MATHEWS

Research Article

Mathews Journal of Cardiology

J Receptor Reflexes Terminate Exercise to Reduce Pulmonary Inflow

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ABSTRACT

A great increase in ventilation and cardiac output is set off by and even before rapid locomotion begins. An investigation into the neural mechanisms involved revealed that the subsequent increase in pulmonary blood flow stimulates J or juxtapulmonary capillary receptors that lie in the pulmonary parenchyma. Reflexes arising from stimulation of these sensory receptors are an increase in ventilation accompanied by sensations of breathlessness, bradycardia and reduction or stopping of exercise. Of these, the significance of reflex bradycardia is to reduce the great rise in pulmonary capillary pressure during strenuous exercise and protect the pulmonary arterioles from rupture as they are vulnerable to mechanical stress.

Keywords: Juxtapulmonary capillary receptors, dyspnoea, bradycardia, strenuous exercise, pulmonary blood inflow, pulmonary arterioles

INTRODUCTION

The juxtapulmonary capillary receptors or J receptors are a group of sensory receptors that lie in the pulmonary interstitium in juxtaposition to pulmonary capillaries innervated by vagal afferents and are accessible only via the pulmonary circulation [1]. They are naturally stimulated by any condition that produces pulmonary interstitial congestion which could either be by an increase in interstitial volume/pressure / pulmonary capillary pressure or a rise in the right atrial pressure [2-4]. As a consequence of a rise in pulmonary capillary pressure, fluid flows out from the capillaries into the interstitial tissue (spaces) where these receptors lie [5], thereby increasing the interstitial volume and leading them to function as interstitial stretch receptors to give rise to their reflex effects. The J receptors however do not signal pulmonary capillary pressure per se.

ACCELERATION OF BREATHING & RESPIRATORY SENSATIONS

The characteristic reflexes elicited by these receptors are an acceleration of breathing, inhibition of exercise and bradycardia. These have been demonstrated in animals by intravenous injection of phenyl diguanide [1-6] and in human subjects by intravenous injection of lobeline, a short-acting alkaloid [7-11]. In human subjects, accelerated breathing

Vol No: 07, Issue: 01

Received Date: March 15, 2023

Published Date: April 15, 2023

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Citation: Anand A. (2023). J Receptor Reflexes Terminate Exercise to Reduce Pulmonary Inflow. Mathews J Cardiol. 7(1):26.

is accompanied by a variety of characteristic sensations in the upper chest such as an upward rising pressure from chest to throat, choking, feeling of being short of breath, a desire to cough or an actual cough [7]. These sensations have been shown to be akin to dyspneic sensations of exercising patients of left ventricular dysfunction, mitral stenosis and Eisenmenger syndrome [8,9,11].

INHIBITION OF EXERCISE

In Animals

In response to right intra-atrial injection of phenyl diguanide, the monosynaptic reflexes evoked from flexor and extensor hind limb muscle nerves in paralyzed, anaesthetized cats were depressed for several seconds [6]. This depression of the monosynaptic reflex is present even after the administration of atropine which lessened the hypotension due to the subsequent injection of phenyl diguanide. The blood gas tensions measured after the administration of phenyl diguanide were not altered to a degree that would suggest that the depression was secondary to a change in arterial gas tensions. The afferent pathway of this reflex runs in the vagus nerve since it was possible to abolish it by vagotomy. Furthermore, since this reflex could be abolished by intercollicular decerebration, it was concluded that the reflex inhibition of the monosynaptic reflex, that was seen to last for several seconds, was produced by stimulation of J receptor endings via regions of the brain cephalad to the intercollicular level.

In Humans Subjects

H-reflex

In a study on healthy individuals, on stimulating J receptors naturally i.e. by increasing pulmonary blood inflow, by releasing lower body negative pressure that increases venous return by a litre [12,13], a reduction in the amplitude of the Hoffman or H-reflex, was seen [10]. The H Reflex reflects a change in the excitability of the motoneurone pool [14] and this study was undertaken to demonstrate if a reflex inhibition of locomotion would occur when J receptor activity increases in the presence of their natural stimulus (see above). The results showed that this manoeuvre was accompanied by a notable respiratory augmentation and respiratory discomfort which the subjects described as a feeling of pressure, choking or an obstruction in the throat with a need to take in more air. These sensations are similar to those felt in response to small doses of intravenous lobeline [10].

Bradycardia

Raising pressure in the pulmonary vasculature produces reflex bradycardia [15,16). Bradycardia also occurs when J receptors are stimulated by PDG or capsaicin [1,17]. It is well known since the demonstration by Krogh & Lindhard in 1912 [18] that even before the start of exercise cortical inputs lead to a great increase in ventilation and heart rate. The resulting increase in cardiac output not only increases oxygen supply to the exercising muscles but also leads to an increase in pulmonary blood inflow through the pulmonary circulation with an increased pulmonary capillary pressure. This results in the stimulation of J receptors [3]. On being thus stimulated impulses from these receptors stimulate certain central pathways lying in the region of the cingulate gyrus and the caudate nucleus [19] which provide inhibitory inputs to the motoneurones of somatic muscles; thereby inhibiting muscular contractions and terminating exercise.

CONCLUSION

Since the J receptors are stimulated by an increase in pulmonary blood flow which normally occurs during moderate exercise [3] to yield powerful reflex effects, the net effect of bradycardia arising from J receptor stimulation during strenuous exercise is to reduce the great rise in pulmonary capillary pressure (a rise that is consequent to the cortical drive to run fast) and protect the pulmonary arteries from rupture as they are vulnerable to mechanical stress [20,21]. Rupture of pulmonary arteries can happen with an explosive increase in ventilation as in the case of elite athletes including swimmers [22], rowers, runners [23], scuba divers and high-performance athletes[21] in whom haemoptysis or haemorrhagic pulmonary oedema, cough, dyspnoea have been reported. Smaller increases in blood flow would also be effective in producing noticeable reflex effects but in extreme cases of athletic and other forms of exercise its reduction or inhibition reflexly by J receptor stimulation will protect the pulmonary arteries from rupture as termination of exercise will reduce cardiac output, and pulmonary blood inflow and pulmonary vascular pressures.

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