Cardiac nociception-induced urinary bladder movement:
the afferent pathways

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Keywords: Cardiac nociception, urinary bladder, left anterior descending coronary artery, nicotine, ventricular receptors, cardiac sympathetic, vagus, intravesicular pressure, vesicular motility, supraspinal centre.

Abstract: Myocardial ischaemia stimulates ventricular nociceptors and induces various visceral symptoms. In cats, occlusion of the left anterior descending coronary artery (LAD) for 3–4 min or epicardial application of nicotine (100-200 pg mL⁻¹) for 30 s induced a biphasic change in the urinary bladder (vesicular) motility – an initial large contraction followed by inhibition. Desensitisation of the ventricular receptors by epicardial application of lignocaine abolished the vesicular responses. The initial large contraction was partially prevented by bilateral vagotomy and partially by cardiac sympathectomy. The inhibition phase was completely abolished by cardiac sympathectomy. Spinal transection at the C7 – C8 level prevented the spontaneous as well as the reflex bladder responses. Decerebration abolished the initial large contraction and potentiated the inhibition phase. These observations indicate that ventricular receptors are involved in LAD occlusion or epicardial –nicotine induced bladder responses. Afferents for such responses run along both cardiac sympathetic and vagus, and the supraspinal centre is involved.

Introduction: Pathophysiological conditions of the heart such as cardiac ischaemia and myocardial infarction are often associated with visceral disturbances including nausea, vomiting, passing of urine and stool [1–3]. Reduction in coronary blood flow in animals activates cardiac receptors with vagal and sympathetic myelinated and unmyelinated fibres [4]. Afferent fibres in the cardiac sympathetic nerve signal the noxious events in the heart [5] and have been considered the essential pathway for the transmission of cardiac pain during myocardial ischaemia [6].

Yet the role played by the vagal afferents in signalling cardiac pain cannot be ignored [7]. Abrahamsson and Thoren [1] have shown that stimulation of ventricular receptors can cause reflex bradycardia, hypotension and gastric relaxation. Kiley et al. have also shown biphasic rectal [2] and bladder movement [3] after ventricular receptor stimulation.

Therefore, in the present study we attempted to elucidate the role of afferent cardiac nerves and the centre involved in the manifestation of ventricular receptor induced bladder movement.

Materials and methods: 21 cats of either sex were obtained from the supplier (Recta Ghosh, Calcutta, India) and kept for at least one week in the animal house. They received water ad libitum and were fed with freshly cooked rice and fish twice daily. They weighed 2–3 kg after an overnight fast with water available ad libitum.

The cats were anaesthetised with α-chloralose (60 mg kg⁻¹ body weight) after initial induction with ether. The femoral vein was cannulated to administer saline and drugs. A glucose solution (5%) in physiological saline (0.95%) with 0.01M sodium bicarbonate was administered by drip feed into the femoral vein at a rate of 0.1 mL min⁻¹ kg⁻¹ body weight throughout the experiment to maintain body fluid volume and pH. Body temperature was maintained at 37 ± 1°C.

The trachea was cannulated to provide artificial respiration. The femoral artery was cannulated for recording the blood pressure on an INCO polygraph (Model 201) via an INCO pressure transducer (Model T-301). The urinary bladder was cannulated with a polyethylene catheter inserted via the urethra. The urinary bladder was filled with 10–15 mL warm (37°C) saline solution and the bladder volume was checked time to time to maintain a constant intravesicular pressure (IVP).

The IVP changes due to contraction or inhibition of the urinary bladder was recorded on INCO polygraph through an INCO pressure transducer following the method of Koley et al. [8]. The left chest was opened by removing the 2nd–5th thoracic ribs, keeping the animals under artificial ventilation. The epicardium was cut longitudinally and a cradle was made with the cut ends of the epicardium. Ventricular receptors were stimulated either by occluding the Left Anterior Descending (LAD) coronary artery for 3–4 min by means of a snare put around it or by applying nicotine (100–200 pg mL⁻¹) with a cotton applicator over the epicardial surface of the left ventricle for 30 s.

Bilateral vagotomy was performed in six cats. Inferior cardiac nerves (ICNs) were sectioned in six cats and the experiments were repeated in vagotomised or cardiac sympathectomised cats. Laminitectomy was performed in four cats at the cervical level and after intradural injection of 0.2 mL of 2% lignocaine the spinal cord was transected at the C5 – C6 level.

Decerebration was performed in three cats at the midcollicular level under ether anaesthesia. Experiments were repeated after 2 h of spinal transection or decerebration.

The initial normal contraction pressure was obtained by averaging peak pressures of 10 successive contractions. The intravesicular pressure (IVP) was recorded in mmHg. Changes in IVP during contraction or inhibition were obtained from the difference between the initial pressure and the pressure during contraction or inhibition. They are expressed as % change ± SEM.

The significance of differences of results between control...
ICN sectioning experiments showed that cardiac sympathetic afferents are involved in the manifestation of the inhibitory phase and also have partial involvement in the manifestation of the initial large contraction. The total abolition of the reflex bladder response after simultaneous sectioning of vagus and ICNs confirms these observations.

Koley et al. [8] found that spinal cord transection at the level of C7-C5 abolished the spontaneous bladder contractions as well as intravenous nicotine or DMPP-induced contractions. In the present study, spinal cord transection at the C7-C5 level abolished the spontaneous as well as reflex vesicular movement. Kuru and Yamamoto [11] reported that the micturition centre of the pontine vesicoconstrictor area has bilateral centrifugal connections with bulbar vesicoconstrictor centres as well as with sacral vesicoconstrictor centres via the reticulospinal tract.

Spinal transection at the C7-C6 level disrupted the connections between the pontine and other vesicoconstrictor centres from the sacral micturition centre and thus abolished the spontaneous as well as reflex bladder movement. This clearly indicates the involvement of the supraspinal centre in this reflex. Tokunaga and Kuru [12] have shown that at each level of the brain stem the vesicoconstrictor and vesicorelaxer areas are situated close to, but are separate from, each other.

Decerebration at the midcollicular level abolished the initial large contraction and potentiated the inhibition. This may be due to withdrawal of influence of the superior or intercollicular vesicoconstrictor areas and also that of the facilitatory centres of the cerebral cortex. In addition, in the absence of these facilitatory influences the vesicorelaxer area of the inferior collicular level becomes more powerful and potentiates the inhibition.

Our observations also suggest that, although the pontine vesicoconstrictor and vesicorelaxer areas remain intact in decerebrate animals, the initial contraction of the cardiovesicular reflex fails to occur. The reason is probably that the superior and intercollicular and also the cerebral cortical vesicoconstrictor centres exert a facilitatory influence on the pontine micturition centre.

Therefore, these observations suggest that both LAD occlusion and epicardial nicotine application stimulates the ventricular receptors afferent fibres which travel in vagal and cardiac sympathetic nerves to cause reflex biphasic movement of the urinary bladder. The supraspinal centres in the cerebral cortex and subcortical centres directly influence the reflex modulation of urinary bladder movement.


We gratefully acknowledge financial support from the University Grants Commission, Government of India.

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VESICULAR REFLEXES OF CARDIAC ORIGIN

THESIS SUBMITTED FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY (SCIENCE)
of the
UNIVERSITY OF CALCUTTA
SEPTEMBER, 1997

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