apparatus. The occipital plate was removed to expose the dorsal surface of the brain stem using methods described previously (Rogers & Nelson, 1984).

Procedure

Extracellular recording. A single-barrel glass pipette, filled with 2 M-NaCl and 1% Pontamine Blue dye, was used to record single-unit extracellular potentials in the DVC. The signals were amplified (Grass P-15 amplifier), displayed on a variable persistence storage oscilloscope (Tektronix 5441), monitored on a chart recorder and stored on magnetic tape for subsequent analysis.

After exposing the brain stem, the recording electrode was positioned over the medial NTS. The electrode was lowered using a Kopf micropositioner while the antrum was inflated 2–3 times per minute until an inflation-related cell was located. A cell was considered to be responsive to inflation if the spontaneous firing rate was altered by 50% during the inflation/deflation cycle. For each responsive cell encountered, the position of the electrode in the medial–lateral/anterior–posterior planes (relative to obex) and the depth (relative to the dorsal surface of the brain stem) were noted.

In animals with vagal stimulating electrodes, the stomach was inflated 2–3 times per minute as the recording pipette was lowered into the brain stem, until an inflation-sensitive cell was encountered. Then, the vagus nerve was stimulated via the cuff electrode. If the response to electrical stimulation was characterized by (a) variable latencies, and (b) failure to respond to short interval, dual pulse stimulation (frequencies = 50 Hz), then the cell was considered to be an orthodromically activated NTS neurone. In contrast, if the response was characterized by (a) constant latency, (b) a following of high frequency dual pulse stimulation (frequencies = 50–100 Hz), and (c) collisions with spontaneous action potentials, then the cell was considered to be an antidromically activated DMN neurone.

Histology. In some animals ($n = 16$), the position of an inflation-responsive cell was marked by the injection of Pontamine Blue dye from the recording barrel using ionophoretic methods. At the termination of the experiment, the animal was perfused transcardially with isotonic saline,

followed by 3% formalin. The brain was removed from the skull and the tissue was permitted to fix in formalin for at least 24 h. Then, the brain stem was cut in 60 μm sections, and stained with neutral red to visualize the location of the spot.

Data analysis. In the mapping studies, the midpoint of a track was considered to be equidistant from the most dorsal point and the most ventral point where inflation-related cells were found. The number of ON and OFF cells located above or below the midpoint for each track was determined. A random distribution would produce a pattern whereby half of the ON cells are above and half are below the calculated midpoint. The same would be true for the distribution of OFF cells. A difference from a random distribution was evaluated statistically using a χ^2 analysis. Also, the average depth of ON *vs.* OFF cells was compared using Student's t test.

RESULTS

Brain stem map of antral distention-related neurones

In our recording experiments, a total of 105 gastric inflation-related cells were encountered in the DVC. Of these neurones, fifty-seven (54%) had elevated firing rates in response to gastric inflation (ON cells; Fig. 1, left panel) whereas the activity of the remaining forty-eight (46%) were inhibited by the same stimulus (OFF cells; Fig. 1, centre panel). Another cell type was also found. These cells had a cycling pattern of firing characterized by augmented firing frequencies occurring 4–5 times per minute (Fig. 1, right panel). The activity of the cycling cells did not appear to be influenced by gastric inflation. The cycling neurones were located dorsal to or among the ON cells in the DVC. Below the ON cells, OFF cells typically were found. Within a single track, OFF cells were always found ventral to the ON cells. The average depth of ON cells was 536 ± 15 μm, whereas the average depth of OFF cells was 627 ± 14 μm ($t = 4{\cdot}38$, d.f. $= 103$; $P < 0{\cdot}0001$). A plot of the location of inflation-responsive cells is shown in Fig. 2. When the depth *vs.* the distance from obex in the anterior/posterior plane was plotted for each inflation-sensitive cell, a dorsal/ventral arrangement was apparent for ON and OFF cells, respectively. This pattern was statistically different from a random arrangement ($\chi^2 = 21{\cdot}8$; d.f. $= 1$, $P < 0{\cdot}01$). These findings were supported by histological evidence. Pontamine spots that marked ON cells were found exclusively in the NTS. Some OFF cells were found in the NTS, but other OFF cells were located within the boundaries of the DMN.

Identification of gastric distention-related cells as NTS and DMN neurones

Air vs. balloon

Both methods of distending the stomach altered the activity of cells in the DVC. However, air inflation produced a less robust effect on firing rate compared to balloon inflation. Because both ON and OFF responses were observed with either method of inflation, the data were combined and are shown in Table 1.

Identification of ON and OFF cells

Numerous cells in the DVC were recorded that responded to inflation, which indicated that the placement of the cuff on the vagus did not interfere with neural transmission from the stomach to the brain stem. Cells that were excited by inflation (ON cells) were almost exclusively (12/13) orthodromically activated by vagal stimulation; these were, therefore, NTS neurones (Fig. 3*A* and *B*). The remaining ON

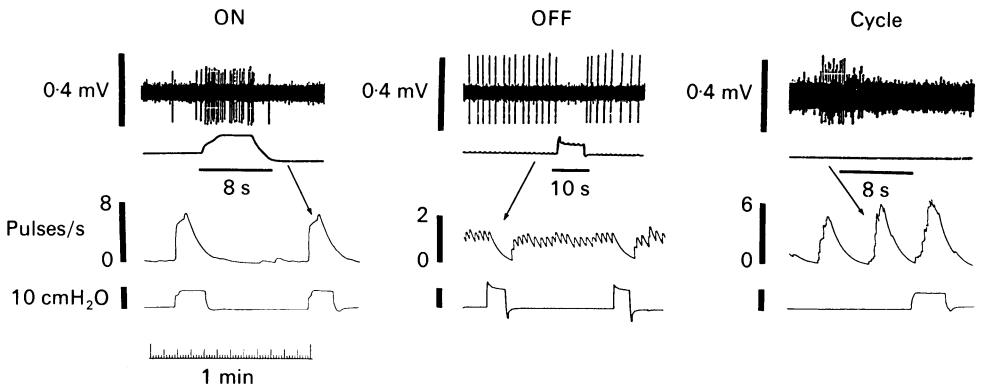

Fig. 1. Extracellular recordings of single-unit activity in the dorsal vagal complex of the rat. Oscillograph (upper traces) and ratemeter records (middle traces) illustrate the changes in firing rate of an ON cell, an OFF cell and a cycling cell in response to an increase in gastric pressure (lower traces).

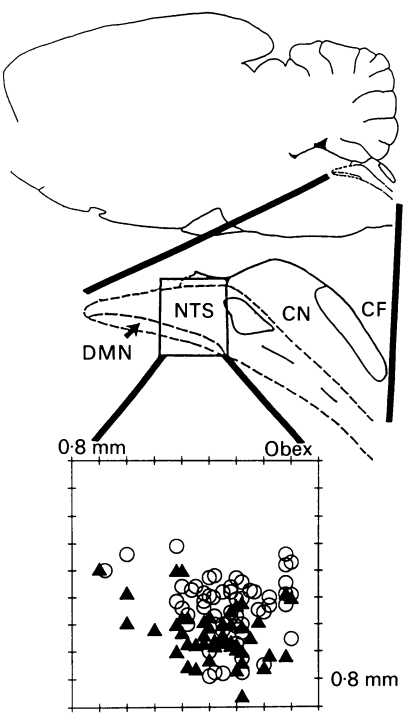

Fig. 2. Sagittal view of the rat brain, indicating the region of the brain stem where ON and OFF cells were encountered. The map indicates the location of ON cells (○) and OFF cells (▲) in the rostral/caudal and dorsal/ventral planes. Abbreviations: CF = cuneate fasciculus, CN = cuneate nucleus, NTS = nucleus of the solitary tract, and DMN = dorsal motor nucleus.

cell was antidromically activated and therefore classified as a DMN neurone. Thus, the vast majority of cells (92 %) that were activated by inflation were NTS neurones.

Of the cells that were inhibited by gastric inflation (OFF cells), ten were

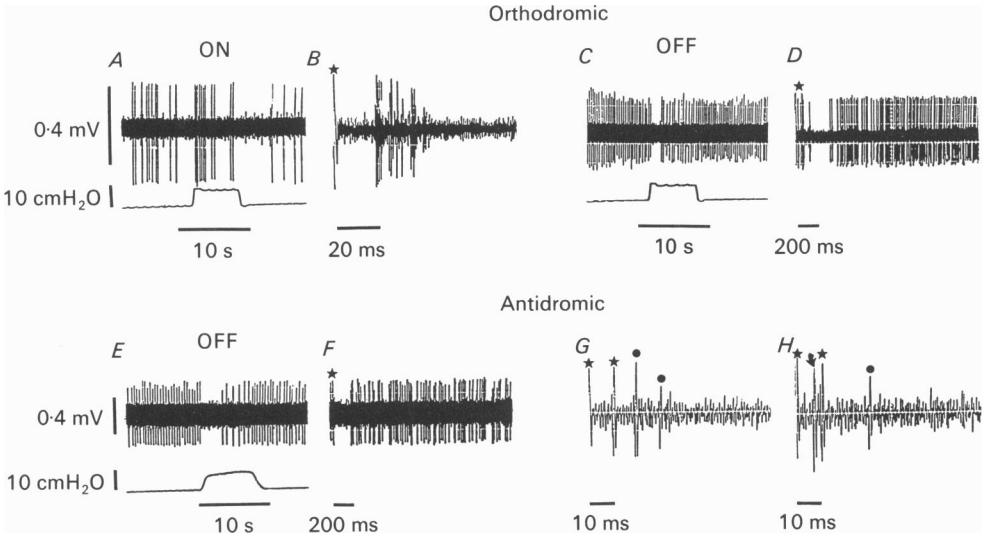

Fig. 3. Identification of inflation-related cells as NTS or DMN neurones using electrophysiological criteria. Panel *A* illustrates an increase in unit activity upon elevation of gastric pressure, i.e. an ON cell. This cell also was activated by vagal stimulation (panel *B*, ★ = stimulation artifact). Because the latency to respond to stimulation was variable, the cell was classified as an orthodromically activated NTS neurone. An OFF cell, which has a decrease in firing rate upon distension of the stomach, is shown in panel *C*. This cell was also classified as an NTS cell; it was orthodromically inhibited by vagal stimulation (panel *D*). Note the absence of action potentials in this cell for approximately 200 ms after vagal stimulation. The OFF cell that is represented in panel *E* was also orthodromically inhibited (panel *F*) by stimulating the vagus (★ = stimulation artifact). In addition, panel *G* shows that this cell had constant latency responses (●) to dual pulse activation of the vagus (★). Panel *H* illustrates a 'collision' of a spontaneously occurring action potential (arrow) with the first antidromic potential (compare panels *G* and *H*). Because this cell was antidromically activated by vagal stimulation, it was classified as a DMN neurone.

TABLE 1. Average depth of ON and OFF cells that were physiologically identified as NTS or DMN neurones

	Depth in μm $\pm$ S.E.M. (n)	
	ON	OFF
Orthodromic (NTS)	438 $\pm$ 26* (12)	572 $\pm$ 43* (10)
Antidromic (DMN)	439 (1)	635 $\pm$ 32 (18)

* Significantly different from OFF/antidromic ($P < 0{\cdot}05$).

orthodromically inhibited by vagal stimulation; these were classified as NTS neurones (Fig. 3*C* and *D*). An additional eighteen cells were inhibited by inflation and by electrical stimulation of the vagus (Fig. 3*E* and *F*). Also, these cells showed clear evidence of antidromic activation (Fig. 3*G* and *H*). Thus, for the OFF cells, activation of the afferent path either by inflating the stomach or electrically stimulating the vagal trunk resulted in an inhibition of spontaneous activity.

Approximately 36% of the OFF cells were considered to be NTS neurones and 64% were vagal efferents in the DMN.

To summarize, activation of gastric mechanoreceptors either enhanced (55%) or inhibited (45%) the firing rate of NTS cells, whereas DMN cells were almost exclusively inhibited by this stimulus (95%). Only one DMN cell was activated by gastric inflation.

Location of ON and OFF cells in the DVC

When the average depths of the cell types were compared, a dorsal/ventral arrangement of ON/OFF cells was apparent. This is in agreement with the results of the initial studies. The depth of the ON/orthodromic neurones was significantly less than the OFF/orthodromic cells ($t = 2{\cdot}83$, d.f. $= 20$; $P < 0{\cdot}025$) and the OFF-/antidromic cells ($t = 4{\cdot}42$, d.f. $= 28$; $P < 0{\cdot}0001$). The depth of the single ON/antidromic neurone was more similar to the average depth of the ON cells rather than OFF cells.

Cycling cells

Cycling neurones ($n = 18$) were found in the dorsal aspect of the DVC, as was observed in previous experiments. The average depth of these cells was 388 ± 17 μm. It is interesting to note that neither gastric inflation nor electrical stimulation of the vagus appeared to influence the firing pattern of the cycling cells.

DISCUSSION

It has been shown in several species that gastric sensory information can alter the activity of gastric vagal efferent neurones and gastric function via the vago-vagal reflex (cat: Harper *et al.* 1959; Jansson, 1969; Abrahamsson, 1973; dog: Grossman, 1962; and ferret: Grundy, Salin & Scratcherd, 1981). What remained uninvestigated was *how* gastric sensory input influenced the brain stem components of the reflex to result in a change in efferent activity. In the present study, we explored in the rat the internal organization of this 'black box' in the brain stem. Although we cannot rule out species variations, our results may provide insight into the neural basis of vago-vagal reflex-induced changes in gastric function that are observed in these species.

Our initial observation was that DVC neurones with increased activity upon gastric inflation (ON cells) were located dorsal to those that had reduced activity to the same stimulus (OFF cells). These findings led to the hypothesis that ON cells are NTS neurones, and OFF cells could be DMN neurones. The histological data, although inconclusive, supported this view. Therefore, we tested this hypothesis directly by using electrophysiological criteria to identify inflation-related cells as NTS or DMN neurones.

We found that the large majority of ON cells were orthodromically activated, i.e. they were NTS neurones. A subset of the OFF cells were orthodromically inhibited, but not antidromically activated, by electrical stimulation of the vagus. Thus, these cells were also considered to be NTS cells. Some of the OFF cells were not only orthodromically inhibited, they were also antidromically activated by vagal

stimulation. In other words, they were DMN cells that received inhibitory afferent input. Therefore, our results support the hypothesis that ON and OFF cells can be functionally distinct populations of neurones, in that almost all of the ON cells are NTS cells, and approximately two-thirds of the OFF cells are DMN neurones.

Functional significance of the ON–OFF cells

The ON and OFF cells that are NTS neurones may be part of the reflex loop, i.e. they may project to DMN neurones. Also, they could contribute to the vago-vagal reflex pathway that involves vagal preganglionic neurones in the nucleus ambiguus (Bieger & Hopkins, 1987). Alternatively, they could transmit sensory information to integration regions of the brain (parabrachial nucleus, hypothalmus, bed nucleus of the stria terminalis, central nucleus of the amygdala, etc.); these nuclei send projections to the DVC (Sawchenko, 1983) and are thought to modulate vago-vagal reflexes (Hermann & Rogers, 1989).

The physiologically identified DMN cells, which were predominantly OFF cells, are most likely related to gastric function because their activity was influenced by antral inflation. Also, the region of the DMN where we recorded corresponds to the site of predominantly gastric preganglionic vagal neurones (Takayama *et al.* 1982; Fox & Powley, 1985). Gastric preganglionic efferents can synapse with either excitatory (cholinergic) or inhibitory (non-adrenergic non-cholinergic, probably peptidergic or purinergic) postganglionic neurones in the enteric plexus of the stomach (Gillespie, 1982; Wood, 1986). Antral inflation was found to inhibit the firing rate of all but one of the DMN neurones. Therefore, inflation has the potential to influence gastric function by either reducing the activity of the excitatory projection (disfacilitation) or reducing the activity of the inhibitory projection (disinhibition). Gastric inflation could potentially reduce or enhance gastric secretory or motor activity.

It is currently not possible to determine whether the DMN cells that we recorded influence secretory epithelium or gastric smooth muscle. However, based on the known physiological effects of antral inflation on these target tissues, we hypothesize that the recorded neurones might be involved in the neural control of smooth muscle tension in the fundus, and not involved with the neural control of acid secretion. Specifically, it is known that gastric inflation increases acid output, partly by local enteric reflex actions and partly by *activating* vago-vagal excitatory cholinergic efferents (Grossman, 1962). Because our findings indicate that the large majority of efferents would have *reduced* activity in response to antral inflation, the majority of DMN cells we recorded most likely do not project to neurones controlling acid secretion.

On the other hand, gastric inflation *reduces* pressure in the fundus via a vago-vagal reflex mechanism (Jansson, 1969; Abrahamsson, 1973). An inhibition of gastric pressure could be due to an activation of inhibitory neurones (active inhibition) and/or an inhibition of excitatory neurones (disfacilitation). Substantial evidence can be found for the operation of both mechanisms. For example, Jansson (1969) reports that stimulation of the central cut end of the abdominal vagus elicts a large drop in gastric tone. Atropinization attenuates the reduction in tone produced by vagal stimulation whereas vagotomy completely eliminates it. Two lines of evidence suggest that activation of the antral mechanoreceptors is producing disfacilitation:

(1) in the rat, antral inflation inhibits the activity of a large majority of DMN neurones (present report), and (2) the findings reported by Miolan & Roman (1978) indicated that much of the tonic input to the stomach is excitatory. Activation of the vago-vagal reflex resulted in a reduction in efferent activity and gastric pressure. Therefore, we propose that in the rat, antral inflation inhibits the excitatory cholinergic vagal efferents that influence fundic smooth muscle tension, resulting in a reflex-mediated reduction in gastric pressure. This does not rule out the possibility that active inhibition plays a role in mediating reflex-induced gastric relaxation in other species (e.g. Jansson, 1969) or other vago-vagal reflexes in the rat.

Functional significance of the cycling cells

In addition to ON and OFF cells, another type of cell in the DVC was observed that had a cycling pattern of firing. It was of interest that these cycling cells had a rate of bursting that coincides with the typical frequency of gastric contractions, i.e. 4–5/min. Davison (1987) observed similar cycling pattern in efferent fibres isolated from the vagal trunk. It is tempting to speculate that the cycling cells in the brain stem are 'central rate generators' that determine the rate of gastric contractions. However, it is clear that the cycling neurones are not vagal efferents because they were not antidromically activated by vagal stimulation. Furthermore, it is doubtful that the cycling cells influence the activity of DMN neurones projecting to the stomach, because we did not observe cycling in any DMN cells that we recorded. Indeed, the cycling cells may not be involved in the vago-vagal reflex at all, given that they were not influenced by vagal afferent stimulation. Thus, the contribution of these spontaneously cycling cells to the vagal control of gastric function remains elusive.

Possible mechanisms mediating the gastric vago-vagal reflex

It is clear that the ON/OFF responses of DVC neurones upon gastric inflation result from the activation of the primary vagal afferents. These responses can be elicited by electrical stimulation of the vagal trunk and are lost after vagotomy (Davison & Grundy, 1980; Grundy *et al.* 1981). Because DVC cells can be either excited or inhibited by vagal afferent stimulation, it is possible that the primary afferents contain excitatory or inhibitory transmitters. With regard to putative excitatory transmitters, there is evidence that glutamate is released from some vagal afferent neurones (Perrone, 1981; Meeley, Underwood, Talman & Reis, 1989). Indeed, glutamate may mediate the increased activity of ON cells when the stomach is inflated. In preliminary studies from our laboratory, we have demonstrated that ON cells were also activated by ionophoretically applied glutamate (Rogers, McCann & Stephens, 1990). More significantly, both glutamate- and inflation-induced excitations were blocked by the glutamate antagonist, kynuretic acid. These findings suggest that, upon gastric inflation, glutamate may be released from the gastric mechanoreceptor afferents that project to the NTS. Furthermore, both activation of antral mechanoreceptors and injection of glutamate in the medial NTS results in a reduction of gastric pressure (Spencer & Talman, 1986).

With regard to the mechanisms mediating an OFF response, two possibilities exist. First, the primary afferents could be releasing an inhibitory transmitter upon inflation. This would reduce DMN activity directly if the primary afferents synapse

on DMN cells. The cell bodies of primary vagal afferents in the nodose ganglia contain catecholamines and various peptides (e.g. Mantyh & Hunt, 1984) which may serve as inhibitory transmitters in the mechanoreceptor afferent projection to the NTS.

Second, it is possible that an NTS neurone contains an inhibitory transmitter. Thus, activation of vagal afferents would excite an NTS inhibitory interneurone, resulting in a decreased activity in the cell on which it synapses, i.e. an OFF cell. If this OFF cell is a DMN cell, then this represents another possible mechanism by which vagal afferent activation can inhibit DMN cell activity. Candidates for inhibitory transmitters in NTS interneurones include the catecholamines noradrenaline and adrenaline. Using electron microscopic analysis, some functionally unidentified primary afferents were shown to synapse on tyrosine hydroxylase positive NTS neurones (Siaud, Denoroy, Assenmacher & Alonso, 1989). Also, phenylethanolamine *N*-methyltransferase positive (presumably adrenaline-containing) NTS neurones have been shown to project onto DMN neurones (Pickel, Chan, Park, Joh & Milner, 1986; Siaud *et al.* 1989). The role of catecholamines as putative transmitters in the gastric vago-vagal reflex warrants further investigation.

In conclusion, this report represents the first account of the response of physiologically identified brain stem vagal neurones to mechanoreceptor activation. This information provides insight into the possible organization of the brain stem circuits that underlie gastric vago-vagal reflexes.

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