Localized myocardial responses to stimulation of small cardiac branches of the vagus

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Armour, J. A., W. C. Randall, and S. Sinha. Localized myocardial responses to stimulation of small cardiac branches of the vagus. Am. J. Physiol. 228(1): 141-148. 1975.—Direct electrical excitation of small cardiac branches from the thoracic vagus elicited highly localized and differential responses from individualized segments of the myocardium. For example, small nerves from the vagus at the level of the superior pulmonary veins frequently induced moderate inhibition in contrast to the ipsilateral atrium with little or no influences elsewhere. Branches from more rostral levels of the thoracic vagus induced changes in atrial contractility, with or without changes in sinoauricular (SA) nodal discharge rates, and were often associated with atrioventricular (AV) nodal blockade. Excitation of individual, small vagal branches sometimes initiated acceleration in atrial rate and augmentation in atrial contractile force concurrently with complete AV nodal blockade. The negative chronotropic response was eliminated by atropine, leaving only the positive chronotropic and inotropic changes, thus revealing the intermingling of both sympathetic and parasympathetic components even in these small branches. There are frequently in excess of 20 small branches from the vagal trunk between the level of the caudal cervical ganglion and the superior pulmonary vein on each side which will induce highly selective changes in cardiac function upon stimulation. Inhibitory branches are particularly concentrated in the region of the recurrent laryngeal nerve on either side.

Myocardial innervation; vagal innervation of the heart; inotropic, chronotropic, dromotropic responses to vagal stimulation; vagal stimulation of the systemic circulation; effects of vagal stimulation on systemic vascular resistance; and the influence of vagal stimulation on heart rate.

The bradycardia, slowed atrioventricular (AV) nodal conduction, and inhibition of atrial contractility which characterize efferent cardiomyotrophic influences of vagal stimulation are well known. It is generally held that direct branches from the vagus to the heart are less numerous than those issuing from the sympathetic system, and indeed, one of the most widely quoted works in the field states that there are only two, one on either side, which can properly be termed “cardiovagal” nerves (15). Surprisingly few investigators have studied specific distributions of the vagal efferent pathways to the heart, particularly in combination with techniques which reveal differential functional effects of vagal control. Barry (2) recognized two anatomical groupings of cardioinhibitory branches of the dog’s vagus, one arising from the region of the caudalcervical ganglion and the other arising between the origin of the recurrent laryngeal nerve and the root of the lung. By studying electrocardiograms while stimulating various segments of the cervical and thoracic vagosympathetic trunk, Mizieres (11, 12) described individual cardioinhibitory branches of the right and left vagi in the dog. Unfortunately, he was unable to differentiate inhibitory influences other than those associated with heart rate. The anatomical course of the vagus nerves in the dog’s thorax has been described, but little emphasis has been placed upon its innervation of the heart (8). Cooper et al. (5) devised a chronic preparation in which the canine heart was freed of vagal control, while lungs, stomach, and esophagus remained innervated. Again, only heart rate data were derived from the experiments.

In earlier studies of the projections of the branching sympathetic nerves to the heart (18), we became aware of the rich intermingling of sympathetic and parasympathetic fibers in many of the cardiac nerves distal to the caudal cervical ganglion. Atropine and propranolol were employed to separate adrenergic from cholinergic responses. The anatomical course (6) and fiber components (7, 13) of the small cardiac nerves and the results of simultaneous excitation of sympathetic, parasympathetic, and afferent nerves within the vagosympathetic trunk have been reported (16, 17). However, a systematic experimental design has not previously been proposed to isolate and electrically stimulate each individual branch of the thoracic vagi while recording chronotropic, inotropic, and dromotropic events from the heart. This paper presents, therefore, a first description of the vagal origins of individual thoracic vagal branches, together with the simultaneous recordings of their multiple and differential influences on heart function.

Methods

Experiments were carried out in 22 anesthetized (phencyclidine HCl, 2.0 mg/kg, and alpha-chloralose, 60-80 mg/kg) open-chest dogs. Walton Brodie strain gauge arches were sutured under moderate tension to each of six myocardial segments including right atrial (RA) and left atrial (LA), right ventricular conus (RVC), right ventricular sinus (RVS), left ventricular base (LVB), and left ventricular lateral surface (LVL). Femoral arterial blood pressure (BP) and lead II of the electrocardiogram (ECG) were also recorded, all on an eight-channel model R Offner dynograph. Square-wave pulses were delivered from a Grass model S4 stimulator with stimulation parameters of 20-40 Hz, 2-5 ms, and 1.8-8 V, each parameter being monitored during stimulation on a cathode ray oscilloscope. The thoracic vagus was carefully dissected from the mediastinal
pleura, and each individual nerve branching from it between the caudal cervical ganglion and the level of the superior pulmonary veins on each side was prepared for separate electrical stimulation. Each vagosympathetic trunk was transected in the neck in order to ascertain efferent stimulation.

RESULTS

Left thoracic vagus. There is a dense network of very fine nerves branching from the left recurrent laryngeal nerve as well as from the main trunk of the thoracic vagus down to the region of the left atrium, pulmonary and aortic arches, and along the pulmonary veins to the lungs. While electrical excitation of these individual nerves frequently elicited cardioinhibitory responses when the ipsilateral vagosympathetic trunk was intact (16), this rarely occurred after its transection. Figure 1 illustrates the actual branching of grossly evident nerves from the left and right thoracic vagi in two different animals which were employed to illustrate the functional responses in Figs. 2 and 3. Each branch was carefully isolated and stimulated sequentially, proceeding from the level of the pulmonary veins to a point immediately rostral to the caudal cervical ganglion. Nerves which induced a predominantly sympathetic response were neglected for the purposes of this report, since they have been described previously (18). A few experiments were carried out before and after atropinization, however, and the com-

![Figure 1](http://ajplegacy.physiology.org/)

**FIG. 1.** Sketch of small branches arising from right and left thoracic vagi in different animals which were employed for experiments illustrated in Figs. 2 and 3. While all of nerves shown were electrically stimulated, only those responses designated by letter are illustrated (in Figs. 2 and 3). Many of the nerves elicited cardiac responses which could not be reproduced due to lack of space. Relatively larger branches from the region of the caudal cervical ganglia have been shown elsewhere to contain predominantly sympathetic fibers, and thus responses to their stimulation are not incorporated in this report.

![Figure 2](http://ajplegacy.physiology.org/)

**FIG. 2.** Responses to stimulation (10 Hz, 5 ms, 4 V) of small branches from left thoracic vagus at positions indicated in Fig. 1 (letter designations at top) of Fig. 2. Simultaneous recordings of electrocardiogram (ECG) and femoral blood pressure (BP), together with contractile force on right (RA) and left (LA) atria, right ventricular conus (RVC) and sinus (RVS), and left ventricular anterior (LVA) and lateral (LVL) surfaces are shown.
common occurrence of mixtures of both sympathetic and parasympathetic fibers within a common nerve sheath was thoroughly confirmed. Such admixtures certainly contribute to the complex combinations of inhibitory and excitatory responses observed, and their participation in the postvagal tachycardias and "rebound" phenomena following cessation of stimulation requires further study.

Figure 2 illustrates the responses to stimulation of the predominantly cardioinhibitory nerves on the left side. All of the nerves designated below A were stimulated without visible response. It is known that many of these contain afferent fibers that have important reflex influences upon cardiovascular function (12), but since the cervical vagosympathetic trunk was sectioned in the present experiments, these effects were eliminated. Nerve A (Fig. 2A) branched off the vagal trunk at a level immediately rostral to the left superior pulmonary vein. Its excitation elicited a slight but distinct suppression in atrial contractile force (LA 25%, RA 10%), without significant alteration in heart rate or conduction pattern as measured from the ECG. Nerve B branched from the vagus only 2-3 mm rostral (to A) and elicited approximately comparable changes in force of LA but considerably more suppression in RA. There was also a minimal decrease in sinus rate (from 150 to 135/min), but more noticeably, a clear 2:1 block in transmission of the excitation potential to the ventricles. Nerve C was located approximately 1 mm rostral to B and its stimulation elicited still greater suppression in atrial contractile force, this time somewhat greater in RA (60%) than in LA (50%). Atrial rate decreased from 155 to 120/min while the ventricular rate was stable at 60/min. Thus, the individual fiber components of these branches were distributed differentially from this small segment of the distal vagus to innervate left atrial contractile muscles primarily (panel A) to both atrial muscles plus the AV node (panel B) and to SA node, atrial muscles, and AV node (panel C).

Panel D illustrates the functional responses to the simultaneous stimulation of both small nerves B and C branching off the main vagal trunk with summation of results shown in panels B and C. Greater inhibition in both rate and contractile force characterized the combined stimulation. Nerve F was a tiny branch from the recurrent laryngeal nerve which elicited profound inhibitory influences on rate of discharge by the SA node, conduction through the AV node, and contractility of both atrial and ventricular muscles. ECG tracings describe initial prolongation in P-R intervals with block as well as depression in amplitude of the P waves. Thus, these records depict the general, overall influence most commonly attributed to vagal cardiac nerves, and it is important to note that such influences can be exerted by very small twigs branching off the primary vagus and its branches. The two test segments from right ventricular musculature show distinct depression in contractile force in panels D and E. Atrial fibrillation occurred during stimulation of nerve F, a small branch directly off the recurrent nerve, and followed complete cardiac standstill with a few atrial and a single ventricular escape beats. The fibrillation was accompanied by severe ventricular dysrhythmia with spontaneous return to normal rhythmicity simultaneously with atrial reversion. Panel G was recorded during direct stimulation of the re
current laryngeal nerve at the level of the aortic arch, and while it did not induce complete atrial standstill, AV block was complete. This relatively large nerve exerted lesser direct influence upon the sinoauricular (SA) node than did many smaller nerves. In general, however, the laryngeal nerve exerts very potent inhibition, particularly upon ventricular segments (note RVC and RVS segment inhibition). Panel H represents responses to stimulation of a small fiber branching from the primary vagal trunk approximately 1 cm rostral to the recurrent nerve, and the contrasting influences are striking. Only very minor inhibition was exerted upon right atrial contractile force with no other visible effect. While stimulation of the thoracic vagus itself resulted in massive cardiac inhibition, no other individual vagal branch above the recurrent nerve exerted inhibitory influences in this animal.

Right thoracic vagus. Figure 3 presents data accumulated during electrical stimulation of the small nerves branching from the right thoracic vagus depicted in Fig. 1, again after transection of the right cervical vagosympathetic trunk. Panel A shows responses to stimulation of a very small nerve taking exit immediately rostral to the azygous vein. Evidence for efferent cardiac nerves below this anatomical level was not found. This nerve induced rather profound suppression in RA contractile force (88%) with concurrent LA depression of approximately 60%, without slowing in atrial rate. Presumably, therefore, this nerve did not impinge upon the discharge rate of the SA node. It did, however, induce AV nodal block. Note also the progressive diminution in atrial contractile force, more rapidly in RA than LA. It is of particular interest that this small nerve from the right side exerted such specific and differential influences upon atrial tissues.

Panels B and C offer interesting contrasts in localization of inhibitory effects. These nerves were closely adjacent and took exit from the trunk at the level of the superior vena caval-right atrial junction. Panel B shows complete heart block without slowing in atrial rate while panel C shows only partial block but moderate slowing in SA nodal firing rate. Here again, depression in RA was considerably greater (75%) than in LA (30%) in panel C, but these differences were distinctly less in panel D. Another feature of panel D is the presence of the SA nodal slowing without evidence for AV nodal blockade.

Total AV nodal blockade characterized the stimulation of nerve E with marked SA nodal inhibition and atrial escape beats. Postvagal tachycardia and "rebound" in contractile force developed upon cessation of stimulation, but the rebound was largely confined to the atrial tracts, more in the right than in the left. Panel F reveals complete cardiac standstill upon stimulation of a small nerve leaving the thoracic vagus at the level of the subclavian vein, with onset of atrial fibrillation promptly upon cessation of stimulation and a single atrial escape beat. The fibrillation persisted for several minutes and required administration of lidocaine (0.5-1.0 mg/kg into root of the aorta) for its reversion. It is of great interest and importance that this was a consistent experience in many animals of this series. That is, electrical excitation of a small nerve at this anatomical level regularly induced atrial fibrillation in a majority of animals. While the experience has not been universal, it has occasioned its identification as the "atrial fibrillation nerve." Such nerves were often localized in close association with the recurrent nerve on both right and left sides.

Panels G, H, and I all show different degrees of inhibition upon the several myocardial test segments and upon discharge rates and conduction properties of nodal tissues. Panel G shows complete cardiac standstill with two atrial escape beats but relatively little suppression in left atrial contractile force. Panel H shows SA nodal slowing without AV blockade but marked suppression in right atrial force. Again, right atrial rebound together with postvagal stimulation tachycardia was a prominent accompaniment of this experiment. Panel I reveals complete standstill and AV nodal block with very gradual recovery in contractile force and heart rate, but some rebound in arterial blood pressure and left ventricular contractile force.

Figure 4 illustrates the results of electrical stimulation of four small branches of the right thoracic vagus before (A through D) and after (A' through D') administration of atropine. Stimulation of a small nerve branching off the vagus at the level of the azygous vein (with the cervical vagus transected) elicited marked sinus bradycardia with concurrent suppression in both atrial and ventricular contractile force (panel A). Identical stimulation of the same nerve (panel A') after atropine elicited no measurable change in either rate or force of contraction, and, of course, no alteration in blood pressure. Excitation of another small branch from the thoracic vagus just caudal to the subclavian vein (panel B') induced distinct slowing in heart rate, although this negative chronotropic influence progressively waned during the latter part of the stimulation period. There was simultaneous inhibition in contractile force in some areas but not in all. Immediately following cessation of stimulation, the recovery period was marked by clear post-stimulation tachycardia and the development of pulsus alternans in some of the test segments. After atropine (panel B'), stimulation elicited only a positive chronotropic effect (150-210/min). Panel C reveals a complex response to electrical stimulation of a nerve branching from the vagus at the level of the subclavian vein, being characterized by post positive chronotropic and inotropic changes in atrial tissues but not atrial or ventricular tissues. Thus, while atrial rates increased from 150 to 210, ventricular rates decreased to 30/min. Such results are best interpreted to indicate the presence of sympathetic fibers in this nerve coursing to the SA node and atrial musculature, together with parasympathetic inhibitory fibers passing to the AV node as well as to ventricular contractile tissues. These conclusions are substantiated by the appearance of essentially sympathetic results following atropinization (panel C'). Finally, the recurrent cardiac nerve was stimulated (panel D') with consequent cardiac slowing and inhibition in contractile force. An interesting feature of this response was the differential escape rate, that of the ventricles being considerably faster than that of atrial segments. Here again, stimulation after administration of atropine (panel D') resulted in fundamentally sympathetic responses with augmentation in both rate and contractile force. The latter characterized both atria but only the right ventricular segments.

Figure 5 shows responses to stimulation of small nerves...
LOCALIZED MYOCARDIAL RESPONSES TO VAGAL STIMULATION

CONTROL

ATROPINE

A

B

C

D

A'

B'

C'

D'

EKG

RA

RVC

RVS

LA

LVA

LVL

BP

FIG. 4. Responses to electrical stimulation of 4 small branches of right thoracic vagus before (A through D) and after (A' through D') atropine (0.4 mg/kg). Nerve A was at a level immediately rostral to superior pulmonary vein, nerve B was located just caudal to subclavian vein, nerve C was precisely at level of subclavian vein, and nerve D was recurrent cardiac nerve. Control heart rates remained essentially unchanged at 130/min before and after administration of atropine.

branching from the right thoracic vagus before (panels A through D) and after (panels A' through D') propranolol. Panel A was produced during electrical excitation of a small nerve at the level of the subclavian vein and reveals marked suppression in right contractile force with distinctly less influence upon the left atrium. Complete AV block lead to ventricular asystole and accompanying progressive fall in arterial blood pressure. An ongoing atrial rate of 90/min bespeaks a lesser inhibitory influence at the SA node. Stimulation of the same nerve after propranolol (panel A') elicited essentially similar results except for the fact that the changes were superimposed upon a much slower basal heart rate. A small nerve approximately 5 mm rostral to the subclavian vein gave rise to the responses shown in panels B and B'. In the control state, this nerve exercised relatively little negative chronotropic changes accompanied by modest depression in atrial contractile force. There was no evidence of AV nodal blockade except during the first 2 or 3 s of stimulation when 2:1 block occurred. The effect was very short lived. After propranolol (B') atrial suppression was the only response to nerve stimulation. In panel C a small nerve 1 cm caudal to the caudal cervical ganglion was stimulated to produce inhibition in contractile force in both atria, but again distinctly greater in RA than in LA. Modest atrial slowing occurred concurrently with 2:1 AV nodal blockade. Recovery was marked by clear postvagal stimulation tachycardia together with ventricular (but not atrial) pulsus alternans. The latter was presumably associated with the tachycardia, and it progressively disappeared as heart rate returned to control levels. Following propranolol (panel C'), complete AV block developed with relatively little slowing in atrial rates. Arterial pressure fell markedly as a result of ventricular asystole. Finally, panel D was recorded during stimulation of the right thoracic vagosympathetic trunk at the level of the caudal cervical ganglion and was accompanied by cardiac standstill. Only two atrial escape beats appeared and these were followed by atrial fibrillation which persisted for approximately 15 s after cessation of stimulation. The fibrillation was accompanied by severe ventricular dysrhythmia, of course, and the latter resulted in low arterial blood pressure. Fibrillation did not occur during stimulation of the same nerve following propranolol (panel D'), although ventricular standstill was again complete.

DISCUSSION

It is clear from the present structural-functional studies that sympathetic and parasympathetic fibers are intermingled in many and probably a majority of the thoracic cardiac nerves, and that perhaps the only level at which reasonable certainty may be had for stimulation of purely vagal fibers to the heart may be achieved at the caudal pole of the nodose ganglion. Certainly many small branches leaving the primary vagal trunk below the caudal cervical.
ganglion are primarily parasympathetic, but a great deal of variation in levels of outflow and in content (sympathetic, parasympathetic, afferent nerves) exists. As pointed out by Keng (10), the left thoracic vagus lies in a ventral plane and is distributed to both dorsal and ventral surfaces of the heart. The right vagus is destined for the deep cardiac plexus which is connected with the dorsal surfaces of the heart.

In his classical treatise, Nonidez (15) found only two distinct “cardiovascular nerves,” one on each side. Depending entirely upon anatomical identification, he found only “small and inconstant” vagal contributions to the heart below the level of the caudal cervical ganglia, although he acknowledged that branches from the recurrent laryngeal nerves may contain vagal fibers destined for the heart. In clear contrast to the earlier view of Nonidez, the present study reveals a myriad of small branches from the thoracic vagus to the heart, some carrying predominantly inhibitory fibers, others carrying excitatory fibers, and most representing varying mixtures of both. Afferent fibers, probably transmitting impulses from all cardiac chambers, as well as from the large central vessels and the lungs, greatly complicate patterns of traffic within these individual nerves. As has been shown to characterize the small sympathetic cardiac nerves (18), distribution of the parasympathetic nerves is also highly localized to discrete portions of the myocardium. Fibers within a given branch may innervate atrial contractile muscle but completely bypass the SA and AV nodes (Fig. 2A). Closely adjacent nerves may contain fibers which inhibit atrial contractile force and block AV conduction without obviously influencing SA nodal firing rates (Figs. 2B, 3A, etc.). Inhibition in contractile force and/or heart rate may be graded by carefully selecting individual nerves which supply contractile or pacemaker cells. The central nervous system may accomplish this, of course, by selectively activating fibers distributed to any conceivable combination of such cells.

It is important to recognize that such potential for selectivity exists and contrary to conventional views, vagal control is not necessarily exercised through diffuse generalized discharges to all portions of the heart in tonic fashion or during reflex bursts of impulses by way of the efferent vagus. Such nonhomogeneous distribution of vagal endings is confirmed by studies of acetylcholine esterase distribution in atrial myocardium (3, 4, 9). Cholinergic stimulation favors development of nonuniform excitability and recovery, creating situations ideal for electrical instability (1, 14).

The degree of intermingling of sympathetic and parasympathetic fibers within a single small branch of the thoracic vagus is illustrated in Fig. 4C. This tiny nerve was anatomically isolated by lifting it upon the stimulating electrodes to induce the unusual result of accelerated atrial rate and augmented contractile force concurrently with strong inhibition of conduction through the AV node and

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**Fig. 5.** Responses to stimulation of 4 small branches from right thoracic vagus before (A through D) and after (A' through D') propranolol (1 mg/kg). Note A was located at level of subclavian vein, nerve B was approximately 5 mm rostral to subclavian vein, nerve C was 1 cm caudal to caudal cervical ganglion, and D represented a location upon right thoracic vagosympathetic trunk at caudal cervical ganglion. Control heart rate decreased from approximately 160/min before to 115/min after administration of propranolol.
inhibition in ventricular contractile force. Similar stimulation after atropine confirmed the presence of sympathetic fibers supplying the atria and parasympathetic fibers to the ventricles. Essentially comparable data are offered in Fig. 5 in which sympathetic responses were blocked by propranolol, thus unmasking largely inhibitory responses to excitation of parasympathetic fibers within selected cardiac nerves.

A prominent feature of these experiments has been the elicitation of atrial fibrillation by means of direct electrical excitation of individual small branches from the thoracic vagus. While atrial fibrillation is known to occasionally result from direct stimulation of the cervical vagus, it is, to our knowledge, not generally known to be induced by stimulation of small individual branches from the thoracic vagus. In fact, fibrillation rarely if ever occurred during stimulation of small branches in the region of the pulmonary veins or the more distal portions of the vagosympathetic trunk. As one approached the region of the recurrent laryngeal nerves, higher on the right than on the left, atrial fibrillation could be expected. Stimulation of nerves in this area was found frequently to induce fibrillation so that the investigators became inclined to look for the atrial fibrillation nerve. Repeated stimulation of such a nerve commonly (but not invariably) induced fibrillation, while immediately adjacent nerves often failed to do so. Unfortunately (or fortunately, depending upon the point of view), the occurrence of this phenomena was not sufficiently consistent to warrant such designation. Atrial fibrillation was, of course, invariably accompanied by grossly dysrhythmic ventricular excitation and contraction, and this phenomenon gives rise to extremely important clinical implications.

The recurrent cardiac nerves are perhaps the largest single nerves carrying parasympathetic fibers to the heart and represent the most important inhibitory inputs to all portions of it. While the right recurrent nerve contains rich intermingling of sympathetic and parasympathetic fibers, the left recurrent consists primarily of parasympathetic. On the right, profuse vagal branching exists between the levels of the caudal cervical ganglion and the superior pulmonary vein. Mizeres (11-13) divided these nerves into cranial- and caudalvagal divisions, and this oversimplification may have obscured the remarkable diversity in distribution of nerves to the heart. Only rarely does one find individual cardiac branches from the vagus above the caudal cervical ganglion. Similarly, on the left, the major inhibitory influence upon the heart is exercised by fibers within, or close to, the recurrent nerve. This large nerve sends masses of small branches into the cardiac plexuses, as well as individual nerves to the left atrium and closely adjacent tissues. A few efferent twigs may be demonstrated from the thoracic vagus immediately distal to the point of exit of the recurrent, but these are variable in distribution and inconsistent from animal to animal. Branches from the thoracic vagus above the left recurrent are also sparse, although parasympathetic fibers are generally found interspersed within the parallel sympathetic (ventrolateral, ventromedial, dorsal, and innominate) nerves.

Cardiac responses to direct electrical stimulation of the vagosympathetic trunk have thus been shown to vary remarkably, depending upon precisely where the electrodes are applied. From the cervical level, a major parasympathetic response may be anticipated because here are concentrated the descending vagal inhibitory fibers to the heart. However, it is well known (19) that varying numbers of sympathetic fibers in this region may greatly modify the response. Stimulation of the vagosympathetic trunk within the thorax may elicit predominantly sympathetic, or parasympathetic, or combinations which may entirely suppress atrial contractility and markedly slow the heart, but simultaneously augment ventricular contractility and elevate systemic arterial blood pressure (16, 17). Stimulation of small branches in the region of the superior pulmonary veins may elicit bradycardia and decreased contractile force provided both vagi are intact, but these responses may be obliterated upon ipsilateral vagotomy, thus revealing the participation of afferents mediating cardiocardiac (and/or pulmonary afferents) reflexes.

Vagal excitation is known to elicit sinus tachycardia and rebound in contractile force immediately upon cessation of stimulation. Figure 3 illustrates these phenomena (panels E and H), with rebound in force being most prominent in the atrial chambers. In fact, the rapid recovery and exaggeration in contractile force is rarely observed in all of the test segments. Elevation in systemic arterial blood pressure also sometimes accompanies the rebound phenomena. These observations are pointed out here only because of their prominence in some panels of the accompanying figures, but attention is specifically drawn to the inconsistency of these occurrences, and in the fact that they do not characterize responses to all small nerve stimulations. In fact, nerves branching off the vagus within fractions of a millimeter from one another may or may not induce similar postvagal stimulation recovery events.

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