

Mechanical Stimuli Exciting Type A Atrial Vagal Receptors in the Cat

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SUMMARY The activity of type A right atrial vagal receptors was recorded from the right cervical vagus in cats anesthetized with sodium pentobarbital, immobilized with gallamine, and with their chests open. Nerve impulses initiated by receptor activation were recorded simultaneously with instantaneous right atrial pressure and dimensional changes under various hemodynamic conditions. Atrial volume changes induced by infusion of saline, bleeding, and occlusion of the inferior vena cava did not alter consistently the systolic activity of the receptors. Electrical stimulation of the right stellate ganglion significantly increased the frequency of discharge during systole, whereas electrical stimulation of the left thoracic vagus significantly reduced the frequency of discharge. These inotropic interventions produced similar effects when the heart was paced at a fixed rate. Pacing the right atrial appendage increased the systolic discharge of

the receptors only when at high rates the atrium contracted against closed atrioventricular valves. To investigate the influence of tonic efferent sympathetic activity on spontaneous receptor discharge, three receptors were studied before and after bilateral surgical stellectomy, and in cats with their chest closed three receptors were studied before and after infusion of propranolol. Both of these interventions markedly reduced the systolic activity. In addition to having effects on systolic activity, injection of saline, vagal stimulation, and sympathetic "denervation" always activated the receptors during filling. Our results indicate that: (1) the systolic discharge of type A receptors is a function of the active tension developed by atrial muscle during contraction; and (2) the pattern of discharge of the receptors during the atrial cycle depends on both the degree of atrial distention and the state and extent of contraction.

THE NATURAL stimulus for atrial vagal receptors which are spontaneously active during atrial systole (type A and intermediate type receptors) is unknown.¹ It has been suggested that the receptors may respond to atrial systolic pressure,² active wall tension,³ or heart rate.⁴ In our present experiments we recorded nerve impulses caused by activity of right atrial receptors simultaneously with the instantaneous pressure and dimensional changes of the right atrium.^{5, 6} The results obtained demonstrate that the systolic discharge of type A and intermediate type receptors is a function of the active tension developed by atrial muscle during atrial systole and that the pattern of discharge during the cardiac cycle depends on atrial distention and the contractile state of the atrial muscle.

Methods

Our results were obtained from 29 experiments on cats (2.5–4.0 kg) anesthetized with sodium pentobarbital (35 mg/kg, ip). The trachea was cannulated and each animal was artificially ventilated after intravenous injection of a paralyzing dose (3 mg/kg) of gallamine triethiodide (Sincurarina, Farmitalia). The guidelines of the American Physiological Society regarding experimentation on anesthetized, paralyzed animals were observed. The respirator was adjusted to maintain arterial PO_2 , PCO_2 , and pH

(measured on an Astrup model RM 1304 blood acid-base analyzer) within physiological limits. Polyethylene catheters were inserted into a femoral artery, into the right atrium through the external jugular vein, and into a femoral vein. Right atrial and femoral arterial pressures were measured with Statham P23De strain gauges. The frequency response of the catheter-manometer systems was flat ($\pm 5\%$) to 30 Hz. In 26 cats the chest was opened bilaterally from the 2nd to the 5th intercostal spaces and the sternum was removed. The left thoracic vagus and the right stellate ganglion were dissected from the surrounding tissues and decentralized.

In 16 experiments an incision was made in the pericardium to expose the right atrium. Two miniature piezoelectric crystals were sutured to the atrial epicardium near its junction with the superior vena cava.⁶ The pericardium was closed after the implantation of the crystals. A sonomicrometer measured the transit time of ultrasound between the two piezoelectric crystals at a sampling rate of 5,000 times per second.⁷ The instantaneous changes in the distance between the two crystals have been defined as right atrial dimensional changes (RAD) or, alternatively, as right atrial length changes. The same measurement was defined as "right atrial diameter" in two previous publications.^{5, 6}

Activity of afferent fibers was recorded, by a method previously described,^{6, 8} from filaments isolated from the right cervical vagus under a dissecting microscope. In three experiments the activity of three receptors was recorded before and after removal of both stellate ganglia and section of the left vagus in the thorax.

Right atrial pressure (RAP) and dimension (RAD), femoral arterial pressure (AP), electrocardiogram (ECG) (Grass P511 preamplifier), tracheal pressure, and the impulses in afferent fibers were recorded with a multichannel

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optical recorder (Hewlett-Packard 4578A) and a magnetic tape recorder (HP 3907C). All the variables could also be photographed (Grass C4 camera) from a slave cathode-ray tube arranged in parallel with a Tektronix 565 oscilloscope.

Right atrial volume was changed either by slow (10–50 ml at approximately 0.5 ml/sec) and fast (3 ml at approximately 2 ml/sec) injections of saline at body temperatures, by withdrawal of 25–35 ml of blood, or by occlusion of the inferior vena cava. The right stellate ganglion or the cut peripheral end of the left thoracic vagus was stimulated electrically (Grass S4 stimulator) with rectangular pulses (5–10 V, 3 msec) at frequencies of 10–20 Hz. On occasion isoproterenol (Alupent, Boehringer) (0.2 μ g/kg) and acetylcholine bromide (Pragmolina, Farmitalia) (1–10 μ g/kg) were injected intravenously. In seven experiments heart rate was controlled by stimulating the right atrial appendage through a bipolar electrode connected through an isolation unit to a Tektronix 161 pulse generator. In three additional cats with the thorax intact, activity of vagal fibers was recorded before and after intravenous administration of propranolol (Inderal, ICI 45520) (0.5 mg/kg). Data were collected from cats with a systolic blood pressure above 100 mm Hg.

Analysis of neural activity was restricted to the expiratory phase of the respiratory cycle. We calculated: (1) the number of spikes per burst (n), (2) the duration of the burst (db), (3) the mean frequency of discharge during the burst (reciprocal of the mean interspike interval: $n - 1/db$), (4) the peak frequency in the burst (pf), and (5) the average discharge rate (number of spikes/burst \times heart rate)/60 ($n \cdot \text{HR}/60$).⁴ Each interspike interval in the burst was measured by a digital neural spike analyzer with an accuracy of 1%. The analog output level of the neural spike analyzer was proportional to the pulse interval and was displayed with a delay of one interspike interval (see Figs. 1, 2, and 5).

In six experiments we studied the relationships between impulse frequency and each of the following variables: atrial dimension (or length) at the beginning of atrial systole (initial dimension, ID); atrial dimension at the end of atrial systole (end-systolic dimension, ESD); amount of systolic shortening (ΔD , that is, the difference between the initial and end-systolic dimension); mean rate of shortening ($\Delta D/\Delta t$, that is, the relation between the amount of systolic shortening and its duration); pressure at the foot of the a wave (initial pressure, IP); peak atrial systolic pressure (P_{max}); amplitude of the a wave (ΔP); and slope of the upstroke of the a wave ($\Delta P/\Delta t$) (see Fig. 4). To assess the correlations between impulse frequency and each of these variables, a logarithmic transformation of the latter was made, regression lines were fitted to the data, correlation coefficients were calculated, and their significance was tested.

To ascertain that all the receptors studied were located in the right atrium we used the maneuvers suggested by Paintal.⁹ At the end of each experiment the receptor was localized accurately by probing the internal and external surfaces of the right atrium in the opened heart.¹⁰ Fifteen receptors were located at the junction of the superior vena cava with the right atrium, eleven at the junction of the inferior vena cava with the right atrium, and three at the atrioventricular junction.

Results

We studied 23 atrial vagal receptors that were spontaneously active during atrial systole (type A).¹¹ The average peak frequency of discharge during atrial systole was 99.5 ± 9.8 impulses/sec (mean \pm SE), the average mean frequency of discharge was 77.0 ± 6.3 impulses/sec, and the number of spikes was 4.3 ± 0.4 . Nine receptors also displayed a spontaneous low frequency discharge during atrial filling (intermediate type receptors:¹² the number of spikes during filling was 3.22 ± 0.8 and the mean frequency of discharge was 32.8 ± 1.6 impulses/sec. No relationships were found between spontaneous impulse activity of the receptors and their location.

ATRIAL VOLUME CHANGES

Increases or decreases in atrial volume slightly enhanced or diminished, respectively, the systolic discharge of three atrial receptors, whereas the activity of five other receptors was unaffected by changes in atrial volume of similar magnitude. As shown in Table 1, the average effect of atrial volume changes on systolic impulse activity was not significant.

Furthermore, with the exception of the receptor discharging spontaneously at the highest rate (220–240 impulses/sec) infusion of saline always elicited receptor activity during atrial filling (Fig. 1). On the other hand, bleeding or inferior vena caval occlusion diminished or abolished the diastolic discharge of the intermediate type receptors.

INOTROPIC INTERVENTIONS

Electrical stimulation of the right stellate ganglion and injection of isoproterenol enhanced the systolic activity of the 14 receptors studied (Fig. 2A and Table 2). This enhancement was inversely related to the rate of spontaneous activity (Fig. 3A). As is shown in Figures 2B and 3A, negative inotropic interventions (acetylcholine administration and stimulation of the left thoracic vagus) always reduced the systolic activity of the nine receptors studied (Table 2). In addition, inotropic interventions modified atrial dimensions with respect to control conditions (Table 2).^{5, 6}

To compare the effects of sympathetic and vagal stimulation at similar atrial dimensions, in six experiments we performed these interventions during simultaneous alterations of venous return. In the example shown in Figure 4 the peak frequency of systolic discharge, and the mean frequency of discharge as well, were higher during sympathetic stimulation than during control conditions at the same initial dimension (initial length) and pressure (Fig. 4A and E), for the same amount of systolic shortening (Fig. 4C) and rate of shortening (Fig. 4D). At any given initial length the amplitude and the slope of the a wave always were higher during sympathetic stimulation than during control conditions or during vagal stimulation. Moreover, at any given atrial length the discharge of this receptor was curvilinearly related with peak, amplitude, and mean rate of change of the a wave (Fig. 4F, G, and H).

To assess quantitatively the relationships between impulse frequency and pressure or length, we calculated correlation coefficients (r) and tested their significance (see Methods).

TABLE 1 Effects of Atrial Volume Changes

	No of spikes/burst (systole)	Mean frequency of discharge (impulses/sec)	Peak frequency of discharge (impulses/sec)	Average discharge rate (impulses/sec)	No. of spikes/burst (filling)	Initial dimension (mm)	Pressure foot of a wave (cm H ₂ O)	Amplitude of a wave (cm H ₂ O)	Slope of a wave (cm H ₂ O/sec)
Control (n = 8)	5.2	85.7	107.2	12.2	1.4	4.6	2.5	1.6	36.2
	± 0.3	± 6.5	± 13.9	± 1.1	± 0.5	± 0.3	± 0.3	± 0.3	± 4.1
Volume increases (n = 8)	5.5	88.0	110.7	12.9	5.2	5.1	4.1	1.9	40.6
	± 0.6*	± 11.1*	± 15.7*	± 1.1*	± 0.9†	± 0.3‡	± 0.2‡	± 0.2‡	± 5.2‡
Volume decreases (n = 8)	4.9	79.9	102.9	11.5	0†	4.2	1.5	1.0	25.0
	± 0.5*	± 7.9*	± 12.8*	± 1.2*		± 0.2‡	± 0.2‡	± 0.2‡	± 3.0‡

n = number of receptors studied; initial dimension = the diameter at which atrial active shortening begins.

All values are mean ± SE. Significance was determined by paired *t*-test.

* Not statistically significant.

† *P* < 0.01.

‡ *P* < 0.05.

The relations of impulse frequency to amplitude and slope of the a wave were statistically significant for all six receptors (r , 0.994 to 0.807, P always < 0.01). The relations to peak systolic pressure (r from 0.948 to 0.369) and to initial pressure (r from 0.809 to -0.486) were less exact and were significant at the 1% level for only two receptors, data for one of which are illustrated in Figure 4F. This lack of a consistent correlation with peak systolic pressure also can be deduced from the data for the nine receptors summarized in Table 2: vagal stimulation and acetylcholine administration markedly reduced impulse frequency, whereas peak systolic pressure (which can be calculated by adding the amplitude of the a wave to initial pressure) was slightly increased with respect to control conditions. The relationship between impulse frequency and amount of shortening (r from 0.880 to 0.139) or rate of shortening (r from 0.689 to -0.067) were even more variable (P < 0.01 in only one case). Finally, there was no significant relationship between impulse frequency and initial dimension (r from 0.556 to -0.206) or

end-systolic dimension (r from 0.523 to -0.446) for any of the six receptors.

Inotropic interventions affected diastolic impulse activity of all the receptors studied. Sympathetic interventions reduced or abolished the diastolic burst of intermediate type receptors, while vagal stimulation elicited or increased the impulse activity during atrial filling (Fig. 2B).

The effects of inotropic interventions on the systolic and diastolic activity of the receptors were independent of changes in heart rate because they were reproduced in paced hearts (Fig. 2).

HEART RATE CHANGES AND ISOVOLUMIC ATRIAL CONTRACTION

Pacing the heart from a spontaneous rate below 150 beats/min to 180–200 beats/min only slightly modified the systolic activity of seven receptors studied (Figs. 3B and 5a and b). As already reported, although preload for active shortening and amount of shortening increased, right atrial dimensions (both end-diastolic and end-systolic) were unaltered by atrial pacing.⁵ At heart rate above 200 beats/min the firing rate of four receptors increased (Fig. 3B). In the example shown in Figure 5, at a heart rate of 233 beats/min (Fig. 5c), atrial contraction started before the opening of the atrioventricular (AV) valves and the increased receptor discharge preceded atrial shortening and was coincident with a marked rise in atrial systolic pressure. By further increasing the pacing rate we obtained the record shown in Figure 5d. The receptor discharge was maximal when the atrium was contracting "isovolumically" against the closed AV valves. However, when the atrium was able to empty into the ventricle, the activity was greatly reduced (two atrial beats marked by asterisks in Fig. 5d). During isovolumic atrial contractions the peak firing rate in the burst occurred before the peak pressure was reached.

Similarly, the firing rate of three other receptors increased during atrial pacing only when the atrium started to contract before the AV valves opened. The increase in impulse frequency was coincident with an abrupt rise in atrial systolic pressure.

It should be mentioned, moreover, that the diastolic discharge of two intermediate type receptors gradually was decreased by increasing the rate of stimulation, although

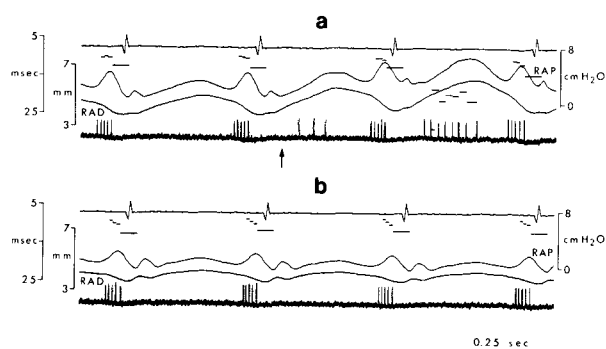


FIGURE 1 Acute changes in atrial volume. Tracings in panels a and b represent (from top to bottom): ECG, instantaneous interspike interval (horizontal lines above pressure recording), right atrial pressure (RAP), right atrial dimensional changes (RAD), and recording of nerve impulses of a type A right atrial receptor. The analog output level of the digital neural spike analyzer is proportional to the pulse interval and is displayed with a delay of one interval. The reading of the last interspike interval of the burst persists for the preset range of reading (50 msec in this figure and others). Intervals longer than 25 msec (as for the diastolic discharge after the arrow in a) are off scale. a: Injection of 3 ml of warm saline starting at the arrow. b: After occlusion of the inferior vena cava.

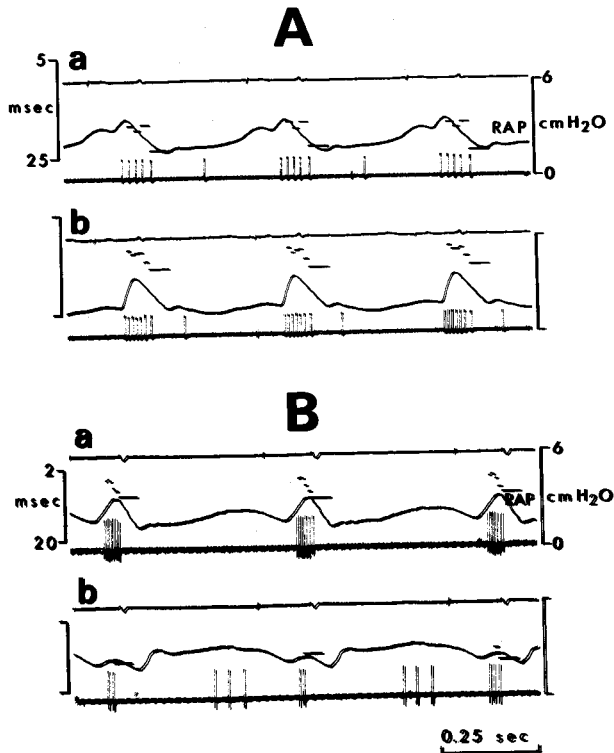


FIGURE 2 Effects of right stellate ganglion stimulation (A) and acetylcholine administration (B) on two different receptors at fixed heart rate. Tracings represent (from top to bottom): ECG, instantaneous interspike interval, right atrial pressure (RAP), and recording of nerve activity. Aa: Control discharge at paced heart rate of 146 beats/min. Ab: Effect of electrical stimulation of the right stellate ganglion. Ba: Control discharge at paced heart rate of 120 beats/min. Bb: Effects of acetylcholine administration.

atrial diastolic pressure and atrial dimensions were modified only slightly (Fig. 5).

REMOVAL OF THE STELLATE GANGLIA AND ADMINISTRATION OF PROPRANOLOL

To analyze the role of the tonic efferent sympathetic activity on receptor discharge we studied three receptors before and after removal of the stellate ganglia and, in cats with a closed thorax, we studied three other receptors before

and after administration of propranolol. As shown in Figure 6 and Table 3, bilateral stellectomy (and propranolol administration) decreased systolic activity and increased diastolic activity with respect to the control (Fig. 6a and b). After injection of isoproterenol (Fig. 6c), receptor discharge was similar to that which had occurred before surgical stellectomy. After injection of acetylcholine (Fig. 6d) the receptor was active only during atrial filling, and the peak frequency of discharge corresponded to the end-diastolic length.

Discussion

Type A atrial vagal receptors are slowly adapting stretch receptors¹³ that are spontaneously active and show a prominent burst of impulses during atrial systole.^{11, 12} Intermediate type receptors, in addition to the systolic burst, display a spontaneous low frequency discharge during atrial filling.¹² Both types of receptors are considered together in the following discussion, because their responses to the alteration in atrial dynamics during systole were similar qualitatively. Moreover, since it has been demonstrated already that type A receptors¹³ as well as type B^{6, 13} are sensitive to static and dynamic components of passive muscle stretch, in the present study the relationships between receptor activity, pressure, and change in dimension during atrial filling were not evaluated quantitatively.

NATURE OF THE EXCITING STIMULUS

To determine whether force or displacement was the stimulus exciting the receptors during atrial systole, we recorded activity together with the instantaneous pressure and dimensional changes of the right atrium.⁶ The response of the receptors was evaluated mainly in terms of frequency of discharge in the burst¹⁴ rather than number of spikes per burst, since the latter has been shown to be dependent on the duration of the stimulus as well as on its intensity.^{6, 13}

The systolic activity of the receptors always was modified by interventions which altered the strength of atrial contraction and, under different inotropic conditions, it always was found to correlate significantly with the amplitude and slope of the a wave. Conversely, impulse frequency was affected inconsistently by changes in atrial volume and heart rate. Moreover, type A receptors are active during isovolumic

TABLE 2 Effects of Inotropic Interventions

	No. of spikes/burst (systole)	Mean frequency of discharge (impulses/sec)	Peak frequency of discharge (impulses/sec)	Average discharge rate (impulses/sec)	No. of spikes/burst (filling)	Initial dimension (mm)	Pressure foot of a wave (cm H ₂ O)	Amplitude of a wave (cm H ₂ O)	Slope of a wave (cm H ₂ O/sec)
Control (n = 16)	3.8	73.6	93.6	9.2	1.17	4.8	2.7	1.4	32.0
	± 0.6	± 10.4	± 14.4	± 0.6	± 0.3	± 0.3	± 0.4	± 0.3	± 5.1
Sympathetic (n = 14)	5.0	101.2	123.5	14.4	0.2	4.5	2.6	2.7	63.2
	± 0.5*	± 6.3*	± 6.4*	± 0.8*	± 0.1*	± 0.2†	± 0.5‡	± 0.4*	± 7.9*
Parasympathetic (n = 9)	1.4	24.4	23.2	3.1	2.8	5.3	3.7	0.6	15.4
	± 0.5*	± 11.0*	± 12.3*	± 0.3*	± 0.5*	± 0.4†	± 0.3*	± 0.2*	± 2.6*

n = number of receptors studied; initial dimension = the diameter at which atrial active shortening begins.

All values are mean ± SE. Significance was determined by paired *t*-test.

* *P* < 0.01.

† *P* < 0.05.

‡ Not statistically significant.

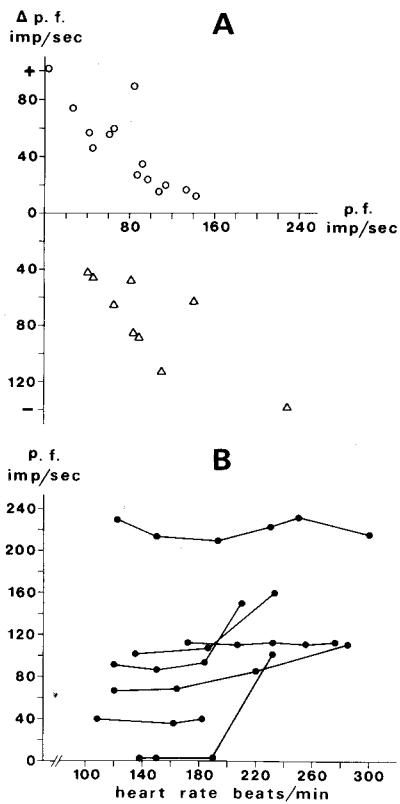


FIGURE 3 A: Changes in peak frequency of discharge produced by positive (O) and negative (Δ) inotropic interventions in relation to peak frequency of discharge during control conditions (abscissa). B: Relationship between peak frequency of discharge in the burst and changes in heart rate. The activity of four receptors was increased by pacing the heart above 200 beats/min.

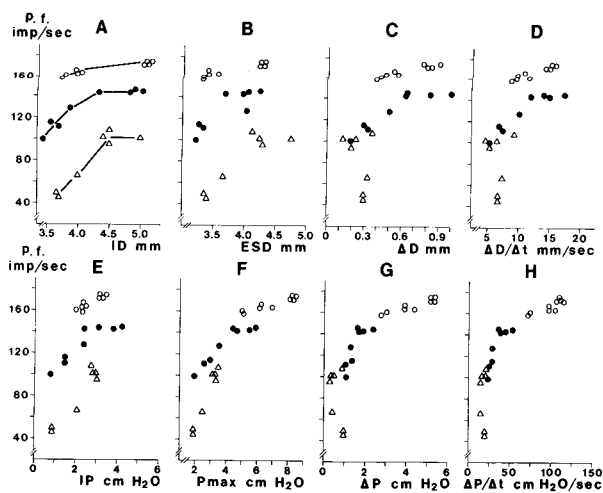


FIGURE 4 Relationship between peak frequency of discharge in the burst, atrial dimension, and pressure for one receptor studied during changes in atrial volume performed under control conditions (●) and sympathetic (○) and vagal (Δ) stimulation. ID = initial dimension; ESD = end-systolic dimension; ΔD = amount of systolic shortening; ΔD/Δt = mean rate of shortening; IP = pressure at the foot of the a wave; P_{max} = peak of the a wave; ΔP = amplitude of the a wave; ΔP/Δt = slope of the upstroke of the a wave.

atrial contractions and during the contractions of a widely opened atrium, although no pressure is generated.¹² We thus may conclude that the systolic activity of these receptors is a function of the active tension developed by atrial muscles during contraction.

It has been suggested previously³ that the receptors are sensitive to the dynamic component of muscle contraction. In fact, at high frequencies of impulse activity, the peak of the firing rate in the burst always occurs before the peak of the a wave is reached.^{13, 15}

Receptor activity was enhanced markedly during atrial pacing at rates above 200 beats per min¹⁶ whenever the atrium contracted against closed AV valves. Since heart rate changes in this range do not affect contractility of atrial muscle,^{17, 18} the increase in afterload for atrial contraction was responsible for the large rise in atrial pressure and for receptor excitation. It has been demonstrated that, below a critical aortic pressure, adequate increases in afterload progressively prolong the period of isovolumic ventricular contraction and thus allow the cardiac muscle to develop full force before external shortening begins.¹⁹ This may explain the excitation of the receptors which occurred during isovolumic atrial contraction.

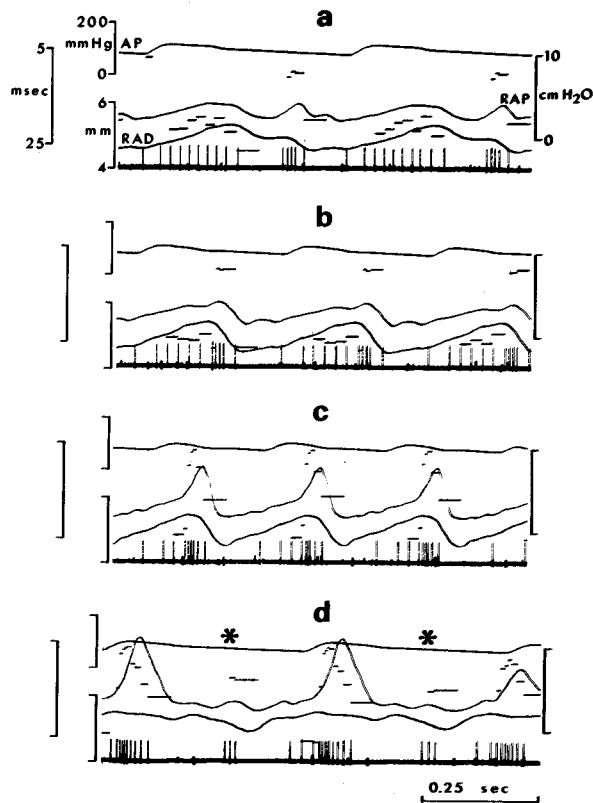


FIGURE 5 Effects of changes in heart rate on the activity of an intermediate type receptor. Tracings (from top to bottom) represent: femoral arterial pressure (AP), instantaneous interspike interval, right atrial pressure (RAP) and recording of nerve activity. a: Control. b: Pacing at 183 beats/min. c: Pacing at 233 beats/min. d: The atrium contracts at 270 beats/min while the ventricles contract at 133 beats/min. Asterisks indicate two atrial contractions during which atrial emptying occurs.

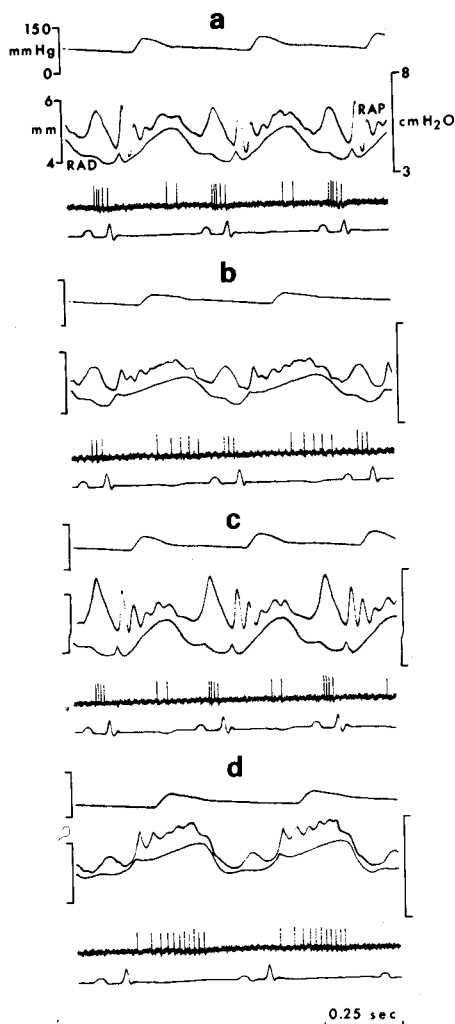


FIGURE 6 Effects of bilateral stellectomy, isoproterenol administration, and acetylcholine administration on the spontaneous activity of one receptor. Tracings (from top to bottom) represent: femoral arterial pressure (AP), right atrial pressure (RAP), right atrial dimension (RAD), recording of nerve impulses, and ECG. a: Control. b: After removal of both stellate ganglia. c: Isoproterenol administration. d: Acetylcholine administration.

The response of a receptor to a given stimulus depends not only on the mechanical driving force but also on the working range of the receptor.¹⁴ Thus, the response to an increase in the strength of atrial contraction produced by sympathetic stimulation was inversely related to the control spontaneous firing rate, while a spontaneous high frequency discharge

could be affected only by a stimulus which decreased the strength of atrial muscle contraction. Arndt et al.⁴ were not able to find any relationships between atrial systolic pressure and receptor activity because in their experiments the receptor discharge was saturated and the effects of negative inotropic interventions were not tested. As pointed out already, it seems doubtful that efferent sympathetic activity directly depolarizes receptor endings.^{20, 21}

DIASTOLIC ACTIVITY AND PATTERN OF DISCHARGE

All the receptors studied displayed bursts of activity during atrial filling when atrial volume was increased either by infusion of saline²² or by vagal stimulation. The diastolic firing rate was diminished or abolished by decreases in atrial volume produced by reduction in venous return or positive inotropic interventions, although the receptors still were active during atrial systole. In contrast, type B receptors never displayed a prominent burst of impulses during atrial systole and displayed only one or two impulses after the peak of the a wave during sympathetic stimulation.⁶ These findings indicate that (1) the discharge pattern of type A receptors is dependent on both atrial distention and the contractile state of atrial muscle; (2) the threshold of type A receptors is higher to passive stretch than to active contraction; (3) only type A receptors may display an intermediate pattern of discharge.

When atrial contractility is depressed and atrial volume is increased, these receptors may be active only during filling; they thus become indistinguishable from type B receptors. It is known that only a few type A atrial vagal receptors have been identified in the dog.¹ In addition, in the dog the peak firing rate of type B atrial receptors occurs at the peak of the v wave.²³ Both of these experimental findings may be explained on the basis of a high vagal and low sympathetic tone which would influence the contractile state of the heart. These observations also may suggest that type A and type B receptors can be clearly distinguished from each other only at high levels of atrial contractility.

The functional properties of the Golgi tendon organs, the "in series" muscle receptors,²⁴ are very similar to those of type A atrial receptors. They may be active during both muscle stretch and contraction,²⁴ their threshold is higher to passive stretch than to active contraction,²⁵ and their discharge during contraction is a function of the active tension developed²⁶ and is higher during isometric than during isotonic contraction.²⁷ Thus, according to Whit-teridge's hypothesis,²⁸ we may define type A receptors as functionally "in series" with atrial muscle and type B receptors, which are unloaded by atrial contraction,⁶ as "in

TABLE 3 Effects of Surgical and Pharmacological Sympathetic "Denervation"

	No. of spikes/ burst (systole)	Mean frequency of discharge (impulses/sec)	Peak frequency of discharge (impulses/sec)	Average discharge rate (impulses/sec)	No. of spikes/ burst (filling)	Heart rate (beats/min)
Control (n = 6)	4.5 ± 0.4	78.0 ± 9.3	109.5 ± 16.1	13.3 ± 0.9	1.5 ± 0.6	178 ± 11
Sympathectomy (n = 6)	2.4 ± 0.3*	50.1 ± 14.2*	58.3 ± 16.2*	6.1 ± 1.0*	4.5 ± 1.6*	147 ± 11*

n = number of receptors studied.

All values are mean ± SE. Significance was determined by paired *t*-test.

* *P* < 0.01.

parallel" atrial receptors. This does not exclude the possibility that type A and B receptors, above their static threshold, may have similar sensitivities to the static and dynamic components of passive stretch.¹⁸

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