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**Vagal Modulation of Recurrent
Laryngeal Motoneurone
Discharge**

**A thesis presented in partial fulfilment of the
requirements for the degree of Master
of Science in Physiology at Massey
University, New Zealand**

William Kian Meng Tan

1986

Vagal Modulation of Recurrent
Laryngeal Motoneurone
Discharge

Volume I

To Mum and Dad

and all in my family

for all of the richness of life

and love they have shared with me

".....the Lord God formed man of dust from the ground,
and breathed into his nostrils the *breath of life* and man
became a living being."

Genesis 2:7

Vagal Modulation of Recurrent Laryngeal Motoneurone Discharge

Abstract

The larynx has considerable influence on the rate of respiratory airflow, particularly during expiration. The laryngeal muscles are largely controlled by the recurrent laryngeal nerve (RLN) which exhibits respiratory periodicity in its discharge. The effects of the Hering-Breuer inflation reflex, on this periodicity has been studied, but there is little information on the role of the various groups of lung receptors in modulating the patterns of recurrent laryngeal motoneurone (RLM) discharge during eupnoeic breathing.

The purpose of this investigation was to examine the effects of changes in volume-related feedback and other vagal afferent inputs on the activity of RLMS. The discharge patterns of single fibres in the recurrent laryngeal nerve were classified and their responses to pulmonary inflation and deflation before and during pulmonary stretch receptor (PSR) block by sulphur dioxide were compared, using anaesthetized, spontaneously breathing rabbits. On the basis of observations in this study, RLMS were classified into:

- a) phasic-inspiratory (P-ILMS)*
- b) tonic-inspiratory (T-ILMS)*
- c) phasic-expiratory (P-ELMS)*
- d) tonic-expiratory (T-ELMS)*

and their firing patterns were described.

It was found that the frequency and duration of P-ILM discharge in inspiration increased after the inhibition of PSR activity. During lung inflation, some RLMs were inhibited whereas others showed either a low-frequency or a high frequency tonic discharge when PSR were intact. During PSR block, lung inflation failed to inhibit phasic P-ILM discharge. While ILM activity was increased during lung deflation, that of ELM was decreased. These responses persisted in a modified form during PSR block. Breathing through an added dead space increased the frequency of P-ILM discharge during inspiration. During augmented breaths, the frequency and duration of P-ILM activity greatly exceeded P-ILM activity during normal breaths, whereas the activity of T-ELM was reduced.

Further experiments were carried out on rabbits during neuromuscular junction block and artificial ventilation. With stretch receptors functioning, simultaneous recordings of the recurrent laryngeal nerve (RLN) and phrenic nerve (PN) showed that the onset of the activities of both nerves occurred during the deflation phase of ventilation. Changing the tidal volume to 50% or 100% of eupnoeic value could not unlink the recurrent laryngeal and phrenic bursts from the deflation phase of the pump cycle. During PSR block, RLN and PN discharges occurred with no set relation to ventilation at spontaneous

resting tidal volume. At 100% higher tidal volume, RLN and PN bursts were initiated more frequently by the deflation phase. These timings were changed into a "free-running" pattern by decreasing the tidal volume to half eupnoeic value.

These results suggested that vagal afferents modulate RLM discharge during eupnoeic breathing: PSR inputs terminate ILM discharge whereas RAR activity, which occurs at functional residual capacity, initiates the onset of ILM discharge and extends its activity. The role of RAR is compatible with its effects in shortening ELM activity if the initiation of ILM discharge is considered as a termination of ELM activity. That RLN and PN responded in almost identical manner to changes in vagal afferent inputs suggests that PSR and RAR may operate through similar central pathways to modulate both neural outputs. The discharge patterns of RLNs and their responses to changes of pulmonary afferent inputs, are probably related to the role of the larynx in regulating upper airway resistance.

Acknowledgements

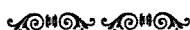
" If I have seen further than the rest, it is
because I have stood on the shoulders of giants."

Sir Isaac Newton

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" *Sola Dei Gloria* "

Johann Sebastian Bach

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Chapter One : Introduction

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