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Original Article

Experimental Myocardial Ischemia-Induced Defecatory Reflex in Anesthetized Cats: A Study to Explore Neural Pathways for This Cardiogenic Reflex

Basak Asim Kumar[1*], PhD; Koley Juthika[2], PhD

ABSTRACT

Background: Clinically, heart failure due to cardiac ischemia or myocardial infarction is associated with different reflex responses, including bradycardia, hypotension, and the urge to urinate or defecate. Stimulation of the cardiac nociceptors of the left ventricle initiates cardiogenic reflexes, such as the mass rectal contraction, which may be responsible for defecation urge. We aimed to study the afferent and efferent pathways responsible for this cardiogenic rectal response.

Methods: Experiments were performed on artificially ventilated anesthetized cats of either sex. Myocardial ischemia was induced by occluding the main left anterior descending coronary artery (LAD), and rectal movement was recorded. The effects of LAD occlusion on intact and sectioned left inferior cardiac sympathetic, cardiac vagus, gastric vagus, splanchnic, inferior mesenteric ganglion, and pelvic nerves on the rectal response were studied.

Results: LAD occlusion induced a biphasic rectal response, which was abolished after sectioning the left inferior cardiac sympathetic nerve but not by cardiac vagotomy. The same response was also abolished by pelvic nerve sectioning. Moreover, the relaxation and contractile phase was abolished by nitric oxide and cholinergic inhibitors.

Conclusions: The defecatory urge associated with cardiac ischemia was due to the reflexogenic contraction of the rectal muscle. The afferent and efferent pathways for this reflex were the cardiac sympathetic nerve and the pelvic nerve, respectively. Additionally, nitric oxide and cholinergic pathways were associated with this reflex ***(Iranian Heart Journal 2024; 25(1): 6-18)***

KEYWORDS: Cardiogenic reflex, LAD occlusion-induced rectal movement, Cardiac ischemic pain and GI movement

[1] Department of Allied Health Sciences, Brainware University, Barasat, West Bengal, India.
[2] Department of Physiology, University of Calcutta, India.

***Corresponding Author:** Basak Asim Kumar, PhD; Department of Cardiology, Faculty of Medicine, Ain Shams University, Cairo, Egypt.
Email: asim_bsk@rediffmail.com **Tel:** 7982740481

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Myocardial ischemia-induced myocardial infarction is a common cause of heart failure. [1, 2] Clinically, it has been observed that heart failure due to cardiac ischemia or myocardial infarction is often associated with different reflex responses, such as bradycardia, hypotension, and the urge to urinate or defecate. [3-7] Experimentally, it has been reported that the occlusion of the anterior descending artery (LAD) or the circumflex branch of the left coronary artery initiates the excitation of cardiac sympathetic afferents, both myelinated [8] and unmyelinated, [9] with the impact originating from receptors located within the ischemic myocardium or the coronary arteries. Although it is widely accepted that the sympathetic nerves are essential to the perception of cardiac pain, [10-12] it is not ruled out that the vagal afferents of the anteroposterior wall of the left ventricle are activated during coronary occlusion in dogs. [13] There have also been many attempts to study cardiac pain with endogenous putative nociceptive mediators, including 5HT, [14] adenosine, [15-17] prostaglandin, [18] potassium, [19] histamine, [20] nicotine, and bradykinin. [21, 22] Application of these substances over the inferoposterior wall of the left ventricle results in reflex responses such as hypotension, bradycardia, gastric relaxation, urination, and defecation by exciting either sympathetic or vagal afferents. [3-7] Hence, it appears reasonable to propose that if cardiac pain leads to the activation of cardiac afferent endings by chemicals and if a number of these substances are released during myocardial ischemia, they may act in concert to produce cardiac pain, which in turn causes various gastrointestinal symptoms and other reflexes by stimulating either of these afferent nerve endings through its interconnection with spinal or supraspinal centers.

Accordingly, in the present set of experimental protocols, we sought to study the effects of LAD occlusion on the motility of the large intestine with reference to its neural mechanism in lightly anesthetized cats.

METHODS

Protocols for Animal Preparation

Experiments were carried out on 54 cats of either sex with a body weight of 2 to 3 kg after an overnight fast with water *ad libitum*. The animals were anesthetized with α-chloralose at a dose of 60 mg/kg body weight after initial induction with anesthetic ether. The anesthesia was maintained throughout the experiment with a maintenance dose of α-chloralose (10 mg/kg, iv) as and when required. The femoral artery, the femoral vein, and the trachea were routinely cannulated. The glucose solution (5%) in physiological saline (0.9%) was administered by drip feed into the femoral vein (l mL/min) throughout the experiment to maintain body fluids and pH. Blood pressure was recorded from the femoral artery on a Beckman RM Dyno Graph using a Bell and Howel pressure transducer (Type-4327 -0129). For the monitoring of the body temperature, a thermometer was placed into the anus, and body temperature was maintained at 37 ℃ with a heating pad. ECG was recorded based on the standard protocol on a Beckman Rm Dyno Graph using standard limb lead II.

The Surgical Procedure

The chest was opened by removing thoracic ribs 2 to 6 while keeping the animals under artificial respiration with a Starling Ideal Respiratory Pump. The left stellate ganglion and the left inferior cardiac nerve (LICN) branches of the vagus to the heart were exposed carefully and cleared from the surrounding connective tissues under a dissecting microscope. Vagotomy, stellatectomy, or LICN sectioning was performed on the animals according to the experimental protocol as and when required. The animals were then allowed to rest for a

minimum of 1 hour before experimentation with LAD occlusion.

The vagus nerve was isolated below the diaphragm level. The splanchnic nerves and the inferior mesenteric ganglia were also isolated and sectioned as and when required. Laminectomy was performed at the sacral (L1-S4) level, and the spinal cord was opened following Koley and Mukherjee. [23] The ventral roots of the S2-S4 level were isolated and transected as per requirement. After the transection of each nerve ganglion or ventral root, the animals were rested for at least 1 hour. Afterward, further experimentation was performed.

Myocardial ischemia was induced by occluding the LAD with the help of a fine snare for 3 to 4 minutes, and the cats were allowed to rest for 30 minutes before the procedure was repeated.

Rectal motility in the form of intrarectal pressure was recorded on the INCO polygraph with a distended balloon inserted into the rectum or the descending colon. A flaccid balloon (1.0-1.5 cm of a condom) distended with 8 to 12 mL of warm saline via a polyethylene tube was inserted into the rectum or the descending colon via a small incision in the descending colon and fixed at the incision point. The animals were given pancuronium (1 mg/kg, iv), a skeletal muscle relaxant, to eliminate the participation of the external anal sphincter and other adjoining skeletal muscles in the resting intrarectal pressure. The experiments were repeated on all the cats after the recording of at least 3 producible cardiorectal reflexes.

The entire study was approved by the PhD Committee of the Department of Physiology at Calcutta University. The study considered all the requisite maintenance of proper animal care and handling during the experimentation process.

Drugs Used

Alpha-chloralose (Koch-Light Lab, UK), pancuronium bromide (Pavulon, Infar [India] Ltd, India), lignocaine (Xylocaine, Astra-IDL, India), atropine sulfate (Bengal Immunity, India), d, I-propranolol hydrochloride (ICI, India), and L-arginine analogue NG-nitro-arginine (LNNA) (Sigma, USA) were used. All the drugs were dissolved in physiological saline at a concentration so that not more than 0.5 mL was needed for the drug application.

Statistical Analysis

The results were expressed as the mean ± the standard error of the mean (SEM). The significance was tested using the Student *t* test. Concerning the control cardiorectal reflex, significance tests were performed using the average initial intrarectal pressure (mm Hg) and intrarectal pressure during the reflex relaxation and contractile phases. Percentage changes in the integrated relaxation pressure (IRP) during the reflex relaxation and contractile phases were compared between the control group and each experimental group.

RESULTS

1. The Cardiac Ischemia-Induced Rectal Response: Occlusion of the LAD for 90 to 120 seconds (depending on the condition of the heart) with the help of a snare caused myocardial ischemia as evidenced by the blackening of the ventricular surfaces, fall of blood pressure and heart rate, and alterations in ECG. Hypotension was encountered immediately after LAD occlusion (Fig. 1A- Upper Right Panel). Prominent ST-segment elevation and T-wave inversion were observed in ECG studies (Fig. 1A- Lower Right Panel). At the same time, the occlusion resulted in biphasic changes in rectal motility. These biphasic changes included initial inhibitions or decreases of spontaneous movements with or without

relaxation, followed by large sustained contractions (Fig. 1A- Left Panel).
Out of 268 observations in 28 cases, no relaxation was found. Nevertheless, the inhibition of the spontaneous motility of the rectum, followed by sustained contractions, persisted. In 18 cases, the contractile phase was absent, but the initial relaxation was present. Nine cats were unresponsive to the occlusion. Figure 3 shows that during the relaxation phase, the IRP was reduced to 24.82 + 0.64 mm Hg ($P < 0.001$), and in the contractile phase, it was increased to 46.72 ± 0.82 mm Hg ($P < 0.001$) compared with the normal IRP (30.68 ± 0.18 mm Hg). On the other hand, a similar experimental protocol to LAD occlusion failed to evoke any changes in colonic motility, expressed in terms of intro-colonic pressure (Fig. 1D).

2. LAD Occlusion After the Desensitization of the Ventricular Receptor: Desensitization of the ventricular sensory receptors with the epicardial application of local anesthetics (2% lignocaine for 5-10 min with the help of fine cotton films) was performed. Five minutes after the withdrawal of the lignocaine-soaked cotton film, LAD occlusion following the desensitization of the ventricular receptors failed to evoke biphasic changes in the rectal response (Fig. 1B- Left Panel). Nevertheless, neither reductions in blood pressure (Fig 1B- Right Upper Panel) nor ECG changes (Fig. 1 B- Right Lower Panel) in response to LAD occlusion were altered, indicating that blood pressure reduction and ECG changes were still manifest after LAD occlusion, even after the desensitization of the ventricular receptors with lignocaine.

3. A Rectal Response of Cardiac Origin due to Atrial Distension: Stimulation of the atrial volume receptors (n = 8) by distending the left atrial appendages with 0.5 to 2.0 mL of warm physiological saline through a balloon did not evoke any change (Fig.1 C) in the mean IRP (29.28 ± 1.62 mm Hg) compared with the normal IRP (30.75 + 0.82 mm Hg).

4. The Afferent Pathways:

a. The role of vagal afferents in the rectal response of cardiac origin:

i) Bilateral vagotomy: Sixty minutes after the sectioning of the branch of the vagus nerve supplying the heart, the cardiac nociceptors were stimulated with LAD occlusion, and rectal movement was recorded in each case. In the vagotomized animals, the reflex biphasic rectal movement induced by LAD occlusion remained unaltered even after vagotomy (Fig. 2B).
The change in the reflex IRP induced by LAD occlusion in the control and bilateral vagotomized animals was statistically insignificant, and the values are presented graphically in Figure 3.

ii) The electrical stimulation of the cardiac vagal afferents: For the confirmation of the role of the cardiac vagal afferents in the initiation of such a reflex, the distal cut-end of the cardiac vagus nerve was stimulated using a Grass SD 9 Stimulator with square wave pulses (20-40 Hz, 4-6 V, 0.2 ms for 60-120 s). Figure 2C demonstrates that the stimulation of the distal cut-end of the cardiac vagus failed to show such a rectal biphasic response.

b) The role of the sympathetic afferents:

i) The effects of stellatectomy: To study the role of cardiac sympathetic afferents in such a reflex response, we sectioned the stellate ganglia unilaterally and bilaterally. In these cases, LAD occlusion failed to produce any reflex rectal response (Fig 2 C). There was a significant change in the rectal response compared with the controls (Fig 3).

ii) The transection of the LICN: In another set of experiments, the LICN arising from the stellate ganglia was sectioned. In these cases, LAD occlusion failed to initiate the reflex rectal biphasic response (Fig. 2D).

iii) The electrical stimulation of the LICN: For the confirmation of the role of the sympathetic afferents in eliciting such a reflex rectal response, the distal cut-end of the LICN was stimulated using the Grass SD 9 stimulator with square wave pulses (40-60 Hz, 4-6 V, 0.6 ms for 60-120 s). Figure 2E shows that the stimulation of the distal cut-end of the LICN induced significant relaxation ($P < 0.01$), followed by significant contractions in the rectum ($P < 0.01$), as observed after LAD occlusion in the intact animals. In addition, the mean IRP was reduced by 21.21 + 1.65% in the relaxation phase and increased by 46.62 + 3.52% in the contractile phase due to the stimulation of the central cut-end of the LICN (Fig. 3).

5. The Efferent Pathways:

a) The role of the vagus:

i) Vagotomy below the diaphragm level: Bilateral vagotomy below the diaphragm level did not alter the normal movement of the rectum. In these animals, LAD occlusion induced a similar biphasic rectal response (Fig. 4B.). There was no significant change in the mean IRP during the relaxation and contractile phases in response to LAD occlusion compared with the animals with intact nerves (Fig. 6).

ii) The electrical stimulation of the vagal efferents: Electrical stimulation of the peripheral cut-end of the vagus nerve below the diaphragm level using the Grass SD 9 stimulator with square wave pulses (40-60 Hz, 4-6 V, 0.2 ms for 60-120 s) did not induce any change in the normal movement of the rectum, although the biphasic rectal response was totally absent (Fig. 4C).

b) The role of the splanchnic nerve:

i) Splanchnic nerve section: Sectioning of the splanchnic nerve at the abdominal level neither altered the normal spontaneous movement nor abolished LAD occlusion-induced biphasic rectal response (Fig. 4D). The mean IRP was reduced by 20.66 + 1.97% during the relaxation phase and increased by 43.64 + 4.20^{0}/0 during the contractile phase in response to LAD occlusion (Fig. 6). These average percentage changes were nonsignificant compared with control observations (Fig. 6).

ii) The electrical stimulation of the peripheral cut-end of the splanchnic nerve: The electrical stimulation of the peripheral cut-end of the splanchnic nerve using the Grass SD 9 stimulator with square wave pulses (40-60 Hz, 6 V, 0.2 ms for 60-120 s) failed to elicit any change in the spontaneous motility of the rectum (Fig. 4E). Figure 6 also shows no significant alterations in the normal intrarectal pressure after splanchnic nerve stimulation.

c) The role of the inferior mesenteric ganglia:

i) Sectioning of inferior mesenteric ganglia: Sectioning of the inferior mesenteric ganglia significantly reduced the spontaneous motility of the rectum ($P < 0.01$), but LAD occlusion-induced rectal biphasic response remained unaltered (Fig. 5B). There was an insignificant percentage change in the mean IRP during relaxation and contractions compared with the control biphasic response (Fig. 6).

d) The role of the pe1vic nerve:

i) Ventral rhizotomy at the S2-S4 level: Ventral root sectioning at the sacral (S2-S4)

region or spinal transection at the S2-S4 level reduced the spontaneous movement of the rectum significantly ($P < 0.01$, n=8). The rectal biphasic response induced by LAD occlusion was also absent in these ventral rhizotomized or spinal transected (S2-S4) animals (Fig 5C). There was a significant percentage change in the mean IRP during the relaxation and contractile phases compared with the control observation (Fig. 6).

ii) The electrical stimulation of the ventral roots at the S2-S4 level: The electrical stimulation of the peripheral cut-ends of some strands of the sacral (S2-S4 segment) ventral roots resulted in the contraction of the rectum, whereas the stimulation of some other split strands of the ventral roots led to the relaxation of the rectum or the inhibition of the spontaneous movement of the rectum (Fig. 7). Still, contractions and relaxations together were not encountered in response to ventral root-strand stimulation (electrical). During relaxation, the mean IRP was reduced by 17.28 ± 0.77%, and during the contractile phase, it was increased by 42.89 + 1.88% (Table 1). This contractile phase remained unaltered in animals pretreated with propranolol (Fig. 7B) or LNNA (Fig. 7D) but disappeared completely after atropinization (Fig. 7C). In contrast, the relaxation phase was not abolished after atropinization (Fig. 7C) or propranolol (Fig. 7B) pretreatment, but it was abolished after 10 to 20 minutes of the intra-arterial or intravenous administration of LNNA (Fig. 7D). Table 1 presents the experimental data with these neurotransmitter blockers.

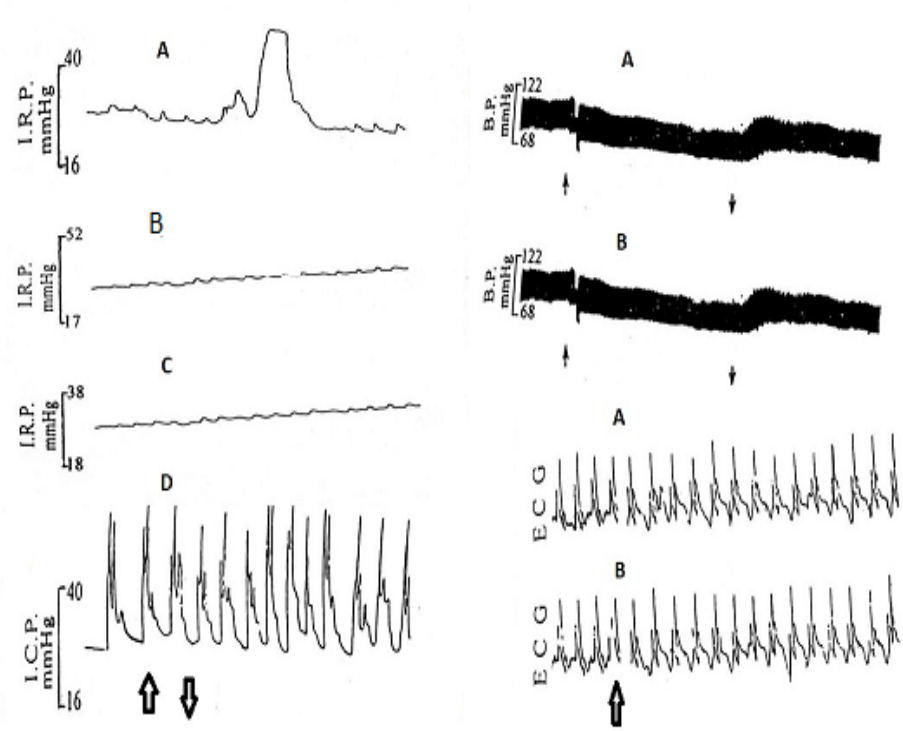

Figure 1: The left panel shows recordings of rectal movements after LAD occlusion. (A) The image shows LAD occlusion after desensitization of ventricular receptors with lignocaine pretreatment. (B) The image demonstrates atrial volume receptor stimulation by inflating the atrium. (C) The intracolonic pressure changes after LAD occlusion. (D) The right upper panel shows the effects of LAD occlusion. (A) The image illustrates the effects of LAD occlusion after ventricular desensitization (B) on blood pressure. The right lower panel shows ECG changes after LAD occlusion (A) and LAD occlusion after ventricular sensitization. (B) The upward arrow indicates the onset of LAD occlusion, and the downward arrow indicates the withdrawal of LAD occlusion.

LAD: left anterior descending

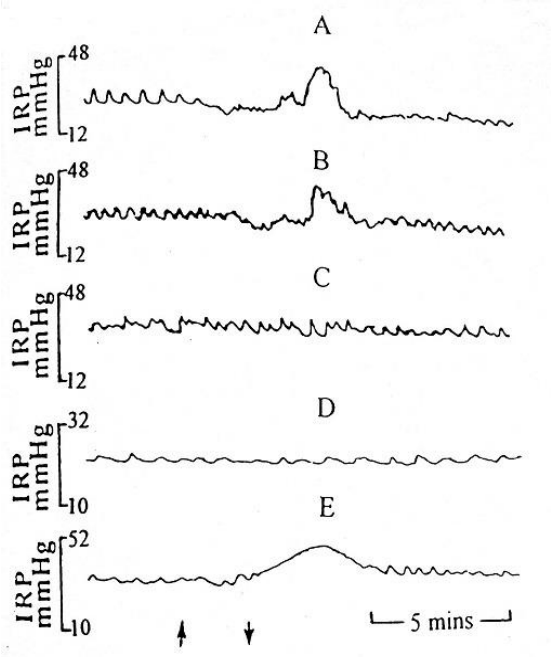

Figure 2: The images present changes in the IRP in mm Hg after LAD occlusion in the control (intact) animals. (A) The image shows the effects after cardiac vagotomy. (B) The image shows the effects after the sectioning of the LICN. (C) The electrical stimulation of the peripheral cut-end of the cardiac vagus does not evoke any rectal biphasic response, (D) whereas the stimulation of the afferent cut-end of the LICN evokes the rectal biphasic response (E).

IRP: intrarectal pressure; LAD: left anterior descending; LICN: left inferior cardiac nerve

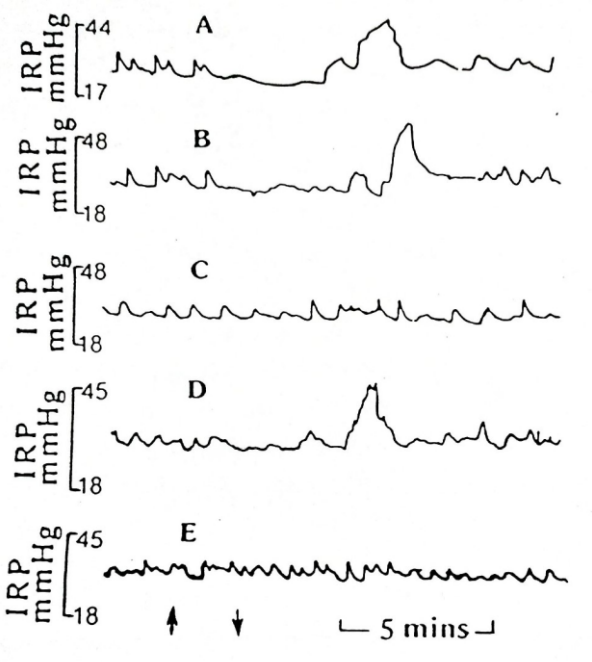

Figure 3: The images show changes in the IRP in mm Hg after LAD occlusion in the control (intact) animals. (A) The image shows the effects after gastric vagotomy (B) and in the splanchnicotomized animals. (D) Tracing C and E show the rectal motility changes after the electrical stimulation of the peripheral cut-end of the gastric vagus and splanchnic nerve, respectively. The arrows indicate the duration of LAD occlusion or the electrical stimulation of a nerve.

IRP: intrarectal pressure; LAD: left anterior descending

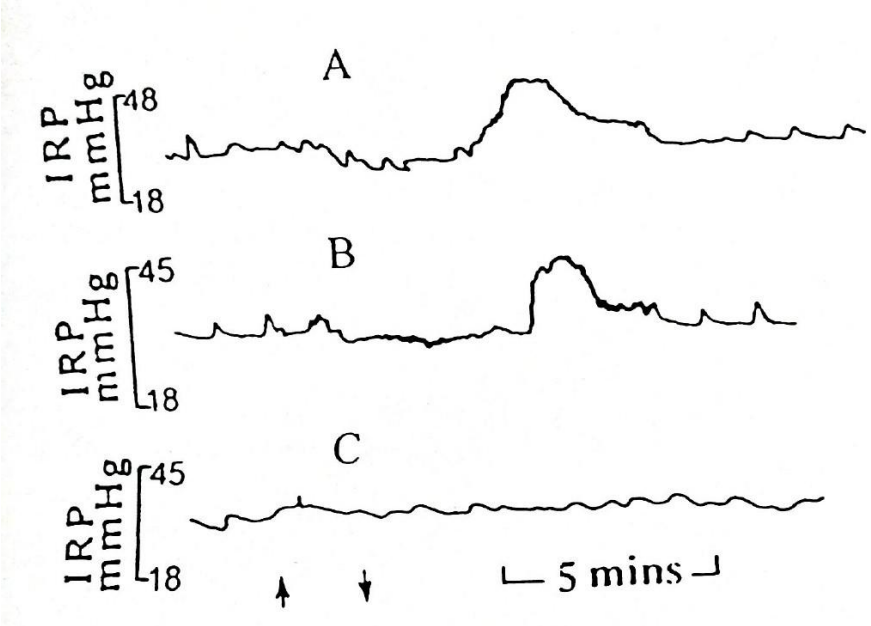

Figure 5: The images illustrate changes in the IRP in mm Hg after LAD occlusion in the control (intact) animals. (A) The image presents information regarding the animals with sectioned inferior mesenteric ganglia. (B) The image shows the effects after ventral rhizotomy at the S2-S4 level. (C) The arrows indicate the duration of LAD occlusion or the electrical stimulation of a nerve.

IRP: intrarectal pressure; LAD: left anterior descending

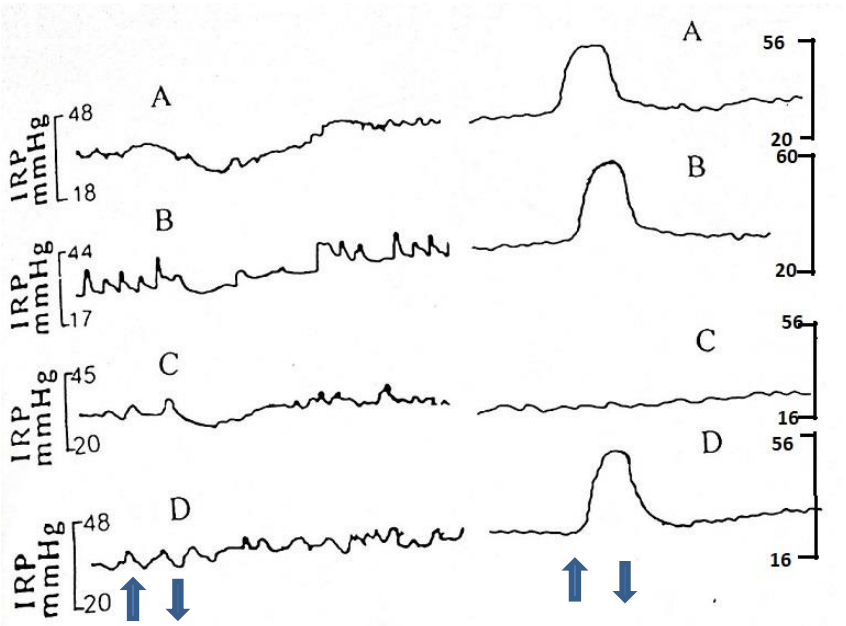

Figure 7: The images demonstrate changes in the IRP in mm Hg after the stimulation of the peripheral cut-end (efferent) of the sacral pelvic nerve-induced relaxation (the left panel) or contractions (the right panel) only in the rectum. Efferent stimulation-induced relaxation or contractions were studied in the control animals (without any drug administration). (A) The image presents information concerning the animals pretreated with propranolol. (B) The image presents information regarding the animals pretreated with atropine. (C) The image presents information concerning the animals pretreated with LNNA. (D) The arrows indicate the duration of the electrical stimulation of a nerve.

IRP: intrarectal pressure; LAD: left anterior descending; LNNA: L-arginine analogue NG-nitro-arginine

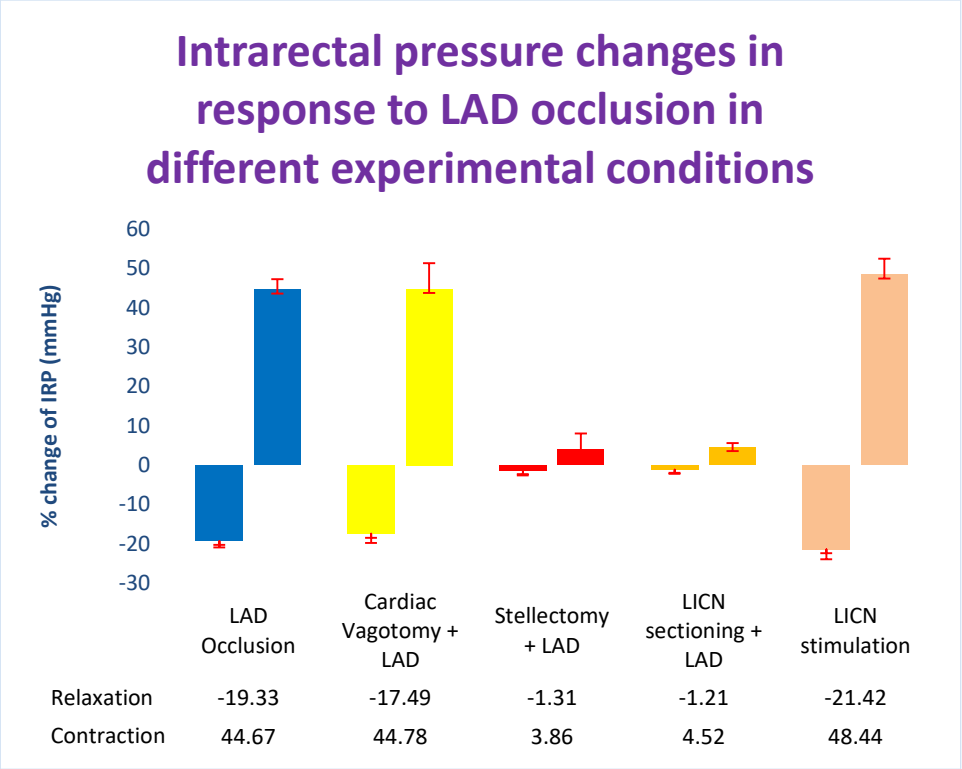

Figure 3: The image is a graphical presentation of intrarectal pressure changes in response to LAD occlusion in different experimental conditions to study afferent pathways.

LAD: left anterior descending; LICN: left inferior cardiac nerve

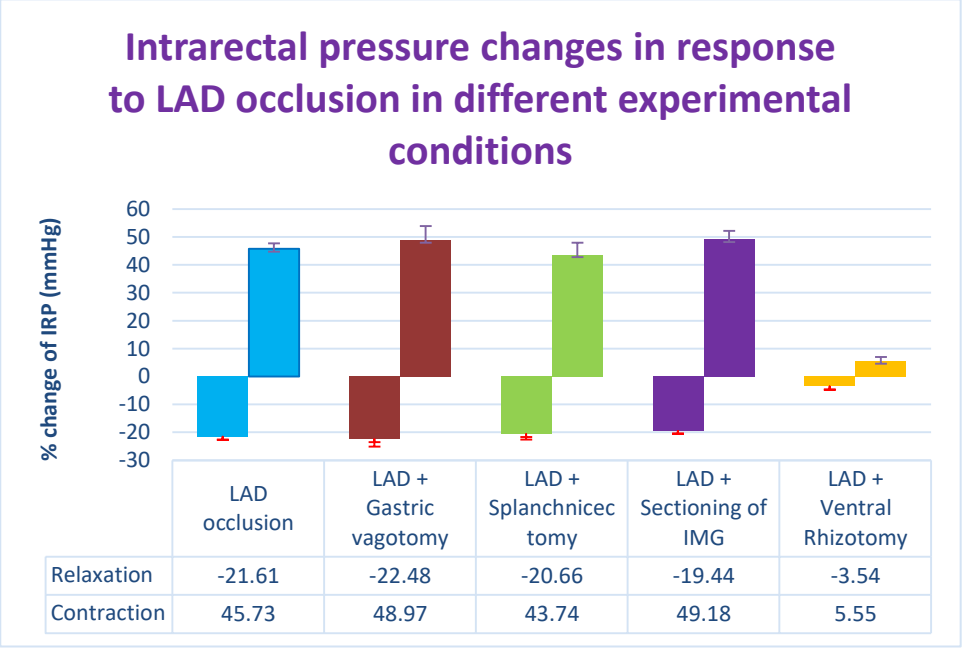

Figure 6: The image is a graphical presentation of intrarectal pressure changes in response to LAD occlusion in different experimental conditions to study efferent pathways.
LAD: left anterior descending

Table 1: Changes in the Intrarectal Pressure (IRP) (mean ± SEM) in Response to the Electrical Stimulation of the Distal Cut-End of the Ventral Roots in the S2-S4 Spinal Segments in Different Experimental Conditions

(The stimulation of some split ends caused relaxation and some other contractions.)

Experimental Conditions	Initial Mean IRP± SEM (N)	Ventral Root Stimulation	
	(mm Hg)	Relaxation (mm Hg)	Contractions (mm Hg)
Control stimulation	30.08± 1.38 (24)	25.35 ± 1.36 (22)	42.83 ± 1.18 (24)
Stimulation after pretreatment with propranolol	38.83 ± 2.13 (6)	31.67 ± 2.02 (6)	46.0 ± 3.05 (6)
Stimulation after pretreatment with atropine	35.53 ± 1.97 (6)	28.0 ± 2.06 (6)	37.0 ± 1.84 (6)
Stimulation after pretreatment with LNNA	30.0± 2.47 (8)	29.33 ± 2.51 (8)	42.33± 2.60 (7)

L-arginine analogue NG-nitro-arginine

DISCUSSION

It is well-documented that chemical mediators such as bradykinin, prostaglandins, and lactic acid are released during myocardial ischemia and participate in the genesis of cardiac pain by exciting the cardiac nociceptors. The cardiac pain initiates different visceral and somatic reflexes. In experimental terms, pain is a conscious experience. Nonetheless, according to Woodworth and Sherrington [24] and Sherrington, [25] in animals recovering from anesthesia (light anesthesia or decerebrate acute preparations), it is easy to obtain pseudo-affective reflexes by applying noxious stimuli to the heart. Brown [26] and Malliani et al [27] reported that experimental coronary artery occlusion in lightly anesthetized cats often provoked cardiac reflexes. The excitation of cardiac sensory receptors with the epicardial application of chemical substances (eg bradykinin, nicotine, prostaglandins, lactic acid, and veratridine) may elicit bradycardia and hypotension, along with the relaxation of the stomach. [3-7] Koley et al [5-7] reported that the stimulation of the cardiac nociceptors of the left ventricle by LAD occlusion or the local application of different algesic agents on the surface of the left ventricle increased the movement of the forelimbs, the contraction of the nictitating membrane, urine flow, and the contraction of the urinary bladder and rectum. The results of the present study confirmed that myocardial ischemia experimentally induced with LAD occlusion excited the cardiac sensory receptors, presumably nociceptors, to initiate a biphasic rectal response. Our findings also indicated that the increased contraction of the rectum might be the causative factor of defecation during cardiac ischemia-induced heart failure, whereas colonic movements were not associated with this defecatory urge since no change was seen in colonic movements after LAD occlusion. These rectal responses to LAD occlusion may result from cardiac ischemia-induced pain, supported by the electrophysiological study of the heart. ECG changes in response to LAD occlusion showed ST-segment elevation and T-wave inversion, the characteristic signs of myocardial ischemia and, consequently, cardiac ischemic pain. [28,29] Moreover, these changes in rectal motility following LAD occlusion were reflexogenic because they were totally abolished after the desensitization of the cardiac sensory receptors with the epicardial application of lignocaine, which inhibits catecholamine release from the sympathetic nerve endings in the ischemic myocardium with its endo-anesthetic and membrane-stabilizing properties. [30]

Evidence also indicates that atrial receptors also initiate different reflexes. Douffeil and Kramer [31] claimed that left atrial distension was at least equally important for inducting a depressor response upon aortic occlusion.

Diuretic and natriuretic responses to atrial receptor stimulation were demonstrated by Sivanathan, Kappagoda, and Linden [32] and also Karim et al. [33-35] Nevertheless, our study on atrial distension showed that the atrial receptors had no role at least in initiating a rectal response because the stimulation of these receptors by distending the left atrial appendage failed to elicit any change in the rectal response.

The results of the current study also confirm our previous study, in which we found that the afferent limb for such a reflex lay in the cardiac sympathetic nerves because sectioning of the cardiac vagal fibers did not alter such a reflex. However, in these sympathectomized animals, the occlusion of the left coronary artery failed to show such a rectal biphasic response. The stimulation of the distal cut-end of the LICN reprecipitated the reflex, while the stimulation of the distal cut-end of the vagus failed to initiate such a reflex. It is accepted that sympathetic nerves are essential to the perception of cardiac pain. [36, 37] Malliani and Brown [38] and Brown and Malliani [39] recorded the impulse activity of afferent cardiac sympathetic nerve fibers excited by the interruption of the left coronary artery flow, leading to myocardial ischemia. Brown and Malliani [39] and Uchida and Murao [9] also observed a few silent fibers in the sympathetic that became active during the interruption of coronary artery blood flow. Guzman et al [20] and Pal et al [12] reported that the administration of bradykinin, lactic acid, or nicotine and the occlusion of the LAD resulted in increased afferent sympathetic activity. They showed that cardiac nociception was related to the excitation of the cardiac sympathetic A-delta and C fiber endings. The present study also confirms the finding since sectioning the LICN abolished the LAD occlusion-induced rectal biphasic response. The finding was further confirmed with the electrical stimulation of the distal cut-end of the LICN, which produced a similar type of rectal biphasic response as seen after LAD occlusion. Thus, the result indicates that cardiac receptors, presumably nociceptors, [12, 27] are excited due to the occlusion of the left coronary artery, resulting in cardiac-rectal reflexes, with the afferent pathways lying in the cardiac sympathetic fibers.

Gastrointestinal motility is influenced by 2 divisions of efferent nervous activity: sympathetic and parasympathetic. The sympathetic fibers emerge from the thoracolumbar spinal cord, and the parasympathetic axons leave the cerebrospinal axis as vagal and pelvic innervation. [40] We found that the biphasic rectal response of cardiac origin was mediated through the efferent activity of the spinal sacral ventral roots, originating in the pelvic nerve plexus. Both rectal relaxation and contractions are mediated by the sacral ventral roots since sacral ventral rhizotomy or spinal transection in the S2-S4 region totally abolished such a reflex. This finding is further evident from the stimulation of the ventral roots, resulting in both relaxation and contractions of the rectum. Our findings also confirmed that LAD occlusion-induced rectal relaxation and contractions were present even after the sectioning of the inferior mesenteric ganglia, from which the hypogastric nerve plexus arises. In other words, the sectioning of the inferior mesenteric ganglia failed to abolish such a cardiac-rectal reflex, indicating no role for such ganglia in mediating this type of rectal biphasic response. The vagus or splanchnic nerves also play no role in eliciting such a reflex since gastric vagotomy or splanchnic nerve sectioning failed to alter this cardiac-rectal reflex. The stimulation of either of these nerves also failed to induce such biphasic changes in the rectum. Hence, it may be concluded that the pelvic nerve is the efferent limb for such a reflex rectal biphasic response.

The relaxation phase, in response to the electrical stimulation of the distal cut-end of the ventral roots arising from the S2-S4 region, led to either relaxation or contraction of the rectum. The stimulation of some fibers resulted in relaxation, which was abolished in NO inhibitor (LNNA)-pretreated cats but not in atropine or propranolol-pretreated animals. However, the stimulation of some other fibers resulted in only the contraction of the rectum, which was abolished after atropine pretreatment and not propranolol or LNNA. These sets of findings indicate that the motor pathway of this rectal biphasic response is mediated by the inhibitory NO pathway (the relaxation phase), chiming with the results reported by Toda et al [41] and Rattan et al. [42] In some other experimental setups, the contractile response is mediated by the stimulatory cholinergic pathway (the contractile phase).

CONCLUSIONS

Based on the results of the present study, we conclude that myocardial ischemia induced by LAD occlusion is the origin of the rectal biphasic response, which may account for defecatory urge during myocardial infarction-induced heart failure. Furthermore, we conclude that the efferent and afferent pathways for these reflexogenic rectal responses are the sacral pelvic nerve and cardiac sympathetic nerve, respectively. In addition, the relaxation and contractile phases of this response are mediated through the NO pathway and the cholinergic pathway, respectively.

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Conflict of Interest: None.

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