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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 RLMs and their responses to changes of pulmonary afferent inputs, are probably related to the role of the larynx in regulating upper airway resistance.

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"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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"Soli Dei Gloria"

Johann Sebastian Bach

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Chapter One : Introduction

1.1 A Brief History of Ideas About the Larynx

> "The larynx can close so accurately that the breath violently forced out of the thorax does not open it. Nature placed the epiglottis before the orifice of the larynx as a lid, to stand erect during all the time the animal breathes and to fall down upon the larynx when anything is swallowed."
>
> Galen (AD 130-199)

Writing in the second half of the fourth century B.C., Aristotle referred to the larynx in his Historia Animalium (" Collection of facts about animals "). In his review of the parts of the human body, he said,

> "The neck is the part between the face and trunk. The front part of this is of gristle, and through it speech (phone) and respiration (anapnoe) take place; it is known as the windpipe."

After Aristotle, five centuries were to pass before there was a mention of the larynx in the records of Galen. Galen taught that the larynx comprises three cartilages namely thyroid, innominate (cricoid) and arytenoid. His explanation of effort

closure of the larynx, cited earlier, was impressive for his time. He pointed out that the passageway of the larynx changes gradually from wide to narrow and then back again to wide during each breath, noting the presence of an opening in each side of the glottis leading to a relatively large cavity, the ventricular air sac. Galen continues (in: Fink, 1975):

> "When the air enjoys the use of broad passageways to enter and leave the body, none of it is thrust aside into these cavities. But when the passage is obstructed, the confined air is thrust laterally with violence and opens the orifice. When the cavities have been filled with air, the mass of the glottis (the vestibular folds) necessarily spreads out into the passageway and closes it completely."

A comprehensive explanation of the various mechanisms of the larynx was never attempted by Galen or the Renaissance anatomists. Physiologists before 1861 were preoccupied with deciphering the method of sound production and gave little attention to the role of the larynx in glottic closure or in respiration. Only with the invention of the laryngoscope did a correct description of the behavior of the parts of the larynx become possible.

The 20th century, with the development of new methods of recording and of powerful new techniques, including high-speed cinematography, X-ray photography, electrophysiological methods and, in particular, electromyography and electroneurography, heralded in a new era in laryngeal research. The purpose of the present study which employed techniques of electroneurographic recording, was to identify the efferent activity in the recurrent laryngeal nerve and to determine the role of lung receptors in modulating recurrent laryngeal motoneurone discharge. To have a better understanding of the influence of vagal afferent inputs on recurrent laryngeal motoneurone discharge, a brief review of the literature pertaining to the following topics is necessary:

a. Anatomy of the larynx
b. Respiratory function of the larynx
c. Pulmonary receptors and their effects on laryngeal function

1.2 Anatomy of the Larynx

The detailed internal structure of the larynx varies considerably from species to species (Negus, 1949) and that of the rabbit has been described by Barone (1976). For the purpose of this study, only a brief account of the laryngeal cartilages, and the intrinsic laryngeal muscles and their innervation is included here.

The larynx is situated high in the neck and forms a continuous tube with the trachea. It consists of a skeletal framework of articulated cartilages bound together by ligaments and membranes and activated by the intrinsic laryngeal muscles.

The rabbit's larynx is supported by nine cartilages : the thyroid and cricoid cartilages and the epiglottis are single cartilages; the arytenoid, corniculate and cuneiform cartilages are paired (Figure 1).

The thyroid cartilage is the largest cartilage and encloses the other cartilages ventrally and laterally. The cricoid cartilage is annular in shape and its dorsal border is broader than the ventral one. From the caudal aspect of the cricoid cartilage there is a membranous attachment to the first tracheal ring. The epiglottic cartilage is attached to the middle of its cranio-ventral border and the whole cranial margin of the thyroid cartilage is connected to the hyoid apparatus. The dorsal border articulates caudally with a point on the lateral aspect of the cricoid cartilage. The arytenoid cartilages are triangular pyramids which articulate with the cranial border of the cricoid cartilage. The lateral cricoarytenoid, thyroarytenoid and dorsal cricoarytenoid muscles are attached to the arytenoid

cartilages (Figure 2). The action of these muscles is to cause adduction and abduction of the vocal cords (Figure 3). The epiglottic cartilage is attached to the cranial border of the thyroid cartilage and supports the epiglottis. Contraction of the thyrohyoid muscle, stylohyoid muscle and the median and inferior constrictors of the pharynx moves the larynx as a whole cranially. The sternothyroid muscle moves the whole larynx caudally when it contracts. The dorsal cricoarytenoid muscles are considered the abductors whereas the lateral cricoarytenoid, the thyroarytenoid and the cricothyroid which increase the tension of the vocal cords are considered the adductors.

The lumen of the larynx can be divided into three areas (Figure 4). The anterior part, which is known as the vestibule, extends from the tip of the epiglottis cranially to the false vocal cords caudally. The false cords or ventricular bands are two folds of mucosa which stretch from the caudal surface of the epiglottis to the tips of the arytenoid cartilages. Caudal to the false vocal cords, the true vocal cords stretch from the apices of the arytenoid cartilages to the thyroid cartilage near the base of the epiglottis. The middle portion of the larynx is that area between the true and false cords. It has a small sac on each lateral aspect, known as the ventricle. The slit between the true vocal cords is

known as the glottis. The width of the glottis is varied by the action of the intrinsic laryngeal muscles.

The motor and afferent innervation of the body muscles is usually found in the same nerve trunk. The neural control of the intrinsic laryngeal muscles, however, is accomplished in a different way, since most of the motor fibres to these muscles travel in the recurrent laryngeal nerve (RLN), whereas the afferent limb of this reflex is provided mainly by sensory fibres in the cranial laryngeal nerve (CLN, also known as superior laryngeal nerve in man) (Andrew, 1955; Green and Neil, 1955) (Figure 5).

The cranial and recurrent laryngeal nerves arise from the vagus nerves. On the left side, the RLN descends far into the chest. It loops under the aorta before it comes back into the neck and supplies the laryngeal muscles. The right RLN loops under the right subclavian artery.

Motor innervation of all the intrinsic laryngeal muscles is served by the RLN, except for the cricothyroid which is innervated by the external branch of the CLN and by the plexus pharyngeus (Exner, 1884; Harreveld and Tachibana, 1961) (Figure 6).

The sensory outflow from the larynx (rostral to the base of the vocal cords) comes out via the cranial

laryngeal nerve fibres (Ogura and Lam, 1953). The recurrent laryngeal nerve supplies the sensory innervation to the posterior end of the organ, caudal to the base of the vocal cords (Murtagh and Campbell, 1951).

A number of sensory receptors have been detected in the laryngeal structures of mammalian species. The most detailed studies have been done with the rat where mucosal touch receptors and chemoreceptors and joint proprioceptors have been found. (Andrew, 1954, 1956). Pressman and Kelemen (1955) suggested that the distribution of sensory receptors throughout the larynx is adapted to elicit prompt motor reactions. The degree of sensitivity is greatest around the entrance to the larynx; a distribution which corresponds with the concept that the larynx forms part of the protective mechanism for the airway.

1.3 Respiratory Function of the Larynx

The Bible says that earning a living by sweat-provoking hard work was Adam's punishment for eating the fruit of the tree of knowledge (Genesis 3:19). Legendarily the fruit was supposed to have stuck in Adam's throat and remained visible there as "Adam's apple". A justification for the biblical connection between man's birthright of heavy manual labour and the prominence of the "Adam's apple" is quite obvious

when one considers certain behavioral facts. Violent efforts such as hurling a stone, or heavy weightlifting, demand not only a firm stance but also the ability to hold one's breath by closing the larynx. The need for breathholding arises because a breathing chest is a relatively weak chest, whereas a chest closed at the larynx can be braced by the abdominal wall muscles for maximal exertion by the arm. Hence, such holding of breath calls for a hermetic seal of the larynx against the escape of air.

The ability to purposely prevent air from escaping the lungs at certain times of physical activity is only one of the functions of the larynx. The larynx is also involved in a multiplicity of other functions ranging from relatively simple reflex events such as gagging, sneezing and coughing to more complex motor activities such as swallowing. Besides guarding the lungs from invasion by foreign matter, it also plays a role in regulating expiratory airflow and duration by modifying airway resistance (Bartlett et al, 1973; Gautier et al, 1973; Remmers and Bartlett, 1977). The vocal cord exhibits a reciprocating respiratory motion, abducting on inspiration and moving toward the midline (adduction) during expiration, thus increasing laryngeal resistance.

1.31 Respiratory Movements of the Vocal Cords

As far back as 1829, Mayo discovered the existence of rhythmical expiratory narrowing of the glottis in a dog whose throat had been incised below the hyoid bone. The presence of a similar rhythm was observed soon thereafter - again by Mayo in several men who had attempted to commit suicide. Garcia (1855) made only a brief reference to the respiration rhythm in his communication to the Royal Society:

> "At the moment when the person draws a deep breath, the arytenoid cartilages become separated by a very free movement."

Czermak (1861) made the following observations:

> "..... in quiet, deep respiration, the glottis which has momentarily the form of a lozenge or is divided into an anterior and posterior part by the projection of the arytenoid processes, assumes the form of a large oblong opening."

A laryngoscopic study of the larynx did not appear until 1942, when Pressman published a set of three photographs accompanied by the following description:

> "The most obvious respiratory movements of the larynx consist of abduction of the cords away from the midline with consequent widening of the chink during inspiration and

> a relative adduction toward the midline, with narrowing of the chink during expiration. This movement is so slight in certain persons during quiet respiration as to be almost imperceptible, with the cords assuming during rest, a position just lateral to the midline. As the depth of respiratory movement increases, this excursion becomes easily recognizable. The lateral movements of the cords during inspiration become progressively wider as the inspiration becomes deeper and more forceful so that in very deep inspiration they may come to lie almost flush against the lateral walls of the thyroid cartilage, as the result of which the chink of the glottis becomes almost the same size as the lumen of the trachea, presenting no obstruction to the passage of inspired air, thereby permitting the very rapid inhalation of large quantities of air, as is necessary for example in singing or forced respiration during extremes of physical exertion as in running."

Since then, the respiratory movements of the vocal cords have continued to be the focus of interest. Subsequent studies confirmed the

observations of Pressman (1942) (Dedo and Ogura, 1965; Murakami and Kirchner, 1972; Bartlett et al, 1973; England and Bartlett, 1982; England et al, 1982a; 1982b; Brancatisano et al, 1983, 1985; Bartlett and Knuth, 1984). According to Negus (1949), the widening of the glottis precedes a contraction of the diaphragm, whereas glottic narrowing heralds the onset of expiration. Nakamura et al (1958) also found that vocal cord abduction precedes any activity in other respiratory muscles.

1.32 Respiratory Activity of the Intrinsic Laryngeal Muscles

In summary, the vocal cords exhibit phasic movements during eupnoeic breathing - abduction during inspiration and relative adduction during expiration. What supplies the motive power for these movements?

It is generally agreed that the respiratory motion of the vocal cords is the result of respiratory activity of the intrinsic laryngeal muscles. However, there are conflicting conclusions as to the role of the adductor muscles in the approximation of the vocal cords.

The dorsal cricoarytenoid muscle of the larynx was first named by Fabricius (1600) as "musculus aperiens glottidis" - that is, the muscle opening the glottis. Explorations in laboratory animals have so

far confirmed the idea that the dorsal cricoarytenoid muscle is the abductor muscle (Negus, 1947; Green and Neil, 1955; Nakamura et al, 1958; Dedo, 1970; Last, 1972; Murakami and Kirchner, 1972).

Green and Neil (1955), from their investigations of the electromyographic activity of the intrinsic laryngeal muscles, noted that the dorsal cricoarytenoid muscle was active during inspiration, whereas the lateral cricoarytenoid and thyroarytenoid muscles were active during expiration. Similar findings were demonstrated in human beings by Faaborg-Andersen (1957).

Nakamura et al (1958) also studied the electrical activity of the cricothyroid, thyroarytenoid, lateral cricoarytenoid and interarytenoid muscles and reported that except for the thyroarytenoid muscle, which remained quiet throughout the respiratory cycle, the other muscles showed activity during expiration.

Though the expiratory activity of the adductor muscles has commonly been reported, their role in bringing about the approximation of the vocal cords with consequent narrowing of the glottis has not been agreed upon. Murakami and Kirchner (1972) demonstrated that adduction of the vocal cords during expiration was not caused by activation of the adductor muscles,

but by a gradual fall in tonic activity of the dorsal cricoarytenoid muscle. Bartlett et al (1973) confirmed the finding of Murakami and Kirchner (1972), observing that, of the intrinsic muscles of the larynx studied, only the dorsal cricoarytenoid muscle is phasically active with respiration. Hence, they suggested that the cords are actively abducted during inspiration and they fall passively toward their relatively closed, relaxed position during expiration.

1.33 Vertical Respiratory Movements of the Larynx

In addition to the abovementioned movements of the vocal folds and intrinsic laryngeal muscles, there is also a respiratory excursion of the laryngeal framework as a whole. Gross movements of the entire larynx taking place during "quiet" respiration - down with inspiration and up with expiration - were noted in man by Rosenthal (1882) and Schaefer (1900). Mink (1916) stated that while the motion was almost imperceptible in fully quiet or "resting" respiration, the amplitude of the excursion increased with the depth of inspiration and attained 4 to 6 millimetres (mm) in forced respiration.

Dessy (1938), who was the first to publish graphic recordings of the excursion, observed that with inspiration there was generally a lowering of the larynx and, in over half the cases, an additional

backward displacement. Rosenthal (1882) has attributed the descent of the larynx to the contraction of the sternohyoid and sternothyroid muscles, while both Dessy and Schaefer proposed that lowering of the diaphragm and of the intrathoracic pressure with inspiration would also favor the downward movement. Opinion concerning the prevalence of the respiratory excursion of the larynx has not been unanimous. Mitchinson and Yoffey (1947) found by means of X-ray photography that there was no perceptible laryngeal movement in 14 of 23 subjects during a maximal inspiration. Of the remainder, five showed elevation and four showed descent of the larynx. This "bulk movement" of the larynx, evidence for which is still incomplete, might be due to changes in tension on the trachea as a result of the movements of the diaphragm.

1.34 Glottic Resistance to Airflow

One important question concerning the larynx during respiration relates to its function in controlling the alveolar pressure. As the narrowest part of the laryngeal passage, the rima glottidis is assumed to contribute the major resistance to airflow in the upper airway.

Applying the equivalent of Ohm's Law (electrical resistance = voltage / current) to the glottis, the resistance, R, is evaluated by measuring the pressure

drop, P1-P2, between two points during the time, t, in which a measured volume, V, passes one of the points. The average flow per unit time is then V/t or $\dot{V}$, and R is defined as

$$R = P1-P2/\dot{V}$$

That is to say, the flow resistance is equal to the pressure drop per unit of flow.

Measurements of the resistance across the human larynx (Ferris et al, 1964; Schiratzki, 1965; Spann and Hyatt, 1971) and upper airway (Hyatt and Wilcox, 1961; Blide et al, 1964; Ferris et al, 1964; Schiratzki, 1964, 1965; Spann and Hyatt, 1971) have shown that these structures provide an appreciable fraction of the total respiratory resistance. About 50% of the resistance to air flow during breathing in man resides in the upper respiratory tract (nose, pharynx and larynx) (in : Widdicombe and Davies, 1983). Stransky et al (1973) reported that the laryngeal resistance is about one tenth of total lung resistance in cat.

Ferris et al (1964) observed in quietly breathing human subjects that upper airway resistance during expiration was greater than that during inspiration. Similarly, England et al (1982a) reported significantly higher total respiratory resistance during expiration than during inspiration

in five healthy human subjects.

The greater upper airway resistance during expiration can be attributed partly to changes in glottic aperture. Bartlett et al (1973) demonstrated that there is an inverse relationship between laryngeal resistance and the area of the glottic aperture and suggested that movements of the vocal cords were responsible for the phasic respiratory changes in laryngeal resistance. The glottic width and glottic area during inspiration are greater than that during expiration at the same lung volume (Bartlett et al, 1973; Baier et al, 1977; England et al, 1982a; Brancatisano et al, 1983). The inspiratory abducted position of the vocal cords therefore provides a low resistance pathway for lung inflation. Movement of the cords toward the midline during expiration, provides a substantial increase in airflow resistance.

In human subjects, when ventilation is stimulated by exercise, hypercapnia, hypoxia, or hyperventilation, the expiratory narrowing of the glottic airway is attenuated, in keeping with the requirement for increased airflow (Hyatt and Wilcox, 1961; Baier et al, 1977; England and Bartlett, 1982; England et al, 1982b; Bartlett and Knuth 1984). The same phenomenon occurs in anaesthetized, vagally intact cats during hypercapnia and hypoxia (Bartlett

et al, 1973; Bartlett, 1980; Gautier et al, 1973; Sherrey and Megirian, 1975). The resulting increase in expiratory airflow is also accompanied by a decrease in expiratory duration (Gautier et al 1973). On the other hand, hypocapnia increases expiratory narrowing of the glottis in anaesthetized animals (Campbell et al, 1963; Sherrey and Megirian, 1974; Bartlett 1979).

1.35 Role of the Larynx in Ventilatory Control

The function of the movements of the glottis during respiration has been the subject of speculation by Negus (1949). He offered the following rationale:

a. The glottic constriction promotes intra-pulmonary air mixing.

b. The obstructive action of the glottis retards the filling and emptying of the bronchi. This provides time for the adequate exchange of gases in the lungs.

c. Obstruction to the entry of air during inspiration assists venous return to the heart, dilates the pulmonary capillaries and facilitates the flow of blood through the lungs by lowering interpleural pressure.

Pressman and Keleman (1955) also shared Negus' views that glottic constriction during expiration has a significant effect upon air mixing within the lungs.

However, this was speculative and there was little evidence to support the hypothesis. The respiratory functions of the larynx enumerated by Negus were not considered to be important (Fink, 1975). They do not explain why loss of the glottic constriction as a result of laryngectomy or during endotrachael intubation seems to have no deleterious consequences. (Tonkin and Harrison, 1971; Gilchrist, 1973). Nevertheless, it is widely known that respiration is shallow and somewhat irregular in the case of laryngectomized patients and it is attributed to the lack of resistance or appropriate load to an outflow of air (Nakamura et al, 1958).

The respiratory rhythm and partially open condition of the quiescent larynx are regarded by Fink (1975) "as a working mechanical compromise between two conflicting twin necessities: the need for a quasipermanent open state of the larynx and the frequent need for a closed glottis or larynx; between the imperative metabolic demand for ventilation and the behavioural exigencies of instant protection, phonation or straining effort." (Fink 1975).

Recent studies appear to support the role of the larynx as an important effector organ of ventilatory control. Regarding the usefulness of the respiratory movements of the glottis, there is no doubt that the

effect of widening at the glottic chink at or just before inspiration is a reduction of resistance to an inflow of air. This effect is particularly important as it facilitates effortless inspiration. An analogy can be drawn here from people passing through swing doors. An entry via such doors can only be made effortlessly at the instance when the doors swing away from the subject and not when they come together.

During quiet breathing, expiration is not passive but involves a braking of expiratory flow by the resistance of the upper airway and post-inspiratory contraction of the diaphragm which therefore slows the collapse of the lungs. (Brody, 1954; Petit et al, 1960). The expiratory narrowing of the glottis by increasing expiratory resistance participates in braking expiratory flow and lung emptying (Gautier et al, 1973; Rattenborg, 1961; Remmers and Bartlett, 1977). Gautier et al (1973) reported that the duration of expiration in spontaneous breathing exceeded the time required for the passive respiratory system to collapse from the same end-inspiratory volume back to functional residual capacity (FRC). Remmers and Bartlett (1977) found that changes in upper airway resistance in unanaesthetized cats elicited compensatory changes in the electrical activity of the diaphragm and the dorsal cricoarytenoid muscle. They showed that sudden

opening of a previously prepared remote-controlled tracheostomy at the onset or during an expiration increased the activity of the diaphragm but inhibited that of the dorsal cricoarytenoid muscles. These responses acted to compensate for the loss of expiratory braking by the upper airway. Occlusion of the tracheostomy during expiration produced the opposite responses.

By retarding expiratory flow, the larynx also aids the post-inspiratory activity of the diaphragm to maintain end-expiratory lung volume (Harding, 1980; Henderson-Smart et al, 1982). It is suggested that alterations in the expiratory flow rate may in turn serve, through the action of volume feedback, to regulate the duration of expiration and hence respiratory frequency (since frequency is the inverse of cycle duration which itself is the sum of inspiratory and expiratory times) and also to influence the time at which the next inspiration is initiated (Gautier et al, 1973; Remmers and Bartlett, 1977). The increase in respiratory frequency associated with rapid expiratory flow found by Nattie and Tenney (1970) in human subjects during resistance unloading, supports the existence of such a feedback system. Remmers and Bartlett (1977) found a consistent vagally mediated relationship between the volume of the lung during expiration and the time of

onset of the next inspiration. When expiratory flow was retarded, the initiation of the next breath was found to be delayed. It appears that the rapid expiratory flow brings into action the rapidly adapting receptors which have recently been shown to be involved in the initiation of inspiration (Davies et al, 1981). A review of this group of lung receptors is given in a later section.

1.36 Advantages of Expiratory Braking

It has been suggested that the expiratory glottic narrowing constitutes a metabolically inexpensive aid to the modulation of expiratory airflow and hence to the control of end-expiratory lung volume (Bartlett et al, 1973; Gautier et al, 1973; Remmers and Bartlett, 1977).

Imagine a respiratory system without the larynx and its upper airway. It will have a relatively low resistance and so during passive expiration, the system will develop high flow rates which if unopposed will rapidly return the lungs to FRC. An appropriate ventilation can be achieved either by decreasing V_T or by prolonging t_I. However, the efficiency of breathing will be sacrificed by either of these adjustments (Mead, 1960). On the other hand, the expiratory period can be prolonged either by instituting an end-expiratory pause, or by slowing expiratory flow by the action of braking mechanisms.

The former will minimise the role of volume feedback in initiating inspiration, whereas the latter has no disadvantages as long as it is energetically economical and rapidly reversible. Based on these criteria, post-inspiratory contraction of the diaphragm or other inspiratory muscles may be an imperfect braking mechanism because it probably involves appreciable energy expenditure. By contrast, the larynx seems to be best suited for the job. Flow resistance is high in the relaxed larynx (Bartlett et al, 1973), yet can be reduced rapidly to low levels by the contraction of the dorsal cricoarytenoid muscle to meet different metabolic requirements with relatively little energy cost.

In order that the larynx responds in a manner appropriate to the conditions in which the animal finds itself, it has to depend on information from the lungs which are mediated by pulmonary receptors.

1.4 Pulmonary Receptors and their Effects on Laryngeal Function

A century ago, Claude Bernard pointed out that the body maintains the constancy of the "milieu interieur" by a series of automatic adjustments. The respiratory system plays its part in this homeostatic mechanism by a process of negative feedback (Figure 7). The larynx which has been shown to be a

significant respiratory effector organ, is therefore part of this feedback loop. Complex regulatory mechanisms exist which modify its function corresponding to the phase of respiration and the physiologic status of the lower airways and the lungs.

Though there are a host of afferent stimuli which can influence the larynx, only the three main types of pulmonary receptors will be reviewed here. The function of the larynx is regulated by lung reflexes originating from pulmonary receptors namely, J-receptors with non-myelinated fibres, and the rapidly adapting and pulmonary stretch receptors both of which have myelinated fibres.

1.41 J-Receptors

Paintal (1953) discovered a group of lung receptors with small-diameter vagal nerve fibres, which responded to intravascular injections of various drugs (such as phenyl diguanide) known to cause vagal reflexes. Their properties were quite distinct from those of rapidly adapting and pulmonary stretch receptors. Later he named them as "deflation receptors" since some of them responded to strong deflations (Paintal, 1955; 1957). In 1969, Paintal renamed them juxtapulmonary capillary receptors, or J-receptors for short, because they seemed to be localized close to the alveolar capillaries and to respond primarily to increased interstitial fluid

outside the capillaries. Extensive research by Paintal (1973a) has documented the behavior of alveolar J-receptors. They are normally silent in eupnoea in the animal and therefore play little part in the normal control of breathing. However, they seem to have a tonic discharge in diseased lungs such as those with pneumonia (Frankstein and Sergeeva, 1966; Trenchard et al, 1972), and to modify the pattern of breathing in these conditions.

Besides being stimulated by pulmonary congestion due to back-pressure from occlusion of the aorta or left heart, J-receptors also respond to conditions such as pulmonary microembolism and inhalation of irritant gases such as ammonia, volatile anaesthetics and poisonous gases. Their reflex actions include apnoea or rapid shallow breathing, hypotension and bradycardia and inhibition of spinal reflexes (Paintal, 1973). The patterns of response of J-receptors to such a wide variety of interventions are consistent with the concept that they are nociceptive endings which are primarily activated by tissue damage, by the accumulation of interstitial fluid and also by released mediators. J-receptors have been much studied by the use of exogenous stimulants such as phenyl diguanide and capsaicin (Paintal, 1973a).

The reflex action of stimulation of alveolar J-

receptors on laryngeal resistance to airflow and on laryngeal motoneurone discharge in cats has been investigated by Stransky et al (1973). Intravenous injections of phenyl diguanide brought about a striking increase in laryngeal resistance due to laryngeal constriction and even laryngeal closure during the reflex apnoea. Recurrent laryngeal motoneurones discharging during expiration were strongly stimulated. Glogowska et al (1974) also reported that induction of pulmonary oedema by intravenous injection of 0.1 ml capric and caprillic acids dissolved in olive oil increased the discharge of expiratory fibres of the recurrent laryngeal nerve but decreased the discharge of inspiratory fibres.

McCaffrey and Kern (1980) reported that the injection of capsaicin into the right atrium produced apnoea and a large increase in laryngeal resistance. Capsaicin produced this reflex when injected into the pulmonary circulation, but not when injected into the systemic circulation. The response of laryngeal resistance to capsaicin was abolished by dividing the vagus nerves below the origin of the recurrent laryngeal nerves, thus indicating that the above reflex was mediated by afferent vagal fibers arising from the lung.

The physiologic role of J-receptors in regulating laryngeal function is still unknown. The

reflex laryngospasm described by McCaffrey and Kern (1980), is considered not to be a protective reflex because J-receptors cannot be stimulated until the irritant agent reaches the alveoli.

The above studies quoted show that pathological changes in the lungs can change the total resistance to flow by reflex changes in the laryngeal calibre, in addition to actions on the lower airways.

1.42 Rapidly Adapting Receptors

In 1926, Keller and Loeser recorded multifibre activity in the vagus nerves from rapidly adapting intrathoracic receptors and concluded that they might be coming from airway receptors responsible for coughing.

Knowlton and Larrabee (1946) extended this work with single-fibre recordings and named those receptors which responded to maintained inflation and deflation of the lungs with a rapidly adapting irregular discharge as rapidly adapting stretch receptors.

Since then, other names have been used in the literature. Deflation or collapse receptors is the name preferred by Koller (1973) and is based on the fact that the receptors respond to lung collapse and play an important role in reflex responses to atelectasis and pneumothorax. Irritant receptors is

another name used because many irritant gases and aerosols stimulate some of the receptors but their responses are not always consistent and other receptors are also stimulated by irritants (Coleridge and Coleridge, 1977a,b; Paintal, 1973a; Sampson and Vidruk, 1975).

At present, there is still no resolution on the naming of these receptors. The term "rapidly adapting receptors" will however be used in this thesis as it is considered more appropriate in view of their responses to various stimuli.

Histological studies with light microscopy (Fillenz and Widdicombe, 1971) suggested that rapidly adapting receptors (RAR) lay in the epithelium of the trachea and larger bronchi. Other investigations have shown that the receptors could be found in intrapulmonary tissues as well as in the walls of the extrapulmonary airways (Homberger, 1968; Ferrer and Koller, 1968; Mills et al, 1969; Mortola et al, 1975; Sampson and Vidruk, 1975).

Recording from single vagal nerve fibres from airway RAR has been extensively used as a method of studying their properties. The physiological properties of extrapulmonary and intrapulmonary RAR are identical (Paintal, 1973a). In the cat, RAR are silent during eupnoea (Knowlton and Larrabee, 1946;

Widdicombe, 1954a) or even during a moderate increase in tidal volume. In the rabbit on the other hand, RAR show activity during eupnoeic respiration and also display a brief burst near the peak of inspiration (Sellick and Widdicombe, 1970). It is proposed that RAR probably exert an appreciable effect on the pattern of breathing, initiating inspiration (Davies et al, 1981) (Figure 8) and shortening inspiratory and expiratory durations (Davies et al, 1978a, 1984; Davies and Roumy, 1982). The spontaneous activity recorded from RAR fibres increases when activated by the following three groups of stimuli :

a) Inhalation of chemical irritants such as ethyl ether, ammonia vapour and cigarette smoke and mechanical irritants such as "inert" carbon dust and the passage of an endobronchial catheter (Sellick and Widdicombe, 1971; Widdicombe, 1974);

b) Contraction of smooth muscle of the airways by bronchoconstrictor drugs such as histamine (Mills et al, 1969; Sellick and Widdicombe, 1971);

c) Mechanical changes in the lungs which lead to a greater pull on the airways and distortion of the wall of the airway during which a rapidly adapting irregular discharge is obtained if the volume change is maintained. Those in the

> trachea usually show an "off effect" when the volume change is released (Widdicombe, 1954a). Davies and Roumy (1982) recorded highest RAR activity by deflation at FRC and at peak tidal volume.

The sensitivity of the receptors to inflation and deflation is enhanced by a decrease in lung compliance (Sellick and Widdicombe, 1970) which is attributed to a greater mechanical pull on the airways.

Stimulation of RAR has been shown to cause hyperpnoea and bronchoconstriction (Mills et al, 1970; Fillenz and Widdicombe, 1971). The receptor discharge increases in hyperpnoea due to hypercapnia or hypoxia or both (Sellick and Widdicombe, 1969).

It has been postulated that RAR are responsible for the deep augmented breaths which normally punctuate breathing and reverse the continuous tendency to collapse the lungs (Davies and Roumy, 1982). Studies on reflexes suggest that this view is compatible with the indications that RAR can also cause tachypnea, primarily by shortening expiratory duration (Davies, 1976; Davies and Roumy, 1977; Davies et al, 1978a,b).

Experiments which eliminate the activity of

pulmonary stretch receptors indicate that RAR exert an important influence on pattern of breathing (Davies, 1976; Davies et al, 1978a).

A further reflex response to the activity of RAR is laryngeal constriction. Stimulation of the receptors in cats and rabbits, by intravenous injections of histamine acid phosphate or inhalation of an aerosol of the drug in solution, was found to augment discharge in expiratory adductor motoneurones to the larynx and increase the resistance of the larynx to airflow in the expiratory phase (Stransky et al, 1973). Laryngeal resistance in inspiration was usually increased. Histamine also increased frequency of discharge of inspiratory fibres but reduced the number of impulses per inspiratory phase. The laryngeal responses to histamine were more than halved by bilateral intrathoracic vagotomy. The laryngeal reflex responses correlated in time with changes in lung mechanics and the drug-induced decrease in compliance is believed to be the primary stimulus to the receptors (Sellick and Widdicombe, 1970).

Although the above laryngeal reflexes are activated by administration of histamine which stimulates RAR, the same reflexes are evoked when they are stimulated in physiological or pathological conditions (Paintal, 1970; Mills et al, 1970). In this respect, anaphylaxis in rabbits excites RAR

(Mills et al, 1969) and brings about an increase in discharge of laryngeal motoneurones both in the inspiratory and expiratory phases (Szereda-Przestaszewska, 1971). Further investigations by McCaffrey and Kern (1980) however, showed that RAR stimulation with histamine hydrochloride in dogs produced a reduction in inspiratory laryngeal resistance. This discrepancy could be due to differences in species or experimental conditions.

Pneumothorax is found to increase expiratory laryngeal resistance but decrease inspiratory resistance (Dixon et al, 1974). Further studies by Glogowska et al (1974) on the underlying motoneuronal changes which control laryngeal calibre showed that pneumothorax increased the frequency of discharge of both inspiratory-phased and expiratory-phased laryngeal fibres; the inspiratory response being greatly reduced by bilateral vagotomy below the origin of the recurrent laryngeal nerves. From the above two studies, it appears that both variables were unequivocally increased by pneumothorax. However, with inspiratory motor fibres, no such consistency exists.

1.43 Pulmonary Stretch Receptors

In 1868, Breuer and Hering found that artificial inflation of the lungs during inspiration terminated

inspiration whereas deflation terminated expiration and initiated inspiration. However, this effect was abolished by vagotomy following which breathing became slower and deeper. Hence, Breuer and Hering proposed that the receptors which detected the state of lung distension are found in the lung tissue and the vagi are the afferent pathway to the respiratory centre (the term "centre" is used in the context of a common function rather than a common location since no true centres have ever been identified anatomically). They suggested that the respiratory centre which produced the normal respiratory pattern required vagal afferent information about the state of the lung. This reflex has since been named as the Hering-Breuer reflex.

Adrian (1933) showed that when a sudden and maintained inflation was applied to the lungs, a slowly adapting vagal fibre discharge was obtained. Hence, the receptors of this fibre became known as the "slowly adapting pulmonary stretch receptors". The abbreviation "pulmonary stretch receptors" (PSR) (Knowlton and Larrabee, 1946) will be used throughout this thesis.

Physiological and degeneration experiments together with histological evidence suggest that many of the PSR are located in the smooth muscle of the larger airways (Widdicombe 1954b). For both cat (Sant'Ambrogio and Miserocchi, 1973) and dog,

(Miserocchi et al, 1973) it had been shown that the receptors are concentrated in the trachea and larger bronchi. For the rabbit, the receptors also have a higher concentration in the extrapulmonary airways (Richardson et al, 1973).

Electrophysiological studies of the action potential discharge in vagal afferent fibres have shown that PSR are stimulated by distension of the lungs and airways. Most receptors have thresholds within the eupnoeic tidal volume range so that lung inflation during quiet breathing increases their discharge. There is considerable variation between receptors in threshold and rate of adaptation; this may depend on the site of the endings, those in the larger airways tending to have a lower threshold and slower adaptation.

The strength of the Hering-Breuer inflation reflex varies considerably between animals, being strongest in rabbits and weakest in man (Widdicombe 1961).

Pulmonary stretch receptors adjust the pattern of breathing and airway calibre in order to minimise respiratory work or inspiratory muscle force in response to changes in the mechanical conditions of the lungs.

The main influence of PSR during spontaneous eupnoeic breathing is to shorten the duration of inspiration and to lengthen the duration of expiration (Clark and von Euler, 1972). Vagal impulse activity from PSR increases with the onset of inspiration and this activity rises until it reaches the central threshold which decreases with time. Once this threshold is reached, an inspiratory off-switch is triggered, thus terminating inspiration (von Euler, 1977). During the early stage of expiration while the lungs are deflating, these receptors are still strongly active, eventually dying down as the lungs empty (Clark and von Euler, 1972).

The way in which the Hering-Breuer inflation reflex adjusts the pattern of breathing has been studied by von Euler et al (1970). During stimulation of breathing by hypercapnia, the increase in tidal volume was accompanied by a decrease in the duration of inspiration. Presumably, the duration of inspiration is reduced because with the more vigorous inspiratory drive and therefore more rapid build-up of tidal volume the increased discharge of PSR cuts short inspiration earlier. In addition, Clark and von Euler (1972) found that there is also a direct relationship between the duration of inspiration and the duration of expiration in hypercapnic hyperpnoea, so that the frequency of breathing would be further increased by a

shortening of expiratory as well as inspiratory time.

The physiological importance of the inflation reflex is believed to be that in situations in which an increased central respiratory drive increases the rate of build-up of tidal volume, it produces an increase in respiratory frequency together with an increase in tidal volume, thereby magnifying the increase in minute ventilation (in : Keele et al, 1984). Moreover, since PSR are able to "sense" the mechanical conditions of the lungs, they contribute to the establishment of an optimal combination of respiratory frequency and tidal volume for any given ventilation, thereby minimizing the work of the respiratory muscles.

The sensitivity of PSR can be inhibited by an increase in inhaled CO_2 (Coleridge et al, 1978). However, the receptors are insensitive to blood-borne changes in pCO_2 (Bartlett and Sant'Ambrogio, 1976; Bradley et al, 1976). The action of CO_2 on PSR is thought to be direct or at least not secondary to a change in airway smooth muscle tone (Coleridge et al, 1978). Sulphur dioxide has also been shown to inhibit or paralyse PSR in the rabbit (Davies, 1976; Davies et al, 1978a,b, 1981; Citterio and Agostoni, 1983; Davenport et al, 1984; Agostoni et al, 1985; Citterio et al, 1985).

The effect of pulmonary stretch receptor stimulation on the activity of the recurrent laryngeal nerve and the intrinsic laryngeal muscles during the classic Hering-Breuer inflation reflex is consistent with the action on other respiratory muscles.

Several studies have demonstrated that during lung inflation, some inspiratory laryngeal fibres stop their rhythmic firing (Green and Neil, 1955; Eyzaguirre and Taylor, 1963; Barillot and Bianchi, 1971; Barillot and Dussardier, 1973) whereas others display a low-frequency, long lasting discharge (Barillot and Bianchi, 1971; Barillot and Dussardier, 1973).

However, in expiratory laryngeal fibres, lung inflation brought about an increase in discharge in some fibres (Green and Neil, 1955; Barillot and Dussardier, 1976), whereas in others it inhibited impulse activity (Barillot and Bianchi, 1971), or reduced the rate of firing (Barillot and Dussardier, 1976). Eyzaguirre and Taylor (1963) however, reported that lung inflation did not affect the discharge of such fibres.

The above electroneurographic findings can be correlated with the results of electromyographic studies of intrinsic laryngeal muscles. Lung inflation inhibits the activity of the inspiratory

muscles of the larynx namely, the dorsal cricoarytenoid muscles (Green and Neil, 1955; Nakamura et al, 1958; Murakami and Kirchner, 1972; Fukuda et al, 1973, Sherrey and Megirian, 1975). Sherrey and Megirian (1975) further reported that while the phasic activity of the dorsal cricoaryteniod muscles (DCA) was abolished, a tonic activity continued throughout the period of inflation.

On the other hand, studies on cats have shown that the lateral cricoarytenoid and thyroarytenoid muscles, which are the adductor laryngeal muscles, are reflexly excited by lung inflation (Green and Neil, 1955; Barillot and Bianchi, 1971). However, some investigations show that there is no increase in the activity of the adductor muscles of cats during lung inflation (Murakami and Kirchner, 1972; Sherrey and Megirian, 1975).

The respiratory activity of the muscular processes of arytenoid cartilage during lung inflation has been extensively studied since they are considered a good indicator of vocal cord motion. Murakami and Kirchner (1972) extended these studies on cats by directly observing the vocal cord motion under a surgical microscope with a 16 millimetres movie camera. They reported that when the Hering-Breuer reflex was elicited by pulmonary inflation with 100 cc. of room air, the vocal cord remained in the same

position as it would be at the end of expiration regardless of the respiratory cycle and never adducted beyond this position. However, during inflation with 150 cc. of room air, it moved to an over-abducted position and then to the paramedian position just after inflation. This paramedian position was not seen under any other conditions. Maximal inflation with 200 cc. or more of room air caused cough reflexes so that the vocal cord moved rapidly but irregularly between adduction and abduction.

McCaffrey and Kern (1980) carried the above studies a step further by looking at how the change in laryngeal calibre evoked by lung inflation affects laryngeal resistance in dogs. They found that the stimulation of PSR by lung inflation eliminates phasic inspiratory activity of laryngeal abductors. This has the effect of inhibiting the phasic reduction of laryngeal resistance during inspiration which corresponds with the observed inhibition of activity of inspiratory laryngeal fibres. Sustained lung inflation produces a tonic decrease in laryngeal resistance which is related to the degree of lung inflation. They explained that the effect of a decrease in laryngeal resistance with large lung inflation would facilitate lung emptying, whereas with small inflation the higher laryngeal resistance would slow lung emptying. In this way, the lung would tend

to reach a constant functional residual capacity during expiration for both large and small inflations (McCaffrey and Kern, 1980).

The response of the intrinsic laryngeal muscles and recurrent laryngeal nerve to lung inflation can be regarded as a counterpart of the Hering-Breuer inhibito-inspiratory reflex which inhibits inspiratory-phased respiratory muscles. This reflex is thought to arise from some intrapulmonary receptors and not to be merely a mechanical effect of lung inflation per se, since it is effectively abolished by bilateral vagotomy below the origin of the recurrent laryngeal nerves.

1.5 The Present Study

As reviewed above, the intrinsic laryngeal muscles contract with phasic respiratory rhythm in response to the underlying motoneuronal changes in the RLN. The recurrent laryngeal motoneurones may be regarded as one of the outputs of the respiratory complex of the brainstem.

The activities of the laryngeal muscles and motoneurones have been shown to be complexly modified by various afferent stimuli of extramedullary origin, chiefly those mediated by the vagus nerves. The effect of the Hering-Breuer inflation reflex on recurrent laryngeal motoneurone discharge has been

extensively studied but there are a number of contradictory results in these studies. It is generally accepted that the afferent pathway of this reflex inhibition on RLN is indeed vagal and it is presumed that the pulmonary stretch receptors mediate this reflex (Green and Neil, 1955; Eyzaguirre and Taylor, 1963). However, very few studies have been carried out to ascertain the extent to which PSR and other types of lung receptors are functionally involved.

Information on whether the operation of the volume-related feedback system is involved in modulating recurrent laryngeal motoneurone discharge during eupnoeic breathing has not been forthcoming. Neither are there any studies to show if the volume-related input acts through the respiratory centre to modulate laryngeal motoneurone discharge.

The role of other pulmonary afferents, in particular of RAR has received even less attention in research investigations. Moreover, investigations of RAR suffer from two main defects: most of the stimuli used affect more than one group of lung receptors and, in addition to this, as soon as the respiratory and laryngeal responses start they may be modified by activity from PSR. Therefore it is difficult to be sure of the primary reflex action of RAR on recurrent

laryngeal motoneurone discharge.

This study therefore makes use of a method whereby the activity of PSR can be blocked selectively and transiently by sulphur dioxide in the rabbit (Callanan et al, 1975; Davies, 1976; Davies et al, 1978a). Abolition of the Hering-Breuer reflex is used as an index of stretch receptor block. With this preparation, it is possible to stimulate RAR without appreciable modification of the response by PSR. The specificity of this differential vagal block is considered greater than that of other methods such as cold or DC current applied to the vagus (Davies et al, 1978a).

1.51 Aim of the Study

The current study was prompted by the gaps in knowledge discussed on the preceding pages. The aim of the study was to classify the discharge patterns of recurrent laryngeal motoneurones with reference to the inspiratory and expiratory phases of the respiratory cycle, and to stimulate the pulmonary stretch receptors and rapidly adapting receptors by lung inflation and deflation before and during stretch receptor block with sulphur dioxide in order to determine the role of the two groups of lung receptors in modulating recurrent laryngeal motoneurone discharge. The time of onset of recurrent laryngeal and phrenic nerve activity (recorded simultaneously)

was also examined in relation to changes in ventilating volumes in pentobarbital anaesthetized, paralysed rabbits before and during PSR block.

In addition, discharge patterns of the different types of recurrent laryngeal motoneurones as well as their responses to changes of pulmonary afferent inputs were interpreted in relation to the function of regulating airway resistance and hypotheses were formulated about the roles of PSR and RAR in modulating recurrent laryngeal motoneurone discharge.

Chapter Two : Materials and Methods

Chapter Two : Materials and Methods

The rabbit was chosen for this study of the recurrent laryngeal nerve because it has strong lung reflexes. Moreover, it was intended from the beginning to make use of the established method developed by Davies et al (1978a) whereby the activity of pulmonary stretch receptors in rabbit could be reversibly blocked with sulphur dioxide. Furthermore, the rabbit was easily available and relatively easy to handle.

Experiments were performed on fourteen adult New Zealand white rabbits of either sex weighing between 2.3-4.5 kg. They were obtained from the stock maintained by the University. Prior to the experiments, the rabbits had free access to food and water.

The main series of experiments involved nine rabbits on which observations were made while the animals were breathing spontaneously through a tracheal cannula. In another series of experiments involving five rabbits, observations were made during neuromuscular junction block (with 60 mg gallamine triethiodide) and artificial ventilation.

2.1 Surgical Preparation

2.11 Anaesthesia

Anaesthesia was induced with 40 mg/kg of body

weight sodium pentobarbitone (Nembutal, Abbott) injected into the marginal ear vein. The first 2/3 of the dose was injected rapidly for speedy induction and the remainder was given slowly while carefully observing the rabbits' breathing pattern for indications of any impending respiratory arrest. Supplementary doses of sodium pentobarbitone mixed with equal volume of physiological saline, were administered through the venous catheter as and when needed to maintain anaesthesia. The depth of anaesthesia was assessed by respiratory rate which was maintained at approximately 60 breaths per minute. Care was taken to maintain the rabbits only lightly anaesthetized and to allow for 5 minutes interval after supplementary injection of anaesthetics before any recordings were made.

2.12 Femoral Vein Catheterisation

The rabbit was placed in a supine position. Fur at the neck and inguinal regions was clipped. 2-3 ml of xylocaine was infused under the skin of the inguinal region on which a skin incision was made. The sartorius muscle was exposed. Beneath the caudal margin of this muscle was found the femoral vein. 1-2 cm of the vein was cleared from the surrounding tissues and catheterised. A saline-filled polyethylene catheter was inserted several centimetres and tied into the blood vessel for the injection of

anaesthetic. A cotton swab soaked in saline was used to cover the wound.

2.13 Tracheal Cannulation

A skin incision of about 8-9 cm long was made along the midline of the neck region. The sterno-hyoid was exposed and divided to expose the trachea. A small incision was made between the C rings of the trachea thus enabling a polyethylene cannula (3.5 mm internal diameter) to be inserted into. At the pressures used to inflate the lungs in the experiments, this formed an air-tight seal around the cannula without the need for tying.

2.14 Exposure of Recurrent Laryngeal and Phrenic Nerves

2.141 Recurrent Laryngeal Nerve Isolation

The sternomastoid and cleidomastoid muscles were pushed aside and the right recurrent laryngeal nerve was identified as a small nerve coursing along the tracheal-oesophageal groove.

After localizing the nerve, further surgery was carried out under a dissecting microscope. The nerve was cut just below the larynx. It was gently freed for 2-3 cm by lifting the central end with fine forceps and clearing the tissue underneath with fine optical scissors. The nerve was disturbed as little as possible. It was placed in a paraffin filled copper

tray which was positioned in such a way that the nerve was not stretched. The perineurium was carefully removed after which the nerve was placed on a pair of platinum electrodes. In the main series of experiments, the nerve was further teased out to obtain single fibres before any recording, whereas in the second series of experiments, the whole nerve was used for recording.

To improve the signal to noise ratio, the platinum electrodes were flamed before use. Throughout the whole experiment, care was taken to ensure that the nerve was covered with oil at all times to prevent it from drying out. Any blood which accummulated on the nerve was cleared when necessary using saline soaked filter paper or cotton buds to maintain a high signal to noise ratio. Fine adjustments of the position of the bipolar platinum electrodes were made using micromanipulators (PRIOR).

2.142 Phrenic Nerve Isolation

In the second series of experiments, recordings from the left phrenic and the right recurrent laryngeal nerves were obtained simultaneously. The left phrenic nerve was dissected free first according to the following procedures:

The sternomastoid and cleidomastoid muscles were identified and loops of thread tied round them to cut

off their blood supplies. They were crushed at the tied ends before cutting to prevent excessive bleeding. The left phrenic nerve was identified as a small nerve arising from the spinal cord at segments C3, 4 and 5. It lies close and parallel to the spinal cord before it angles back towards the mid-line at the level of the shoulder.

Further surgery was carried out under a dissecting microscope. The most caudal end of the nerve was cut, care being taken not to cause any damage to the nerve. The diaphragm twitched on cutting the phrenic nerve, thus confirming the identification of the nerve. The nerve was gently freed for 2-3 cm by lifting the caudal end with fine forceps and clearing the tissue beneath with fine optical scissors.

A loop of silk thread was placed around the nerve. Cotton swabs in saline were used to cover it while the dissection of recurrent laryngeal nerve was carried out, after which the central ends of both nerves were placed on bipolar platinum electrodes and immersed in a pool of liquid paraffin oil. To obtain a high signal to noise ratio, those procedures described earlier were also followed.

2.2 Recording Procedures

2.21 Respiration Recordings

To measure airflow, the tracheal cannula was attached to a Fleisch pneumotachograph head. In the first series of experiments, flow was recorded on magnetic tape. In the second series of experiments, flow was integrated electronically to give tidal volume. This was displayed directly onto a Brush-Gould pen recorder which was calibrated at the end of each experiment. "Integrator drift" was minimised manually. Residual drift was reset as required. End-tidal CO_2 and O_2 were continuously monitored by an infra-red gas analyser (Datex, Normocap). This was carried out by inserting a wide bore needle of the sampling tube into the tracheal cannula. The rate of sampling was 150 ml/min. End-tidal CO_2 and O_2 measurements were used to evaluate the effect of added dead space and also as an aid to set the level of artificial ventilation in paralysed animals.

2.22 Neural Recordings

The electrical activity of the nerves was recorded using a Neurolog Recording System. This consisted of an AC pre-amplifier, AC amplifier, filter and an audio amplifier connected in series. The AC pre-amplifier provided a high input impedance (200 Kohms). The pre-amplifier had a differential input which was balanced before each experiment to give an

optimum common mode rejection ratio (CMRR). The pre-amplifier output was then fed through a band pass filter to reduce noise artefacts. The action potentials were then amplified by an AC amplifier. The electrical signal was also converted to an audible signal via an audio amplifier. At the same time, the signal was displayed on a four-channel Tektronix oscilloscope to allow constant monitoring of nerve activity. In the first series of experiments where single fibre preparations were used, the signal was further fed into a circuit comprising a spike trigger which was connected in sequence to a digital width unit. The spike trigger converted the delivered pulses into a sequence of "Transistor-Transistor-Logic" (TTL) spikes. The digital width unit was required as the TTL pulses produced by the spike trigger were not of sufficient duration to trigger the TTL spikes. The interval was set so that it was less than the time between action potentials. Records of TTL spikes together with flow were stored on magnetic tape using a two-channel tape recorder (Epsylon Labcorder) for subsequent analysis. For the second series of experiments, records of nerve activity were displayed simultaneously with tidal volume directly onto a pen recorder.

2.3 Inflation and Deflation Tests

Two levels of pressure were used namely 10 cm

H_2O or 20 cm H_2O. Inflation and deflation of the lungs were carried out by connecting the free end of the pneumotachograph to a large drum which was maintained at positive or negative pressure by means of solenoid-operated valves. The valves were triggered manually.

2.4 Stretch Receptor Block

Pulmonary stretch receptor (PSR) block was achieved by causing the rabbit to breathe 200 to 300 parts per million sulphur dioxide for 20 to 30 minutes (Callanan et al, 1975; Davies et al, 1978a).

Sixty millilitres of pure sulphur dioxide was drawn from a cylinder and injected into a stream of air which filled a polyethylene Douglas bag. The bag was then shaken to ensure even distribution of sulphur dioxide in the gas mixture. The concentration of the gas was measured using a colorimetric gas sampling system (Kitagawa 400). Fresh gas mixture was prepared for each experiment.

For administration of SO_2, a stream of the gas mixture was drawn across the rabbit's trachea by a suction pump, adjusted so that the pressure at the tracheal cannula was atmospheric and the pressure in the Douglas bag slightly positive. The gas mixture was not administered through the pneumotachograph as SO_2 is corrosive. Pulmonary stretch receptor activity was assessed by testing the strength of the Hering-Breuer

inflation reflex to 10 cm H_2O positive pressure lung inflation. It was measured by the inhibitory ratio which is the ratio of the expiratory time during lung inflation to control expiratory time (Davies et al 1981). Sulphur dioxide was administered until the reflex was abolished as indicated by an inhibitory ratio less than or equal to 1.0. The degree of pulmonary stretch receptor block was assessed for each rabbit.

2.5 Experimental Protocol

For each experiment, the rabbit was prepared as described earlier. The experimental procedures for the two series of experiments can best be described under the following headings:

2.51 Spontaneously Breathing Rabbits

The experimental set-up and protocol are given in Figures 9 and 10. Respiratory flow and the impulse activity in single fibre of the recurrent laryngeal nerve during eupnoeic respiration were recorded over three breaths before, during and after the application of positive or negative pressure of 20 cm H_2O or 10 cm H_2O to the rabbit's lungs. Each run consisted of a positive and negative pressure application. At least two minutes elapsed between each pressure application. Two runs were carried out. The pneumotachograph was disconnected from the solenoid valve between each

treatment to reduce dead space. It was reconnected one or two breaths before the control breaths were taken. In some experiments, the effect of an increase in the inspired concentration of CO_2 on laryngeal motoneurone activity was studied by adding a dead space in the form of rubber tubing of volume 25 ml to the free end of the pneumotachograph head. Once the end-tidal CO_2 and O_2 had stabilized, the motoneurone activity and airflow were recorded during three breaths.

At the end of the runs with stretch receptor intact, the Hering-Breuer inflation reflex to 10 cm H_2O positive lung inflation was tested and the inhibitory ratio calculated.

Pulmonary stretch receptors were blocked by causing the animal to breathe 200 to 300 ppm SO_2 for 20 to 30 minutes. The administration of the gas was carried out according to the procedures described previously.

After sulphur dioxide exposure, the Hering-Breuer inflation reflex was again tested. If the inhibitory ratio was less or equal to 1.0, the above protocol for PSR intact animal was repeated for two runs in the same animal.

In two rabbits, the effect of adding a dead space of volume 25 ml on motoneurone activity was

examined again during PSR block in the same animal.

2.52 Neuromuscular Junction Block Series

The experimental set-up and protocol are shown in Figures 11 and 12. Recurrent laryngeal and phrenic nerves activities and tidal volume were recorded simultaneously before neuromuscular junction block and end-tidal CO_2 and O_2 levels were noted. The animal was paralysed with 60 mg of gallamine triethiodide (Flaxedil : May and Baker) injected through the venous catheter and washed in with physiological saline. The animal was then artificially ventilated at the same frequency and tidal volume as its spontaneous breathing. This kept the end-tidal CO_2 and O_2 close to the spontaneous breathing level. In paralysed animal, supplementary doses of anaesthetic were administered at the same rate as before paralysis. The relationship between tidal volume and recurrent laryngeal and phrenic nerves activities were again recorded over ten ventilatory cycles. Tidal volumes were changed and set at 50% less than eupnoeic level and subsequently at 100% greater than spontaneous eupnoeic value. The new relationship between tidal volume and recurrent laryngeal and phrenic nerve activities was recorded for ten ventilatory cycles. Tidal volume was returned to its original value and the Hering-Breuer inflation reflex tested by connecting the free end of the

pneumotachograph head to a positive pressure of 10 cm H_2O. Lung stretch receptors were blocked by causing the animals to breathe 200-300 ppm SO_2. Complete abolition of the Hering-Breuer inflation reflex was taken as evidence of stretch receptor block. The relationship between tidal volume at eupnoeic level and recurrent laryngeal and phrenic activities was recorded. The rabbits were ventilated again at tidal volumes which were 50% less than eupnoeic level and subsequently at 100% greater than eupnoeic value. The new relationship between lung volume and the above nerve activity was again recorded.

2.6 Validation of Study

2.61 Histological Examination of the Recurrent Laryngeal Nerve

In this study, specimens of the recurrent laryngeal nerves were collected at the following levels:

a) at the level of the clavicle as the nerve ascends between the trachea and oesophagus,

b) near its entry into the larynx to supply the intrinsic laryngeal muscles.

These samples were placed on cardboard frames in a lightly stretched condition and fixed in Bouin's fluid. The nerves were routinely embedded in paraffin and sectioned at 6 u. The sections were stained using Trichrome Blue by the Modified Masson's Method

(Birtles, personal communication).

2.62 Tests of Recording Equipment

To ascertain if integrated recurrent laryngeal signals stored on magnetic tape can be accurately retrieved without being affected by the inherent noise of the tape recorder, recordings of electrical signals from Neurolog's Pulse Generator or Function Generator were replayed from the tape recorder and compared with electrical signals recorded directly onto the pen recorder.

2.63 Bilateral Vagotomy

To confirm the identity of the nerve from which electroneurographic recordings were made, the neural activity was compared with the activity after the vagi were cut at positions above the origin of the left and right recurrent laryngeal nerves at the end of an experiment.

2.7 Data Analysis

2.71 Spontaneously Breathing Rabbits

In the first series of experiments, the flow and integrated recurrent laryngeal motoneurone signals were replayed from the tape recorder at half the recording speed i.e. 3.75 cm/sec on to a Brush-Gould pen recorder played at a speed of 5 cm/sec. The above speeds were chosen to provide a satisfactory record from which individual spikes could be counted and the

time intervals between spikes determined.

The duration of inspiration (t_I) and expiration (t_E) were determined from the flow tracings. The integrated electroneurograms of the recurrent laryngeal motoneurones were analysed in terms of their frequency and duration of discharge.

Impulse frequencies were measured over respiratory cycles since the motoneurone discharge had a respiratory rhythm. The two phases of the respiratory cycle were each divided into four equal intervals and the number of TTL spikes within each quartile was counted. The frequency of discharge was determined by dividing the total number of spikes by the phase duration. The onset and the end of firing during each phase were measured, from which the duration of firing was obtained. The instantaneous minimum and maximum frequencies were determined by measuring the shortest and longest time intervals between the spikes respectively. Figure 13 shows how the above measurements were obtained.

For each fibre, the data were grouped into +ve Intact, -ve Intact (positive and negative pressure with stretch receptor intact); +ve Block, -ve Block (positive and negative pressure during stretch receptor block). Within the +ve Intact or +ve Block group were data obtained during the pre-inflation

breaths, inflation and post-inflation breaths. Within the -ve Intact or -ve Block group were data obtained during pre-deflation, deflation and post-deflation breaths. Data on augmented breaths were analysed separately. A record of all the data collected for every fibre is given in the Appendix.

Results are given as the mean and the standard error of the mean. A Mann-Whitney test analysis was used to determine the significant difference between control values and the corresponding value during the various experimental manoeuvres with $P < 0.05$ as the level for significance.

2.72 Neuromuscular Junction Block Series

2.721 Analysis of Phase Relationship Between Pump Cycle and Recurrent Laryngeal/Phrenic Bursts

The onset of the recurrent laryngeal/phrenic burst in relation to the pump cycle was considered. The two phases (inflation and deflation) of the pump cycle were each divided into ten equal intervals and the interval in which the recurrent laryngeal/phrenic burst started was identified (for example between 10 and 20% of inflation). In each rabbit five consecutive recurrent laryngeal/phrenic discharges in control conditions (i.e. with the rabbit paralysed, artificially ventilated and no blockade of stretch receptors) and a further five consecutive recurrent

laryngeal/phrenic bursts during SO_2 block with the same pattern of ventilation were considered for each of the three different test tidal volumes.

Chapter Three : Results

Chapter Three : Results

3.1 TESTS ON SPONTANEOUSLY BREATHING RABBITS

The discharge patterns of 24 recurrent laryngeal motoneurones from 9 rabbits were studied in this series of experiments. Apart from 2 fibres which were recorded from the left recurrent laryngeal nerve, all the rest were recorded from the right side. 9 of the single fibres from 6 rabbits were further studied during pulmonary stretch receptor block with sulphur dioxide.

3.11 Motoneurone Discharge Patterns with Pulmonary Stretch Receptor Intact

3.111 Characterization of Spontaneous Laryngeal Motoneurone Discharge

The motor fibres in the recurrent laryngeal nerve showed respiratory rhythmicity and exhibited a variety of discharge patterns. A schematic classification of the discharge patterns is given in Figure 14. The recurrent laryngeal motoneurones were classified according to their phase relation of discharge to the respiratory cycle (Figure 15). The observed patterns were grouped into 2 major categories:

a) "inspiration-related" patterns of discharge, of which there were 2 sub-groups:

i) in which discharge occurred during

inspiration and was completely silent or fired at less than 3 spikes/sec during expiration. Such patterns were labelled "phasic-inspiratory" (P-I) (Figure 16);

ii) in which discharge occurred during both phases of the respiratory cycle but with higher frequency during the inspiratory phase. They were labelled "tonic-inspiratory" (T-I) (Figure 17).

Fibres which possessed pattern P-I or T-I discharge were referred to as phasic-inspiratory laryngeal motoneurones (P-ILMs) or tonic-inspiratory laryngeal motoneurones (T-ILMs) respectively.

b) "expiration-related" patterns of discharge, of which there were 2 sub-groups:

i) in which discharge occurred during expiration and was completely silent or fired at less than 3 spikes/sec during inspiration. Such discharge was designated as "phasic-expiratory" (P-E) (Figure 18);

ii) in which discharge was continuous but occurred at higher frequency during expiration. This last pattern of discharge was designated as "tonic-expiratory" (T-E) (Figure 19).

Fibres which showed Pattern P-E or T-E discharge were referred to as phasic-expiratory laryngeal motoneurones (P-ELMs) or tonic-expiratory laryngeal motoneurones (T-ELMs) respectively.

A breakdown of the number of fibres in each of the above categories is given in Figure 20. Of the 24 fibres studied, 14 discharged with Pattern P-I, 7 with T-I, 1 with P-E and 2 with T-E. Hence 87.5% (n=21/24) of all fibres recorded were ILMs and 12.5% (n=3/24) were ELMs. Only one Pattern P-E type of fibre was observed in this study.

The above discharge patterns occasionally varied with time during eupnoeic respiration. Such variations were noted in 4 P-ILMs which changed into a T-I pattern of discharge and back to P-I at some stages of the experiment (Figure 21). The transition from a phasic pattern of discharge to a phase-spanning type and vice versa also occurred during various experimental interventions, the details of which will be reported in the later part of this chapter.

3.112 Details of Patterns of Discharge

Within the same group of recurrent laryngeal motoneurones, there were variations in the onset of motoneurone activity (Figure 22). In 15 out of 21 ILMs for example, the onset of activity was

synchronous with the onset of inspiration. Another type, referred to as "late expiratory-inspiratory", began their activity at 0.05 - 0.10 seconds earlier than the inspiratory phase. Only 2 P-I motoneurones exhibited this type of activity. A third kind of activity commenced after a delay of 0.05 - 0.40 seconds from the first sign of inspiratory activity. This type of "late-inspiratory" activity was observed in 4 P-ILMs and 2 T-ELMs.

The duration of recurrent laryngeal motoneurone discharge was not equal to inspiratory (t_I) or expiratory duration (t_E). Moreover, the motoneurone did not fire at the same frequency throughout the entire period of its discharge.

The inspiratory discharge of Pattern P-I or T-I fibres was characterized by a peak activity during the first 25% of t_I (first quartile) (mean frequency = 24.1±1.6 and 112.5±10.0 impulses/sec respectively) (Figures 23 and 24). While there was no significant change in the firing rate of P-ILMs during the second and third quartiles of t_I (mean frequency = 18.8±1.4 and 23.2±5.6 impulses/sec respectively), the frequency of discharge of T-ILMs during the same period showed a gradual decline (mean frequency = 89.3±9.2 and 79.5±9.7 impulses/sec respectively). The activity of both groups of fibres showed a sharp decrease during the last 25% of t_I (mean frequency = 8.2±1.0 [P-ILMs]

and 40.9±5.6 [T-ILMs]).

In 2 out of the 14 P-I motoneurones with late expiratory-inspiratory activity, there was a silence of motoneurone discharge during the first 95% of t_E, followed by a gradual increase of activity in late expiration and finally by a sharp increase of activity at the onset of the succeeding inspiratory phase.

The pattern of laryngeal activity of T-I motoneurones during the expiratory phase was less variable than during the inspiratory phase. The activity at the beginning of t_E (first quartile) was at its lowest (mean frequency = 9.1±1.4 impulses/sec) compared with that during the other 3 quartiles. This was followed by a gradual increase in firing frequency before reaching its peak in the last 25% of t_E (mean frequency = 22.2±2.1 impulses/sec).

The firing rate of T-ELMs was more consistent and generally did not change as much throughout the expiratory phase as ILMs (Figure 25). Patterns of T-ELMs activity were different from that of ILMs in that the firing rate was lower at the beginning of the expiratory phase (mean frequency = 26.2±3.0 impulses/sec) followed by an increase in the second quartile of t_E (mean frequency = 32.8±1.4 impulses/sec) and a consistent frequency of discharge throughout the rest of t_E (mean frequency = 33.5±5.3

impulses/sec). There was no significant difference in the frequency of discharge between the four different positions in the inspiratory phase.

The only P-E motoneurone detected in this study showed early expiratory discharge during which activity began synchronously with the end of inspiration, reached its maximum soon after its onset (11.5±1.5 impulses/sec) and then gradually declined towards the baseline level (mean frequency = 0.5±0.5 impulses/sec) in the second and third quartiles (Figure 26).

3.113 Effects of Breathing Through An Added Dead Space on Laryngeal Discharge

Breathing through an added dead space increased the mean frequency of breathing by 33%. The effects of increasing respiratory dead space on P-ILM discharge is shown in Figures 27 and 28. The change in mean frequency of P-ILMs in relation to the respiratory cycle is shown in Figure 29. In 5 P-ILMs tested, breathing through an added dead space increased the mean frequency of discharge by 100% ($P<0.05$) during the inspiratory phase (Table 1). The increase in firing frequency during expiration was not significant. The added dead space also caused 3 of the 5 fibres to fire tonically (T-I pattern) during the expiratory phase but with a lower frequency than in inspiration. The remaining 2 fibres remained silent during the expiratory phase.

Table 1

Effect of breathing through an added dead space on phasic-inspiratory laryngeal motoneurone discharge (N - 5 fibres)

	Inspiratory Discharge			Expiratory Discharge		
	Control	During Added Dead Space	Change (%)	Control	During Added Dead Space	Change (%)
Frequency (Imp/sec)	19.4±2.3@	38.8±7.3	+100*	0.5±0.4	6.7±3.0	+1240
Duration (sec)	0.51±0.07@	0.54±0.08	+5	0.13±0.11	0.78±0.36	+515

@ - values are means and standard errors. Significance of difference from controls: * $P < 0.05$ by Mann-Whitney test.

There was no significant change in the duration of P-ILMs activity during both of the respiratory phases.

As few ELMs were recorded, the effect of increasing respiratory dead space on such motoneurones was not investigated. Neither were observations made of the response of T-ILMs to an added dead space.

3.114 Discharge Patterns of Laryngeal Motoneurones During Hering-Breuer Reflex

3.1141 Responses to Lung Inflation

The degrees of inflation used were

a) 20 cm H_2O positive pressure for 18 fibres (from 6 rabbits);

b) 10 cm H_2O positive pressure for 6 fibres (from 3 rabbits).

Inflation of the lungs with a positive pressure of 20 cm H_2O or 10 cm H_2O produced an average pause of four times (9.13 ± 1.21 sec) the duration of the pre-inflation breath (2.27 ± 0.08 sec). However, in one case, inflation of the lungs with 10 cm H_2O positive pressure produced a pause of only 1.4 times the duration of preinflation breath.

The effects of lung inflation on laryngeal motoneurone discharge (Figures 30 and 31) were analysed as follows:

A. transient effects which were assessed by spike counts within the first 0.2 seconds

of the onset and release of inflation (Figure 32).

B. initial effects which were assessed by spike counts obtained:

i) within one second of the start of inflation apnoea,

ii) within one second during mid-inflation and

iii) during the last one second before the end of the Hering-Breuer reflex.

Based on the above spike counts, the mean frequency of discharge was determined;

C. subsequent effects which were analysed based on discharge frequency during the first three breaths after inflation. The mean frequency of discharge was compared with that before inflation.

3.11411 Transient Effects

The transient responses of laryngeal motoneurones during inflation and after inflation is shown in Table 2. These responses were so variable that statistical analysis does not reach significant values in this study.

In 9 out of 28 instances in 21 ILMs, there was an increase in the frequency of discharge (relative to that in inspiration before lung inflation) during the

Table 2

Transient response of recurrent laryngeal motoneurones to inflation of the lungs

Fibre Type	"On-Effect"			"Off-Effect"		
	+	-	X	+	-	X
ILMs	9	10	9	12	11	5
ELMs	3	1	0	3	1	1

"On-effect" and "Off-Effect": Determined by spike counts within the first 0.2 secs. of the onset and release of lung inflation. Numerical figures indicate the number of instances when phenomenon was observed.

+ : increase in frequency of motoneurone discharge relative to control (pre-inflation).

- : decrease in discharge frequency

X : no response

first 0.2 seconds of lung inflation.

There was no transient response in 9 tests and a decrease in discharge in 10 tests. The positions of the respiratory phase at which the above responses were elicited are also shown in Figures 33 and 34. Inflation applied within the first 50% of t_I elicited an increase in response in 6 out of 10 cases.

Following the release of inflation, 23 of 28 tests on 21 ILMs exhibited transient responses, 12 of which showed an increase in frequency of discharge relative to that during pre-inflation breaths. No response was observed in 5 instances (Figure 35).

Transient responses were also elicited in 4 of 5 tests on 3 ELMs, out of which 3 showed increase in firing rate (relative to that in expiration before inflation) (Figures 36 and 37). No response was elicited in 1 test. A greater response was elicited when lung inflation was applied within the last 10% of the inspiratory phase.

The release of lung inflation also evoked a response in 4 out of 5 tests on the 3 ELMs, of which 3 showed increased firing (Figure 38).

3.11412 Initial Effects

The responses to lung inflation were quantitated by an "inflation test frequency ratio" (ITFR), which was the ratio of mean frequency of discharge during

inflation apnoea to that in the control (pre-inflation) inspiratory phase. The ITFR was then used to classify laryngeal responses into one of four types labelled A to D:

i) Type A: the discharge disappears (or an occasional one to two spikes), a condition which is the equivalent of apnoea in terms of neural discharge;

ii) Type B: the motoneurone shows a low frequency, long lasting discharge throughout the expiratory pause, ITFR < 1.0;

iii) Type C: the discharge disappears during the first one second of inflation apnoea but reappears and fires continuously with an increasing firing frequency, ITFR < 1.0;

iv) Type D: there is an increase in frequency of discharge throughout the expiratory pause, ITFR > 1.0.

The types of response elicited by pulmonary inflation are shown in Figure 39:

a) Type A response in 5 tests on 5 P-ILMs, 1 test on 1 P-ELM;

b) Type B response in 7 tests on 5 P-ILMs (ITFR=0.41$\pm$0.11, P<0.05), 6 tests on 6 T-ILMs (ITFR=0.47$\pm$0.11, P>0.05), 3 tests

on 2 T-ELMs (ITFR=0.62±0.15, P>0.05) (Figures 40a and 40b). In 1 of the 7 tests on P-ILMs and in 4 of the 6 tests on T-ILMs, the motoneurones exhibited a gradual increase in firing frequency with peak activity near the end of the expiratory pause. A similar pattern of discharge was recorded in 3 tests on 2 T-ELMs during lung inflation.

c) Type C response in 6 tests on 4 P-ILMs (ITFR=0.28±0.06, P<0.05) (Figure 40b);

d) Type D response in 3 tests on 3 P-ILMs (ITFR=2.88±1.02, P>0.05), 1 test on 1 T-ILM (ITFR=6.9) (Figure 40c).

The effects of inflation on laryngeal discharge were then classified on the basis of the above response type:

I) "Classical" responses which are responses in the same direction as the Hering-Breuer reflex; i.e. inhibition of ILMs (Type A,B,C response) and excitation of ELMs (Type D response) activity. The inhibitory effects of lung inflation were observed in 24 tests on 20 ILMs and excitatory effects in 3 tests on 2 ELMs.

II) "Paradoxical" responses which are responses

in the opposite direction to the Hering-Breuer reflex:i.e. excitation of ILMs (Type D response) and inhibition of ELMs (Type A,B,C response) activity. The excitatory effects of lung inflation were observed in 4 runs on 4 ILMs and the inhibitory effects in 4 runs on 3 ELMs.

Regardless of the type of responses elicited, it was noted that there was no rhythm in any of the types of discharge during lung inflation. Although different laryngeal motoneurones had similar phase relation to the respiratory cycle, they behaved differently in response to lung inflation.

3.11413 Subsequent Effects

Immediately after lung inflation, a T-I pattern of discharge was elicited in 7 out of 14 runs on 14 P-ILMs. The increase in inspiratory and expiratory discharges of P-ILMs was insignificant (Tables 3 and 4). However, there was a significant increase in the duration of the first inspiratory discharge (mean increase=26%, $P<0.01$).

On the other hand, 3 out of 7 T-ILMs showed a P-I pattern of discharge. Significant changes in the mean frequency and duration of inspiratory activity were noted (-55%, $P<0.05$ and 54%, $P<0.05$

Table 3

Response of recurrent laryngeal motoneurones during the first breath after lung inflation

	Inspiratory Discharge			Expiratory Discharge		
	Pre-inflation	1st breath	Change (%)	Pre-inflation	1st breath	Change (%)
P-ILMs (N=14)						
Frequency (Imp/sec)	17.8 ± 3.5 ◎	21.9 ± 4.7	+ 23	0.6 ± 0.2	3.4 ± 1.7	+499
Duration (sec)	0.49 ± 0.03 ◎	0.62 ± 0.07	+26**	0.08 ± 0.04	0.13 ± 0.05	+62.5
T-ILMs (N=7)						
Frequency (Imp/sec)	87.0 ± 19.6	51.3 ± 15.2	-41	16.8 ± 3.1	17.0 ± 3.3	+1
Duration (sec)	0.51 ± 0.02	1.03 ± 0.10	+102**	1.59 ± 0.11	0.84 ± 0.16	-47**
T-ELMs (N=2)						
Frequency (Imp/sec)	16.5 ± 5.9	45.2 ± 3.8	+174	43.0 ± 9.2	47.0 ± 11.4	+9
Duration (sec)	0.77 ± 0.09	1.32 ± 0.02	+71	1.81 ± 0.11	0.64 ± 0.05	-65

◎ = values are means and standard errors. N refers to the number of fibres studied. Significance of difference of response from pre-inflation values: ** $P < 0.01$.

Table 4

Data on recurrent laryngeal motoneurone activity after lung inflation

Before Inflation

	P-ILMs		T-ILMs		T-ELMs	
	Insp.	Exp.	Insp.	Exp.	Insp.	Exp.
Frequency (Imp/sec)	@ 17.8 ± 3.5	0.6 ±0.2	87.0 ±19.6	16.8 ± 3.1	16.6 ± 5.9	43.0 ± 9.2
Duration (sec)	@ 0.49 ±0.03	0.08 ±0.04	0.51 ±0.02	1.59 ±0.11	0.69 ±0.11	1.76 ±0.10
	N = 14		N = 7		N = 2	

After Inflation

Frequency (Imp/sec)	18.6 ±3.5	3.2 ±1.5	38.8 ±11.9	9.7 ±1.3	38.1 ± 9.1	44.4 ±10.0
Duration (sec)	0.53 ±0.05	0.16 ±0.06	0.79 ±0.06	0.57 ±0.14	1.03 ±0.05	0.69 ±0.10

Change (%)

Frequency	+ 4	+ 433	- 55*	- 42	+ 130	+ 3
Duration	+ 8	+ 100	+ 54*	- 64**	+ 49*	- 60*

@ = values are means and standard errors. N = Number of fibres studied.
Insp. = Inspiratory discharge Exp. = Expiratory discharge
Significance of difference of response after inflation to that before inflation:
* $P < 0.05$; ** $P < 0.01$ by Mann-Whitney test.

respectively). The first inspiratory discharge showed a greater increase in duration of firing (102%, $P<0.01$). The duration of expiratory discharge showed a significant decrease (-64%, $P<0.01$).

Changes in the pattern of firing were observed in P-ELM (N=1 fibre) after lung inflation. The fibre which usually fired only in expiration lost its phasic activity and became tonically active. There was an increase in frequency and duration of inspiratory discharge (909% and 933% respectively). Expiratory activity was shortened (-20%) though there was an increase in firing rate (549%).

As for T-ELMs, the increase in inspiratory and expiratory discharge was insignificant. However, the durations of discharge during inspiration and expiration were significantly changed (49%, $P<0.05$ and -60%, $P<0.05$ respectively).

3.1142 Responses to Lung Deflation

Two levels of deflation were used:

a) 20 cm H_2O negative pressure for 18 fibres (from 6 rabbits),

b) 10 cm H_2O negative pressure for 5 fibres (from 3 rabbits).

The laryngeal response to the effects of lung deflation (Figure 41) was analysed as follows:

a) transient effects which were evaluated by

spike counts within the first 0.2 seconds during and after deflation (Figure 42).

b) initial effects, which were evaluated based on laryngeal discharge i) during the first breath immediately following deflation, ii) during mid-deflation and iii) immediately before the release of deflation;

c) subsequent effects, which were assessed based on the discharge pattern during the first three breaths after the release of deflation. The frequency of inspiratory and expiratory discharge was compared with that before deflation.

3.11421 Transient Effects

The recurrent laryngeal motoneurones showed considerable variation in the size of their transient responses to lung deflation (Table 5), however, statistical analysis did not reach significant values.

In 18 of 24 tests on 20 ILMs, transient responses were elicited following lung deflation, out of which 12 showed increase in frequency of discharge when compared to the inspiratory discharge during pre-deflation breaths. There were no responses in 6 instances. The position of lung deflation and the degree of transient response it elicited are shown in

Table 5

Transient response of recurrent laryngeal motoneurones to deflation of the lungs

Fibre Type	"On- Effect"			" Off-Effect"		
	+	-	x	+	-	x
ILMs	12	6	6	11	9	3
ELMs	3	1	1	0	4	1

"On-Effect" and "Off-Effect": Determined by spikes counts within the first 0.2 secs of the onset and release of lung deflation. Numerical figures indicate the number of instances when phenomenon was observed.

+ : increase in frequency of motoneurone discharge relative to control (pre-deflation)

- : decrease in discharge frequency

x : no response

Figures 33 and 34.

The effect of the release of lung deflation on laryngeal activity was an increase in frequency of discharge in 11 out of the 23 tests on 20 ILMs. There was a decrease in activity in 9 tests and no response in 3 tests (Figure 35).

Transient responses were also elicited in 4 of 5 tests on 3 ELMs following lung deflation, out of which 3 showed increase in discharge frequency relative to the expiratory discharge before lung deflation. The greatest response was elicited when lung deflation was applied within the last 30% of the inspiration phase (Figures 36 and 37).

Release of lung deflation also evoked responses in 4 of 5 tests on 3 ELMs (Figure 38). None of these tests exhibited increase in firing rate. There was no discharge in one test.

3.11422 Initial Effects

Lung deflation stimulated breathing. Inspiratory duration and the frequency of breathing always increased (mean increase = 12% and 89% respectively), whereas expiratory duration always decreased (mean decrease = 76%) (Figure 41). The increase in inspiratory duration was greatest in the first breath during deflation (mean increase in duration = 52%) in 93% of all cases.

Following deflation, there was an increase in P-ILM activity (Tables 6 and 7, Figure 43). Deflation elicited an inspiratory excitatory response (mean increase in frequency = 55%, $P<0.01$) with a lengthening of the first inspiratory burst (mean increase in frequency = 44%, $P<0.01$). However, the increase in firing rate during the first inspiration was lower than the mean increase during deflation (42% vs 55%). The mean frequency of discharge during the expiratory phase also increased (mean increase in frequency=550%, $P<0.05$) (Figure 44). The increase in discharge frequency was greatest in the first expiration following deflation (581%, $P<0.01$ vs 550%, $P<0.05$). The change in duration of P-ILM activity in inspiration and expiration was not significantly different from that before deflation.

In 9 out of 16 tests on 8 P-ILMs, the motoneurones acquired a phase-spanning pattern (T-I) of activity, discharging in the expiratory phase as well. However, the P-ILMs continued to fire at a higher frequency in the inspiratory phase. In the other 7 tests on 5 P-ILMs, the neurones continued to fire only in the inspiratory phase.

For T-ILMs, the frequency and duration of discharge during the inspiratory phase were increased by 61% ($P>0.05$) and 24% ($P<0.01$) respectively (Figures

Table 6

Response of recurrent laryngeal motoneurones during the first breath following lung deflation

	P-ILMs (N-13)		T-ILMs (N-7)		T-ELMs (N-2)	
	Frequency (Imp/sec)	Duration (sec)	Frequency (Imp/sec)	Duration (sec)	Frequency (Imp/sec)	Duration (sec)
Insp.						
Pre-deflation	17.3 ± 4.3 @	0.48 ± 0.03 @	75.0 ± 18.9	0.49 ± 0.02	17.4 ± 0.1	0.65 ± 0.06
1st breath	24.7 ± 5.5	0.69 ± 0.05	118.6 ± 21.0	0.83 ± 0.03	35.1 ± 4.2	1.05 ± 0.35
Change (%)	+ 42	+44**	+ 58	+ 69**	+ 101	+ 61
Exp.						
Pre-deflation	1.6 ± 0.5	0.16 ± 0.08	10.7 ± 2.5	1.20 ± 0.20	33.6 ± 3.8	2.08 ± 0.29
1st breath	10.9 ± 5.4	0.15 ± 0.08	63.1 ± 15.1	0.15 ± 0.03	29.5 ± 18.3	0.13 ± 0.03
Change (%)	+581**	- 6	+ 491**	- 87**	- 12	- 93*

@ - values are means and standard errors. N - Number of fibres studied. Insp. - Inspiratory discharge Exp. - Expiratory discharge. Significance of difference from pre-deflation values: * $P < 0.05$; ** $P < 0.01$ by Mann-Whitney test.

Table 7

Effect of lung deflation on recurrent laryngeal motoneurone discharge

	Inspiratory Discharge			Expiratory Discharge		
	Pre-deflation	Deflation	Change (%)	Pre-deflation	Deflation	Change (%)
P-ILMs (N-13)						
Frequency (Imp/sec)	17.3 ± 4.3@	26.9 ± 4.9	+55**	1.6 ± 0.5	10.4 ± 5.0	+550*
Duration (sec)	0.48 ± 0.03@	0.58 ± 0.05	+20	0.16 ± 0.08	0.18 ± 0.06	+12
T-ILMs (N-7)						
Frequency (Imp/sec)	75.0 ± 18.9	121.1 ± 19.2	+61	10.7 ± 2.5	46..8 ± 16.5	+337**
Duration (sec)	0.49 ± 0.02	0.61 ± 0.02	+24**	1.20 ± 0.20	0.17 ± 0.03	-86**
T-ELMs (N-2)						
Frequency (Imp/sec)	17.4 ± 0.1	38.9 ± 3.1	+123	33.6 ± 3.8	28.1 ± 17.8	-16
Duration (sec)	0.65 ± 0.06	0.84 ± 0.15	+29	2.08 ± 0.29	0.20 ± 0.04	-90*

@ - values are means and standard errors. N - Number of fibres studied. Significance of difference of response to lung deflation compared to pre-deflation values: * $P < 0.05$; ** $P < 0.01$ by Mann-Whitney test.

41 and 44). The increase in duration was greater during the first inspiratory discharge (mean increase in duration=69%, $P<0.01$). During expiration, there were also significant changes in the mean frequency (+337%, $P<0.01$) and duration of firing (-86%, $P<0.01$). In the first expiratory discharge the increase in firing rate was greater than the mean firing rate (491%, $P<0.01$ vs 337%, $P<0.01$).

P-ELM showed a decrease in frequency of discharge during the inspiratory and expiratory phases (by -74% and -93% respectively). There was also a decrease in duration of firing during both phases of the respiratory cycle (by -62% and -92% respectively).

Changes in the pattern of firing were also observed in T-ELMs during lung deflation. These fibres which usually fired with higher frequency during expiration showed higher frequency of discharge during inspiration. There was an increase in mean firing rate during inspiration (123%) but a decrease during expiration (-16%) (Figures 41 and 45). However, this was not statistically significant. The mean duration of T-ELM discharge during expiration decreased significantly by 90% ($P<0.05$) but that of inspiratory discharge was not significantly affected.

3.11423 Subsequent Effects

After lung deflation, 5 P-ILMs lost their phasic

activity and became tonically active. An increase in frequency of P-ILM discharge during inspiration was observed (54%, $P<0.01$), a larger response occurring during the first inspiratory discharge after lung deflation (74%, $P<0.01$) (Tables 8 and 9). The change in firing rate during expiration was not statistically significant.

The frequency of T-ILM activity during expiration increased by 103% ($P<0.05$). No significant change in discharge frequency was observed during inspiration.

Both the frequency and duration of P-ELM activity decreased during inspiration and expiration (change in mean frequency = -26% and -93% respectively; change in mean duration = -78% and -99% respectively).

The 2 T-ELMs exhibited a decrease in frequency and duration of firing during inspiration (mean decrease=-66% and -43% respectively) and expiration (mean decrease = -7% and -3% respectively). On the other hand, the activity of the first inspiratory discharge was increased (frequency = 101%, duration = 61%) but not to a statistically significant degree. A significant decrease in duration of the first expiratory discharge was observed (-93%, $P<0.05$).

Table 8

Response of recurrent laryngeal motoneurones during the first breath after lung deflation

Pre-deflation

	T-ILMs		P-ILMs		T-ELMs	
	Insp.	Exp.	Insp.	Exp.	Insp.	Exp.
Frequency (Imp/sec)	@ 75.0 ± 18.9	10.7 ± 2.5	17.3 ± 4.3	1.6 ±0.5	17.4 ± 0.1	33.6 ± 3.8
Duration (sec)	@ 0.49 ± 0.02	1.20 ±0.20	0.48 ±0.03	0.16 ±0.08	0.65 ±0.06	2.08 ±0.29
	N - 7		N - 13		N - 2	

1st breath

Frequency (Imp/sec)	125.2 ± 28.5	20.6 ± 3.5	29.5 ± 4.9	2.0 ±0.9	35.1 ± 4.2	29.5 ± 18.3
Duration (sec)	0.44 ±0.03	1.57 ±0.13	0.49 ±0.05	0.16 ±0.09	1.05 ±0.35	0.13 ±0.03

Change (%)

Frequency	+67	+93	+71**	+25	+101	-12
Duration	-10	+31	+ 2	0	+ 61	-93*

@ - values are means and standard errors. N - Number of fibres studied.
Insp. - Inspiratory discharge Exp. - Expiratory discharge
Significance of difference of response during 1st breath to that during control: * $P < 0.05$; ** $P < 0.01$ by Mann-Whitney test.

Table 9

Data on recurrent laryngeal motoneurone activity after lung deflation

	Inspiratory Discharge			Expiratory Discharge		
	Control	After deflation	Change (%)	Control	After deflation	Change (%)
P-ILMs (N-13)						
Frequency (Imp/sec)	17.3±4.3@	26.7±4.4	+54**	1.6±0.5	1.8±0.9	+12
Duration (sec)	0.48±0.03@	0.47±0.04	-2	0.16±0.08	0.14±0.08	-12
T-ILMs (N-7)						
Frequency (Imp/sec)	75.0±18.9	119±26.7	+49	10.7±2.5	21.8±3.8	+103*
Duration (sec)	0.49±0.02	0.43±0.03	-12	1.20±0.20	1.56±0.11	+30
T-ELMs (N-2)						
Frequency (Imp/sec)	17.4±0.1	5.9±2.2	-66	33.6±3.8	31.4±7.3	-7
Duration (sec)	0.65±0.06	0.37±0.08	-43	2.07±0.29	1.99±0.17	-3

@ = values are means and standard errors. N = Number of fibres studied.

* $P < 0.05$; ** $P < 0.01$ by Mann-Whitney test.

3.12 Motoneurone Discharge Patterns During Pulmonary Stretch Receptor Block

3.121 Degree of Stretch Receptor Block

Among the 24 motoneurones, 6 P-ILMs and 3 ELMs from six rabbits were further studied during pulmonary stretch receptor block with sulphur dioxide (SO_2). The degree of stretch receptor block after SO_2 administration was ascertained by the Hering-Breuer inflation test before and after SO_2 exposure (Table 10). When stretch receptors were intact, inflation of the lungs of the six rabbits with a positive pressure of 10 cm H_2O produced an average pause of five times (9.99 ± 1.11 sec) the duration of control breath (2.24 ± 0.09 sec). In the same animals, after exposure to SO_2, inflation caused an average increase in cycle duration by twice the previous breath (3.03 ± 0.17 sec). In 2 out of 9 runs on six rabbits, the inhibitory ratio was reduced to unity i.e. the Hering-Breuer reflex was completely abolished. The rest of the rabbits had inhibitory ratios ranging from 2.5 to 7.9.

3.122 Spontaneous Laryngeal Motoneurone Activity

After inhalation of SO_2, the mean frequency and duration of inspiratory discharge of 6 P-ILMs increased by 95%, ($P<0.05$) and 85% ($P<0.01$) respectively (Figures 46a and 46b, Table II). On the other hand, the increases in frequency and duration of

Table 10

Assessment of the degree of pulmonary stretch receptor (PSR) block after sulphur dioxide administration

RABBIT NUMBER	FIBRE NUMBER	PSR INTACT INHIBITORY RATIO	PSR BLOCK INHIBITORY RATIO
3	10	14.3	4.3
3	12	15.3	3.3
4	14	20.7	7.9
5	15	7.4	2.0
5	16	11.9	6.1
6	17	21.6	2.5
7	20	8.6	3.4
9	23	5.0	1.1
9	24	8.2	0.9

$$\text{Inhibitory Ratio (i.r.)} = \frac{\text{Expiratory time during lung inflation}}{\text{Control expiratory time}}$$

Hering-Breuer inflation reflex is completely abolished (complete block of stretch receptor activity) if i.r. $\leq$ 1.0 (Davies et al, 1981).

Table 11

Effect of pulmonary stretch receptor block on recurrent laryngeal motoneurone discharge

	Inspiratory Discharge		Expiratory Discharge	
	Frequency (Imp/sec)	Duration (sec)	Frequency (Imp/sec)	Duration (sec)
P-ILMs (N - 6)				
Control	20.5 ± 8.2@	0.50 ± 0.08@	0.6 ± 0.3	0.01 ± 0.01
Receptor Block	40.1 ± 12.9	0.94 ± 0.08	6.1 ± 3.7	0.53 ± 0.20
Change (%)	+95*	+85**	+891	+3585
P-ELM (N - 1)				
Control	2.2	0.06	4.3	1.37
Receptor Block	37.3	0.74	1.2	0.54
Change (%)	+1595	+1133	-72	-60
T-ELMs (N - 2)				
Control	16.6 ± 5.9	0.61 ± 0.12	43.0 ± 9.2	1.67 ± 0.07
Receptor Block	7.1 ± 3.8	1.28 ± 0.12	26.2 ± 5.7	1.99 ± 0.39
Change (%)	-57	+110	-39	+19

@ - values are means and standard errors. N - Number of fibres studied. Significance of differences from controls: * $P < 0.05$, ** $P < 0.01$ by Mann-Whitney test.

discharge during expiration were insignificant. Moreover, changes in the pattern of firing were observed in 3 of these neurones. These fibres, which fired only in inspiration, became tonically active during expiration (T-I pattern) (Figure 47). The activity of T-ILMs during PSR block was not studied.

P-ELM (N = 1 fibre) lost its phasic acitivity and acquired a T-I pattern of discharge during PSR block (Figure 48). The frequency and duration of inspiratory activity increased by 1595% and 1133% respectively, whereas in expiration they decreased by -72% and -60% respectively.

Exposure to SO_2 decreased the firing rate of T-ELMs during inspiration and expiration (Figure 49) but not to a statistically significant degree. The above treatment also had no significant effects on the duration of T-ELM discharge.

3.123 Patterns of Laryngeal Motoneurone Discharge During Hering-Breuer Reflex

3.1231 Responses to Lung Inflation

3.12311 Transient Effects

In 7 out of 9 tests on 6 inspiratory laryngeal motoneurones, lung inflation elicited a transient response. An increase in the discharge frequency was observed in 4 of the above tests. There was no response in 2 tests. The position of lung inflation

and the type of transient response elicited is shown in Figures 33 and 34.

After the release lung inflation, transient responses were elicited in 5 out of 9 tests on 6 ILMs, among which 2 tests showed an increase in discharge frequency (Figure 35). There was no response in 4 instances.

Following lung inflation, all 5 tests on 3 ELMs exhibited increase in firing rate (Figures 36 and 37). The maximum response was obtained when the above manoeuvre was introduced during early expiration.

Following release of lung inflation, transient responses were also triggered in all 5 tests on 3 ELMs, out of which 4 showed increase in firing rate (Figure 38).

3.12312 Initial Effects

During lung inflation, the laryngeal motoneurones continued to discharge in phase with the respiratory cycle in 6 out of 10 tests on 6 P-ILMs (Figures 50a and 50b). In 4 instances (3 P-ILMs), the P-I motoneurones acquired a T-I pattern of discharge. The increase in discharge frequency in inspiration and expiration was not statistically significant. The same motoneurones exhibited the type A response (as defined earlier) in one test, and the type B response in 2 tests in the intact state.

Lung inflation also elicited:

i) Type A response in another 3 tests on 2 P-ILMs which exhibited a Type A (in 2 instances) and Type C response (in one instance) in the intact state;

ii) Type B response (ITFR = 0.04) in one test on a P-ILM which showed a Type C response in the intact state.

As for the expiratory motoneurones, lung inflation elicited:

i) Type A response in one test on one P-ELM, which showed a similar response in the intact state;

ii) Type D response (ITFR = 2.10±0.42) in 3 out of 4 tests on 2 T-ELMs which showed Type D response in 1 instance and Type B response in 2 instances prior to exposure to SO_2. There was no change in frequency of discharge during lung inflation in one case.

3.12313 Subsequent Effects

3 P-ILMs assumed a T-I pattern of discharge after inflation and 1 P-ILM a T-E type. In the intact state, only 2 of these P-ILMs exhibited a phase-spanning pattern of firing. The increase in frequency of inspiratory and expiratory discharges was not

significant (Tables 12 and 13). With stretch receptor activity inhibited by SO_2, there was no significant difference in the response of P-ILMs after lung inflation from that in the intact state.

The P-ELM (N=1 fibre) assumed a T-I discharge after lung inflation. The same motoneurone had a T-E discharge in the intact state. The frequency of inspiratory discharge decreased by 16% whereas that of expiratory discharge increased by 2250%. The durations of firing were increased in both cases by 30% and 104% respectively).

The frequency of inspiratory and expiratory discharges of T-ELMs was not significantly changed. The increase in duration of inspiratory discharge during PSR intact (49%, $P<0.05$) became significantly greater in the blocked state (83%, $P<0.05$). The decrease in duration of expiratory discharge after lung inflation during PSR block was significantly smaller than that in the intact state ($P<0.05$).

3.1232 Responses to Lung Deflation

Lung deflation of 20 cm H_2O negative pressure was applied in 4 tests on 3 P-ILMs (from 3 rabbits) and 5 tests on 3 ELMs (from 2 rabbits). Negative pressure of 10 cm H_2O was used in 6 tests on 3 P-ILMs (from 2 rabbits).

Table 12

Effect of lung inflation before and during stretch receptor block on the first inspiratory and expiratory discharge after lung inflation

	Control		S02 - blocked	
	Pre-inflation	1st breath	Pre-inflation	1st breath
P-ILMs (N-6)				
Insp.				
Frequency (imp/sec)	20.5±8.2@	33.3 ± 7.6	40.1 ± 12.9	46.5 ± 16.9
Duration (sec)	0.55 ± 0.08@	0.64±0.09	0.99 ± 0.07	1.19 ± 0.07
Exp.				
Frequency (Imp/sec)	0.6 ± 0.3	3.6 ± 1.8	6.1 ± 3.7	11.0 ± 6.8
Duration (sec)	0.02 ± 0.01	0.18 ± 0.10	0.51 ± 0.17	0.32 ± 0.14
P-ELM (N-1)				
Insp.				
Frequency (Imp/sec)	2.2	18.8	37.3	27.0
Duration (sec)	0.06	0.81	0.74	1.04
Exp.				
Frequency (Imp/sec)	4.3	28.8	1.2	35.4
Duration (sec)	1.37	1.28	0.54	1.10
T-ELMs (N-2)				
Insp.				
Frequency (Imp/sec)	16.5±5.9	45.2±3.8	7.1±3.8	20.6±6.3
Duration (sec)	0.77±0.09	1.32±0.02	1.29±0.08	2.62±0.29
Exp.				
Frequency (Imp/sec)	43.0±9.2	47.0±11.4	26.2±5.7	31.7±2.5
Duration	1.81±0.11	0.64±0.05	2.06±0.28	1.73±0.20

@ - values are means and standard errors. N refers to the number of fibres studied. Significance of difference from pre-inflation values; * $P < 0.05$.

Table 13

Data on recurrent laryngeal motoneurone activity after lung inflation before and during stretch receptor block

	Control			SO2-blocked		
	Before Inflation	After Inflation	Change (%)	Before Inflation	After Inflation	Change (%)
P-ILMs (N-6)						
Insp.						
Frequency (Imp/sec)	20.5 ± 8.2	24.3 ± 5.9	+18	40.1 ±12.9	45.5 ±17.5	+13
Duration (sec)	0.55 ±0.08	0.56 ±0.09	+2	0.98 ±0.07	1.02 ±0.10	+4
Exp.						
Frequency (Imp/sec)	0.6 ±0.3	2.1 ±0.9	+234	6.1 ±3.7	10.8 ± 6.6	+77
Duration (sec)	0.02 ±0.01	0.15 ±0.09	+650	0.51 ±0.17	0.48 ±0.14	-6
P-ELM (N-1)						
Insp.						
Frequency (Imp/sec)	2.2	22.2	+909	37.3	31.3	-16
Duration (sec)	0.06	0.62	+933	0.74	0.96	+30
Exp.						
Frequency (Imp/sec)	4.3	27.9	+549	1.2	28.2	+2250
Duration (sec)	1.37	1.1	-20	0.54	1.10	+104
T-ELMs (N-2)						
Insp.						
Frequency (Imp/sec)	16.6 ±5.9	38.1 ±9.1	+130	7.1 ±3.8	20.5 ± 9.3	+189
Duration (sec)	0.69 ±0.11	1.03 ±0.05	+49*	1.29 ±0.08	2.36 ±0.42	+83*
Exp.						
Frequency (Imp/sec)	43.0 ±9.2	44.4 ±10.0	+3	26.2 ±5.7	26.9 ±0.9	+3
Duration (sec)	1.76 ±0.10	0.69 ±0.10	-60*	2.06 ±0.28	1.64 ±0.10	-21#

@ = values are means and standard errors. N = Number of fibres studied. Significance of difference from pre-inflation values : * $P < 0.05$; significance of difference of response during receptor block compared with receptor intact : #$P < 0.05$.

3.12321 Transient Effects

In 8 of 9 tests on 6 P-ILMs, transient responses were elicited following lung deflation, 2 of which showed increase in frequency of discharge when compared to the inspiratory discharge during pre-deflation breaths. There were no responses in 1 instance. The position of lung deflation and the degree of transient response it elicited is shown in Figures 33 and 34.

Release of lung deflation triggered off responses in all 9 tests on 6 ILMs, out of which 6 tests showed increase in frequency of discharge and 3 showed a decrease in discharge (Figure 35).

Lung deflation elicited transient responses in all 5 tests on 3 ELMs of which 4 showed increase in discharge frequency. The highest response was elicited when lung deflation was administered during late expiration (76% of t_E) (Figures 36 and 37).

All 5 tests on 3 ELMs showed transient responses following the release of lung deflation, 4 of which showed an increase in discharge frequency and 1 showed a decrease (Figure 38).

3.12322 Initial Effects

The effects of lung deflation are shown in Figures 51a and 51b. In 10 tests on 5 P-ILMs,

deflation of the lungs with 20 cm H_2O or 10 cm H_2O negative pressure during PSR block increased the frequency and duration of discharge in the inspiratory and expiratory phases but not to a statistically significant degree (Tables 14 and 15, Figure 52). The greater increase in duration of the first inspiratory discharge seen with lung deflation in controls (i.e. PSR intact) (67% vs 34%) continued to be observed in the blocked state (25% vs 9%). In 6 tests on 4 P-ILMs, the neurones became active during the expiratory phase as well, thus assuming a Pattern T-I discharge. One P-ILM assumed a T-E pattern of firing. In another 3 tests on 2 P-ILMs, there was no activity in the expiratory phase. The change in frequency and duration of P-ILM discharge in inspiration and expiration in the blocked state was not significantly greater than that in the intact state.

During stretch receptor block, one of the 2 T-ELMs showed a T-I pattern of discharge following lung deflation. In 4 tests on the 2 T-ELMs, negative pressure of 20 cm H_2O increased the mean frequency and duration of discharge in the inspiratory phase by 230% (not significant) and 39% ($P<0.05$) respectively (Figure 52), the increase in duration being greatest in the first breath (53%, $P<0.05$ vs 39%, $P<0.05$). On the other hand, there was a decrease in mean frequency and duration of expiratory discharge but this did not

Table 14

Response of recurrent laryngeal motoneurones during the first breath following lung deflation before and during PSR block

	PSR	Inspiratory discharge		Expiratory discharge	
		Frequency (Imp/sec)	Duration (sec)	Frequency (Imp/sec)	Duration (sec)
P-ILMs (N-6)	Intact				
Pre-deflation		17.3 ± 4.3@	0.52 ± 0.06@	1.6 ± 0.5	0.33 ± 0.18
1st breath		24.7 ± 5.5	0.87 ± 0.11	10.9 ± 5.4	0.06 ± 0.03
Change (%)		+43	+67*	+581**	-81
P-ILMs (N-6)	Block				
Pre-deflation		40.3 ± 16.9	0.97 ± 0.08	5.4 ± 3.9	0.34 ± 0.16
1st breath		58.4 ± 23.1	1.22 ± 0.13	9.1 ± 5.5	0.72 ± 0.19
Change (%)		+45	+25	+67	+111
P-ELM (N-1)	Intact				
Pre-deflation		+2.3	+0.14	+3.7	+1.01
1st breath		+1.7	+0.01	+1.3	+0.06
Change (%)		-26	-92	-64	-94
P-ELM (N-1)	Block				
Pre-deflation		+38.4	+0.78	+3.6	+1.53
1st breath		+53.7	+0.35	+40.8	+1.93
Change (%)		+40	-55	+1033	+26
T-ELMs (N-2)	Intact				
Pre-deflation		17.4 ± 0.1	0.65 ± 0.06	33.6 ± 3.8	2.08 ± 0.29
1st breath		35.1 ± 4.2	1.05 ± 0.35	29.5 ± 18.3	0.13 ± 0.03
Change (%)		+101	+61	-12	-93*
T-ELMs (N-2)	Block				
Pre-deflation		7.4 ± 3.7	1.39 ± 0.08	24.7 ± 0.4	2.28 ± 0.27
1st breath		18.4 ± 2.8	2.13 ± 0.26	20.9 ± 5.2	3.11 ± 0.44
Change (%)		+148	+53*	-15	+36#

@ = values are means and standard errors. N = Number of fibres studied.
Significance of difference from pre-deflation values, * $P < 0.05$, ** $P < 0.01$.
Significance of difference of response in blocked conditions compared with that in the intact state; # $P < 0.05$.

Table 15

Effect of lung deflation on recurrent laryngeal motoneurone discharge before and during stretch receptor block

	PSR	Inspiratory Discharge		Expiratory Discharge	
		Frequency (Imp/sec)	Duration (sec)	Frequency (Imp/sec)	Duration (sec)
P-ILMs (N=6)	*Intact*				
Pre-deflation		23.5±8.9@	0.52±0.06@	2.2±0.8	0.33±0.18
Deflation		38.6±8.2	0.70±0.08	15.5±10.7	0.17±0.10
Change (%)		+64*	+34	+620	-48
P-ILMs (N=6)	*Block*				
Pre-deflation		40.3±16.9	0.99±0.07	5.4±4.0	0.29±0.14
Deflation		66.7±24.7	1.08±0.13	9.0±3.6	0.79±0.24
Change (%)		+66	+9	+66	+168
P-ELM (N=1)	*Intact*				
Pre-deflation		2.3	0.14	3.7	1.01
Deflation		0.6	0.01	1.4	0.08
Change (%)		-74	-93	-62	-92
P-ELM (N=1)	*Block*				
Pre-deflation		38.4	0.78	3.6	1.53
Deflation		62.6	0.52	22.4	1.24
Change (%)		+63	-33	+522	-19
T-ELMs (N=2)	*Intact*				
Pre-deflation		17.4±0.1	0.65±0.06	33.6±3.8	2.08±0.29
Deflation		38.9±3.1	0.84±0.15	28.1±17.8	0.20±0.04
Change (%)		+123	+29	-16	-90*
T-ELMs (N=2)	*Block*				
Pre-deflation		7.4±3.7	1.39±0.08	24.7±0.4	2.28±0.27
Deflation		24.4±3.9	1.94±0.08	20.7±7.7	2.53±0.45
Change (%)		+230	+39* #	-16	+11#

@ = values are means and standard errors. N = Number of fibres studied. Significance of difference of response to lung deflation compared to pre-deflation values: * $P < 0.05$. Significance of difference of response to lung deflation in blocked condition compared with control: # $P < 0.05$.

reach a statistically significant level. The effects of lung deflation on discharge frequency of T-ELM before and during stretch receptor block were not significantly different. However, with stretch receptor activity inhibited by SO_2, the rabbits responded to lung deflation with a greater increase in duration of T-ELM activity during both the inspiratory and expiratory phases ($P<0.05$ in both cases).

The one P-ELM exhibited a T-I pattern of discharge. The increase in frequency of inspiratory and expiratory discharges (mean increase = 63% and 522% respectively) seen during stretch receptor inhibition was greater than that in the intact state (-74% and -62% respectively). It was not possible to ascertain if the effects in the two conditions were significantly different due to the small sample of P-ELM (N=1 fibre).

3.12323 Subsequent Effects

The activity of 4 P-ILMs was changed to a T-I pattern after the release of lung deflation. The frequency of P-ILM discharge in inspiration increased significantly (54%, $P<0.05$) before stretch receptor block but not during block, while the frequency of discharge in expiration was not significantly altered in either condition (Tables 16 and 17).

Table 16

Response of recurrent laryngeal motoneurones during the first breath after lung deflation

	Control			SO2-blocked		
	Pre-deflation	1st breath	Change(%)	Pre-deflation	1st breath	Change(%)
P-ILMs (N-6)						
Insp.						
Frequency (Imp/sec)	23.5±8.9@	39.2±7.1	+66	40.3±16.9	81.3±24.6	+102
Duration (sec)	0.52±0.06@	0.51±0.09	-1	0.97±0.08	0.93±0.10	-4
Exp.						
Frequency (Imp/sec)	2.2±0.8	2.3±1.5	+5	5.4±4.0	10.2±4.2	+87
Duration (sec)	0.33±0.18	0.25±0.17	-24	0.34±0.16	0.62±0.15	+82
P-ELM (N-1)						
Insp.						
Frequency (Imp/sec)	2.3	1.7	-26	38.4	73.3	+91
Duration (sec)	0.14	0.01	-93	0.78	0.54	-31
Exp.						
Frequency (Imp/sec)	3.7	0.8	-78	3.6	7.2	+100
Duration (sec)	1.01	0.01	-99	1.53	0.55	-64
T-ELMs (N-2)						
Insp.						
Frequency (Imp/sec)	17.4±0.1	5.6±1.9	-68	7.4±3.7	6.1±4.2	-18
Duration (sec)	0.65±0.06	0.42±0.07	-35	1.39±0.08	1.01±0.12	-27
Exp.						
Frequency (Imp/sec)	33.6±3.8	30.5±6.4	-9	24.7±0.4	22.8±1.6	-8
Duration (sec)	2.08±0.29	1.93±0.22	-7	2.28±0.27	1.75±0.12	-23

@ - values are means and standard errors. N refers to the number of fibres studied.
Insp. - Inspiratory discharge Exp. - Expiratory discharge

Table 17

Data on recurrent laryngeal motoneurone activity after lung deflation before and during stretch receptor block

	Control			SO2-blocked		
	Before Deflation	After Deflation	Change (%)	Before Deflation	After Deflation	Change (%)
P-ILMs (N-6)						
Insp.						
Frequency (Imp/sec)	23.5 @ ± 8.9	36.3 ±7.5	+54*	40.3 ±16.9	74.5 ±24.5	+85
Duration (sec)	0.52 @ ±0.06	0.51 ±0.08	-1	0.97 ±0.08	0.88 ±0.10	-9 #
Exp.						
Frequency (Imp/sec)	2.2 ±0.8	2.3 ±1.4	+5	5.4 ±4.0	7.3 ±2.5	+35
Duration (sec)	0.33 ±0.18	0.21 ±0.18	-36	0.34 ±0.16	0.52 ±0.12	+52
P-ELM (N-1)						
Insp.						
Frequency (Imp/sec)	2.3	1.7	-26	38.4	63.5	+65
Duration (sec)	0.14	0.01	-93	0.78	0.52	-33
Exp.						
Frequency (Imp/sec)	3.7	0.8	-78	3.6	4.6	+28
Duration (sec)	1.01	0.01	-99	1.53	0.41	-73
T-ELMs (N-2)						
Insp.						
Frequency (Imp/sec)	17.4 ± 0.1	5.9 ±2.2	-66	7.4 ±3.7	12.4 ±10.5	+68
Duration (sec)	0.65 ±0.06	0.37 ±0.08	-43	1.39 ±0.08	1.01 ±0.12	-27
Exp.						
Frequency (Imp/sec)	33.6 ± 3.8	31.4 ± 7.3	-7	24.7 ± 0.4	16.9 ± 4.3	-32
Duration (sec)	2.07 ±0.29	1.99 ±0.17	-3	2.28 ±0.27	1.75 ±0.12	-23

@ - values are means and standard errors. N - Number of fibres studied.
Insp. - Inspiratory discharge, Exp. - Expiratory discharge
Significance of difference from pre-deflation values: *$P < 0.05$; significance of difference of response during receptor block compared with that during receptor intact: # $P < 0.05$.

The frequency of P-ELM discharge was reduced while PSR remained intact but during PSR block there was an increase in the discharge frequency. The duration of P-ELM discharge during inspiration and expiration showed a smaller decrease in the blocked state than in the intact state (-33% vs -93%; -73% vs -99% respectively).

The frequency and duration of T-ELM activity during inspiration and expiration were not significantly changed in the presence or absence of stretch receptor activity.

3.124 Effects of Added Dead Space

Two tests were carried out on 2 P-ILMs after SO_2 exposure (Figure 53, records C and D). An increase in discharge frequency was observed during inspiration and expiration (mean increase = 117% and 4616% respectively) (Figure 54). However, the above changes were not statistically significant. There was no significant change in the duration of firing in both phases of respiration. A comparison of the effect of added dead space on laryngeal discharge before and during stretch receptor block is shown in Figure 53. The increase in firing rate in the inspiratory phase in the PSR blocked state was less than that in the intact state (0.4 : 1), whereas the increase during expiration in PSR blocked state was greater than that in the intact state (1 : 0.3). Due to the small

sample (N=1 fibre), it was not possible to determine if the above changes were significant.

3.13 Laryngeal Activity During Augmented Breaths

During the course of the above experiments with unparalysed rabbits, periodically some breaths with very deep inspiration punctuated normal breathing in rabbits with intact or blocked pulmonary stretch receptors. Both the depth and duration of inspiration exceeded those in normal breaths. Out of 13 augmented breaths recorded from 6 rabbits with intact PSR, 4 occurred during eupnoeic respiration (Figure 55), 4 immediately after lung deflation, 1 when added dead space was imposed, 3 during the early stage of lung inflation with 20 cm H_2O positive pressure and 1 immediately after lung inflation with 10 cm H_2O positive pressure (Figures 56 and 57). During PSR block, 3 augmented breaths were recorded, out of which 1 occurred during the fourth breath after the release of lung deflation and 2 during lung inflation with 10 cm H_2O positive pressure (Figure 58).

The total inspiratory effort of the augmented breath comprised two phases. The first phase resembled that of a normal spontaneous inspiration whereas the second started near the point where the peak of a normal inspiration would have been. The P-ILM activity during an augmented breath was also

two-phased (Figures 59 and 60). The mean frequency and duration of the inspiratory discharge greatly exceeded that occurring during a normal breath (415%, $P<0.01$; 83%, $P<0.001$ respectively) (Table 18, Figure 61). The build-up of discharge in the first half resembled that for normal breaths. However, there was a sudden acceleration in spike frequency during the second half of the breath, an effect which was not abolished by the absence of activity in pulmonary stretch receptors (Figures 58 and 62).

The discharge of P-ILMs in 6 out of 11 augmented breaths (excluding 5 occurring during lung inflation with prolonged expiratory pause) showed a T-I pattern of discharge. The changes in duration and discharge frequency during expiration were not statistically significant. In 9 of 16 augmented breaths, there was a lessening of P-ILM discharge immediately before the other half of the inspiratory breath began (Figure 63). In 4 out of the 9 augmented breaths for which subsequent breaths were also recorded, the firing rate of the P-ILMs dropped drastically during the succeeding breaths which were rapid and shallow.

The prolonged and intense burst of P-ILM activity during an augmented breath was not noted in T-ELM (N=1 fibre) (Figures 64 and 65). Instead, there was a decrease in frequency of inspiratory and expiratory discharges during an augmented breath (-49%

Table 18

Changes in recurrent laryngeal motoneurone discharge in inspiration and expiration during augmented breath

Condition	Inspiratory discharge		Expiratory discharge	
	Frequency (Imp/sec)	Duration (sec)	Frequency (Imp/sec)	Duration (sec)
P-ILMs (N-10)				
Control	12.6 ± 1.2@	0.49 ± 0.06@	0.4 ± 0.2	0.33 ± 0.22
Augmented breath	64.9 ± 13.3	0.90 ± 0.06	14.0 ± 7.5	0.62 ± 0.23
Change (%)	+415**	+84***	+3400	+85
T-ELM (N-1)				
Control	30.1	0.73	33.9	2.88
Augmented breath	15.4	0.92	32.2	2.01
Change (%)	-49	+26	-5	-30

@ - values are means and standard errors. N - Number of breaths.
Significance of differences from control values: ** $P < 0.01$, *** $P < 0.001$ by Mann-Whitney test.

and -5% respectively). The duration of expiratory discharge also showed a decrease (-30%).

3.14 Spontaneous Laryngeal and Phrenic Discharge Patterns

Simultaneous recordings from the right recurrent laryngeal nerve (RLN) and left phrenic nerve (PN) revealed differences between their overall discharge patterns as illustrated in Figure 66, which shows typical activity in an anaesthetized, spontaneously breathing rabbit.

Bulk recordings of the RLN in all five rabbits in this series of experiments showed activity during inspiration. The discharge of RLN occurred synchronously with that of PN. The RLN burst reached a maximum near the start of the inspiratory phase and maintained a constant level till the end of the phase. Usually there was a gradual decrease in activity as the inspiratory phase progressed.

In contrast, the PN burst started abruptly and had a discharge pattern which appeared to reach a maximum at the end of the inspiratory phase (Figure 66). No bursts of activity in RLN occurred during expiration in any of the rabbits studied in this series.

3.2 Tests on Artificially Ventilated Rabbits

The following section of the chapter will describe changes in the times of onset of the RLN and PN discharges when the animals were paralysed with gallamine triethiodide and artificially ventilated with tidal volumes at whole, half and twice of eupnoeic values before and during stretch receptor block.

3.21 Discharge Patterns of Phrenic and Recurrent Laryngeal Nerves with Pulmonary Stretch Receptor Intact

3.211 Laryngeal and Phrenic Discharge At Spontaneous Resting Tidal Volume

After the animals were paralysed with gallamine triethiodide and artificially ventilated by positive pressure with a frequency, tidal volume and with an end-tidal pCO_2 as near as possible to the unparalysed state, the respiratory rhythm of RLN and PN persisted (Figures 67a and 67b). There was still no distinct burst of expiratory activity in RLN recording. The discharge of RLN and that of PN tended to become synchronized with the ventilatory cycle. To distinguish between the ventilatory and neural cycles, the phases of the artificial ventilation cycle were designated "inflation" (when the lungs were expanding) and "deflation" (when the lung volume was decreasing); and the phases of the neural cycle were designated as "inspiratory" (the time from the abrupt

onset of PN discharge to its abrupt decrease) and "expiratory" (the portion of the cycle when PN discharge was absent). In three out of the five rabbits (No.1, 3 & 4), the onset of RLN and PN discharges occurred within the first 80% of the deflation phase of ventilation (Figure 68). In the remaining two rabbits (No.2 & 5), the onset of RLN and PN bursts occurred during the early stage of the lung inflation phase. It should be noted that rabbit 5 actually had a higher end-tidal pCO_2 (pCO_2 = 6.0%, O_2 = 16.0%) than the other four (mean pCO_2 = 4.3%, O_2 = 16.5%).

The RLN and PN bursts were not initiated during every pump cycle. The neural respiratory cycle was not always locked in a 1:1 relation to the pump cycle. In 5 out of 25 instances, the onset of both RLN and PN discharges occurred at the time of lung deflation, the onset of pump inflation stroke then occurred in the middle of the inspiratory phase and finally the RLN and PN discharges terminated abruptly in the middle of the inflation phase (Figure 67a, record A). In 5 instances, the onset of the RLN and PN discharges was linked to the inflation phase of ventilation.

In 10 out of 25 cases, a 2 : 1 sychronization of the pump and PN/RLN cycles was observed, during which one inflation phase was associated with the inspiratory phase while the second inflation phase

occurred in the middle of late expiratory phase (Figure 67b, record B). 10 cases showed a 3:1 synchronization of the pump and PN/RLN cycles (Figure 67b, record A). While one inflation phase was associated with the inspiratory phase, the second and third inflation phase occurred in the expiratory phase of the neural cycle.

3.212 Responses of Phrenic and Laryngeal Nerves to Different Ventilating Volumes

By decreasing the tidal volume to half, the onset of RLN and PN still occurred during lung deflation in 20 out of 25 cases (4 rabbits) (Figures 69 and 70). There was a lengthening of the inspiratory activity of RLN and PN as both discharges terminated at a later stage of the inflation phase, or even ended during the deflation phase of another pump cycle. The state of synchronization remained unchanged in 20 cases. However, in 5 cases, the state of synchronization was disrupted and changed from a 1:1 to a 3:1 pump to neural respiratory cycle ratio.

In another 5 instances, the onset of RLN and PN continued to be linked to the inflation phase. This was noted in the same rabbit (No. 5) which had an unusually high end-tidal pCO_2.

Increasing the tidal volume by a factor of two reinstated the 1 : 1 synchronization of the pump and

neural cycle in all cases. The firing of the RLN and PN which was initiated in late deflation in 20 out of 25 cases invariably stopped sharply in the middle of the inflation phase (Figures 71 and 72). A reduction of the inspiratory phase of RLN and PN followed and an increase in baseline activity was noted in the RLN. Eventually, both the PN and RLN discharges disappeared due to hyperventilation. Record B of Figure 72 which was taken from a different rabbit shows a depression of RLN and PN inspiratory bursts. Shortly afterwards, both the RLN and PN discharges disappeared but gradually recovered their spontaneous activity. In 5 cases, the onset of RLN and PN was initiated in the inflation phase (Figure 71, record C).

The effect on RLN and PN discharge during inflation of the lungs with a positive pressure of 10 cm H_2O is shown in Figure 73. Inflation of the lungs (during marker) inhibited the inspiratory bursts in both RLN and PN. On the other hand, there was a slow rise of tonic RLN discharge occurring during the prolonged expiratory phase.

The response of the RLN and PN activity of rabbit No.5 to different ventilating volumes differed from that of the other rabbits. The onset of RLN and PN discharge continued to occur during the early part of the pump inflation phase after the animal had been

paralysed and artificially ventilated. In spite of the change in ventilating volume to half or twice eupnoeic level, the onset of activity of the two nerves continued to be locked to the ventilator inflation stroke.

3.22 Discharge Patterns of Phrenic and Recurrent Laryngeal Nerves During Pulmonary Stretch Receptor Block

3.221 Laryngeal and Phrenic Discharge During Eupnoeic Tidal Volume

During block of PSR, inflation of the lungs with 10 cm H_2O positive pressure failed to inhibit either the RLN or PN discharge (Figure 73). The disappearance of the Hering-Breuer inflation reflex indicated that PSR had been blocked by $\dot{S}O_2$. The degree of stretch receptor block as indicated by the inhibitory ratio in each of the five rabbits is shown in Table 19.

Expiratory discharge of RLN was rarely seen in recordings taken from spontaneously breathing or paralysed and artificially ventilated rabbits whose PSR were intact. However, in rabbit 2, immediately after SO_2 inhalation, a distinct burst of late expiratory activity appeared in the RLN. A similar type of expiratory discharge also happened in rabbit 3 after SO_2 block but was only elicited on increasing the tidal volume by 100% greater than eupnoeic level.

Table 19

Evaluation of the degree of pulmonary stretch receptor block after sulphur dioxide inhalation

RABBIT NUMBER	INHIBITORY RATIO PSR INTACT	PSR BLOCK
4	7.3	2.5
2	4.1	1.4
3	5.7	0.9
1	14.3	1.1
5	18.9	1.1

$$\text{Inhibitory Ratio (i.r.)} = \frac{\text{Expiratory time during lung Inflation}}{\text{Control expiratory time}}$$

Hering-Breuer inflation reflex is completely abolished (complete block of stretch receptor activity) if i.r. $\leq$ 1.0 (Davies et al, 1981).

The once "out-of-phase" timing of both RLN and PN discharges with ventilation was disrupted during PSR block. In four of the five rabbits, there was no consistent link between the phase of the ventilating pump and the onset of RLN and PN discharges (Figure 74). In 9 out of 25 cases (in 4 out of 5 rabbits), the onset of RLN and PN bursts occurred during inflation, whereas the rest occurred during deflation (Figure 68). Furthermore, when onset of the RLN/PN activity occurred in one of the phases of ventilation, they did not consistently occur at the same time in that phase. Among the above 9 cases (in 4 rabbits), 5 occurred during the first 25% (first quartile) of inflation, 2 during the second quartile and 2 during the third quartile. On the other hand, among the other 11 cases (in 4 rabbits), 3 occurred during the first quartile of deflation, 2 during the second quartile and 6 in the last quartile.

The onset of RLN and PN activity continued to occur synchronously with each other. However, in 10 cases (2 rabbits), the neural respiratory cycle tended to go out of synchrony with the artificial ventilation cycle during which a 2:1 synchronization between the two cycles became a 1 : 1 synchronization and back to 2 : 1 again (Figure 74, record A). While 5 cases maintained 1 : 1

synchronization, there were another 5 instances when the phase relation between the two cycles showed no consistency, changing from 3:1 to 2:1 state, and back to 3:1 again (Figure 74, record B). In such a "free running" situation, the mean RLN or PN discharge and pump frequencies were neither the same nor the multiples of each other.

In 5 cases, (in the same rabbit), the onset of RLN and PN activity was linked to the deflation phase of ventilation and always occurred at the same time in that phase.

3.222 Responses of Phrenic and Laryngeal Nerves to Different Tidal Volumes

When the tidal volume was increased by 100% of the spontaneous eupnoeic value, the "free running" condition was converted to one in which the bursts of RLN and PN activity were initiated by either the inflation (5 cases in one rabbit) or deflation (20 cases in four rabbits) phase of the pump cycle (Figures 72 and 75). The time of onset of the RLN and PN discharge within inflation or deflation phase of the ventilator tended to occur at the same time in that phase.

Decreasing the tidal volume to half the spontaneous eupnoeic value triggered off a "free running" condition in 15 cases (rabbits 1,3,5) where

there was no consistent phase relationship between RLN or PN bursts and pump cycle (Figure 76). The onset of RLN and PN activity occurred during inflation in 5 of the above cases (Figure 70) and during deflation in 10 of the above cases. When the above activity occurred at one of the phases of ventilation, they did not consistently occur at the same position in that phase. Out of 5 cases during inflation, 2 occurred during the first quartile of inflation, 1 each in the second, third and last quartiles. On the other hand, among the 10 cases occurring during deflation, 3 took place during the first quartile, 1 during the second, 4 during the third and 2 during the last quartile.

It was noted that the onset of RLN and PN activity in 5 of the cases (in the same rabbit) was changed from one which was initiated in the deflation phase at eupnoeic tidal volume into a "free running" pattern when the ventilating volume was reduced to half of eupnoeic value. However, when the tidal volume was doubled, the onset of RLN or PN discharge always occurred at the same time in the deflation phase.

The commencement of RLN and PN activity in the other 2 rabbits was linked to the inflation phase in 1 rabbit (rabbit No.2) (Figure 76, record B) and to the late deflation phase in another (rabbit No. 5), and they always occurred at the same time in that phase.

The response of the RLN and PN of rabbit No. 5 differed from that of the other rabbits after SO_2 administration. The onset of RLN and PN bursts continued to occur within the inflation phase but their activities switched to the deflation phase when the tidal volume was increased to twice of eupnoeic value or decreased to half of eupnoeic level.

In summary, among the five paralysed and artificially ventilated rabbits used, RLN and PN bursts were initiated during inflation in 10 cases (2 rabbits) when PSR were intact and tidal volume at eupnoeic level; 5 cases (1 rabbit) both when PSR were intact and tidal volumes were half and twice eupnoeic values respectively; 9 cases (4 rabbits) when PSR were blocked and tidal volume at eupnoeic level; 10 cases (4 rabbits) and 5 cases (1 rabbit) when PSR were blocked and tidal volumes at half and twice eupnoeic levels respectively (Figure 77).

3.23 Laryngeal and Phrenic Activity During Cough

The results so far show that the inspiratory RLN and PN discharges reacted in almost identical fashion to different stimuli. However, it is noteworthy that the RLN and PN responded differently to SO_2 when it was first administered. The animals usually coughed on first introduction to the gas. Recordings of RLN and PN (from rabbits 4 & 5) at this juncture showed

that a short but powerful burst of activity (mean duration = 0.34±0.03) was elicited between two inspiratory RLN bursts (after a lapse of 0.28±0.03 sec from the preceding inspiratory burst) (Figure 78). No such phenomena occurred during the PN silence. There was no difference in the time lapse between the two inspiratory RLN/PN bursts as compared to that during resting respiration. The occurrence of expiratory bursts persisted for 2-3 cycles.

In rabbits 3 and 4, the inspiratory RLN and PN bursts following the expiratory bursts were prolonged and more powerful. The duration of the inspiratory RLN and PN bursts increased by 50%. Moreover, the period between these inspiratory bursts was shortened by 25%. In one rabbit, expiratory bursts were again recorded immediately afterwards (Figure 78, record A). There was no time lapse between the inspiratory laryngeal discharge and the expiratory bursts which were more intense than the ones recorded during the earlier part of the cough. The expiratory laryngeal discharge was not continuous but broken into 2 small ones (Figure 78, record A). In another rabbit, such expiratory bursts were not obtained but the time lapse between two inspiratory bursts was increased by 100% (Figure 78, record B).

3.3 Validation of Results

3.31 Histological Findings

Light microscopy revealed two groups of fibres occupying sharply defined areas in both the following levels of the left and right RLN:

a) at the level of the clavicle (Plates 1-2)

b) near its entry to the larynx (Plates 3-4).

The first group consisted of large diameter, heavily myelinated nerve fibres while the second group was composed of small diameter, finely myelinated fibres. The light microscopic techniques used in this study did not demonstrate non-myelinated fibres.

There were distinct differences in the distribution and population of the various nerve fibre types depending on the level at which the nerve was transected. At the level of the clavicle, the RLN contained many small diameter fibres and fewer large diameter fibres. At the level of its entry into the larynx, a change in fibre population of the RLN was noted within the fascicle. The large diameter, heavily myelinated nerve fibres formed a greater proportion of the fibre population (Plate 3a).

There were no observable differences in the diameters of large or small diameter, myelinated fibres between the left and right RLN. The distribution of large diameter to small diameter fibres did not appear to be different between the two nerves (Plates 3a and 4a).

3.32 Tests of Recording Equipment

It was observed that recordings of electrical signals (produced by the Neurolog's pulse generator or function generator) replayed from the tape recorder onto the pen recorder were identical with those recorded directly onto the pen recorder.

3.33 Bilateral Vagotomy above Origin of Recurrent Laryngeal Nerves

The disappearance of recurrent laryngeal nerve activity from the oscilloscope monitor consequent on cervical bilateral vagotomy confirmed the identity of the nerve (Figure 79).

Chapter Four : Discussion

Chapter Four : Discussion

The purpose of this investigation was to identify the efferent activity in the recurrent laryngeal nerve (RLN) and to stimulate the pulmonary stretch receptors (PSR) and rapidly adapting receptors before and after inhibiting PSR activity with sulphur dioxide in order to determine the role of the two groups of receptors in modulating recurrent laryngeal motoneurone (RLM) discharge.

4.1 Efferent Activity in the Recurrent Laryngeal Nerve

In the present study, motoneurones in the recurrent laryngeal nerve exhibited a variety of discharge patterns which showed respiratory periodicity. The onset and termination of discharge of these motoneurones were closely time-locked to one of the phase transitions of the respiratory cycle. These observations are similar to those of previous investigators (Green and Neil, 1955; Eyzaguirre and Taylor, 1963).

It was observed that the majority of motoneurones were of the inspiratory type (designated as P-ILMs or T-ILMs in the previous chapter). A large number of fibres had phasic-inspiratory (P-I) pattern of discharge. A small minority were difficult to classify as one or two spikes were obtained in the

expiratory phase as well. Since these may be random events, the following rule-of-thumb criterion was applied: any fibre with less than or equal to three spikes occurring during either expiration or inspiration was classified as P-ILMs or P-ELMs (phasic-expiratory laryngeal motoneurones as defined in the previous chapter) respectively. Such fibres amounted to about half of the 14 P-ILMs and one third of the 3 ELMs recorded. It is not possible to draw too fine a line between P-ILMs and T-ILMs or P-ELMs and T-ELMs. At some stages during normal breathing, P-ILMs often started firing with T-I pattern.

It was more difficult to detect fibres of the expiratory type. The difficulty in detecting such fibres has been attributed to the use of barbiturates as anaesthetics (Eyzaguirre and Taylor, 1963). Studies on laryngeal muscle activities showed that activity of adductor muscles was also less frequently found in eupnoea (Murakami and Kirchner, 1971, 1972; Remmers, 1973; Sherrey and Megirian, 1975). This is a probable explanation for the difference of results from those of Green and Neil (1955), who using chloralose-urethane anaesthetized cats were able to find many fibres firing during expiration. A more detailed discussion of the effect of anaesthesia is given in the later part of this chapter.

Motoneurones which exhibit cyclic inspiratory or

expiratory activity with a tonic discharge in the expiratory or inspiratory period respectively were also detected. Such motoneurones were also recorded by past investigators (Bianconi and Raschi, 1964; Barillot and Dussardier, 1973; Glogowska et al, 1974; Sica et al, 1985). The possibility that the tonic firing of inspiratory fibres during expiration may be due to recording from a filament containing inspiratory and expiratory fibres cannot be entirely ruled out although the methods used to obtain a single fibre render this extremely unlikely.

A similar tendency for continuous activity has also been reported by Bronk and Ferguson (1935) in nerve fibres innervating the external intercostal muscles (inspiratory) in cats, and also by Wright (1954) in fibres of the phrenic nerve in the rabbit. Motoneurones with phase-spanning types of discharge (T-I and T-E) may function to promote a smooth transition of the vocal cords from abduction to adduction and vice versa in response to the change in respiratory cycle. The T-I motoneurones could be involved in the transition from adduction to abduction and T-E from abduction to adduction.

It has been suggested that these bursts of efferent impulses in phase with inspiration or expiration may be produced by motoneurones which are

driven by the respiratory centre (Green and Neil, 1955; Bianconi and Raschi, 1964). The characteristics of the inspiratory discharges of the recurrent laryngeal nerves appear similar to those of the phrenic nerve. It has been reported however, that the discharge of the recurrent laryngeal motoneurone constantly began earlier than the onset of activity in the phrenic nerve (Bianconi and Raschi, 1964; Cohen, 1975; Sica et al, 1985). This corresponds with observations in this study that there were signs of P-ILM activity during the end-expiratory phase. In contrast to Barillot and Dussardier's (1973) findings, not all inspiratory bursts of ILMs showed early firing. Within the same fibre, some bursts occurred before the onset of inspiration whereas some occurred simultaneously with the onset of the inspiratory phase. Motoneurones whose activity starts well after the onset of inspiration may have higher firing thresholds and be more susceptible to PSR mediated inhibition (Cohen, 1975). However, evidence to support this hypothesis is still lacking.

The differences between the onset of phrenic and recurrent laryngeal activity are functionally appropriate for the differing respiratory roles of the innervated muscles, namely the diaphragm and the intrinsic laryngeal muscles respectively. Cohen (1975) attributed the difference between the two kinds

of motor activity to differing temporal organisation of the driving networks.

Teliologically, the early onset of recurrent laryngeal activity would be of advantage as the vocal cords have to be ready for the inspiratory flow of air. Using the analogy of the swing door again, those doors have to be flung open earlier to receive the incoming crowd. This is consistent with the findings that the dorsal cricoarytenoid (DCA) muscles contract earlier than the onset of inspiration.

In the present study, whole nerve recordings of phrenic and recurrent laryngeal discharge also appeared similar in their responses during different experimental conditions. However, an accurate comparison of their functional properties must be complemented by observations on individual neurones. Cohen (1975) has shown that the frequency of phrenic discharge shows an increasing firing pattern whereas the recurrent laryngeal discharge reaches a plateau level early in the inspiratory phase and then decreases. This corresponded to the finding of this study that ILMs exhibited peak firing rate during the first 75% of inspiratory duration (t_I) followed by a sharp decline during the last 25% of t_I. Presumably the pattern of discharge in the whole RLN observed by Cohen (1975) could be attributed mainly to the discharge of inspiratory fibres.

As recordngs were made from whole phrenic and recurrent laryngeal nerves, it was not possible to determine how the firing patterns of individual fibres of RLN differed from that of the phrenic. Based on power spectral analysis of inspiratory activity of PN and RLN of decerebrate cats, Richardson (1980) found that the auto-correlogram of the phrenic nerve differed from that of RLN. There were two spectral peaks in the PN power spectral densities (PSDs) at 88.1 Hz and at 37.1 Hz and two spectral peaks in the RLN PSDs at 87.4 Hz and at 55.4 Hz. Simultaneous recording from single fibre and whole nerve of PN and RLN showed that about 70% of the fibres recorded in a nerve had PSDs similar to the whole nerves PSD. A minority of PN fibres had lower spectral peaks like RLN and conversely, a minority of laryngeal fibres had lower spectral peaks like the PN. Richardson (1980) therefore suggested that there may be two central pattern generators in the brainstem with parallel pathways to PN and RLN. On the other hand, Green and Neil (1955) had suggested that the inspiratory laryngeal and phrenic motoneurones are driven by common structures in the respiratory centre. Further evidence to support any of these hypotheses is still lacking. Moreover, there is still uncertainty as to whether or not the expiratory laryngeal neurones are driven by expiratory neurones in the respiratory

centre. Eyzaguirre and Taylor (1963) reported that expiratory laryngeal discharge is enhanced by low oxygen mixtures and depressed by inhalation of high CO_2. This behaviour is different from that of central expiratory neurones which are stimulated by high CO_2 (Haber et al, 1957) and depressed by overventilation (Burns and Salmoiraghi, 1960), as well as being depressed by oxygen lack and stimulated by pure oxygen (Baumgarten, 1956).

That the recurrent laryngeal motoneurones show variations of discharge associated with fluctuations in the respiratory cycle suggests that the recurrent laryngeal-driving neurones and the respiratory centre are functionally closely connected. The recurrent laryngeal motoneurones may be secondarily driven by the respiratory centre. It is obvious that there is a marked respiratory modulation present in the RLN during quiet breathing. The respiratory rhythm in the laryngeal motoneurones may be inherent in these neurones and merely modified by interconnections and interactions between them and neurones in the respiratory centre. Conversely, the rhythm in the laryngeal motoneurones may originate from interactions involving an appropriately connected feedback system. Though recurrent laryngeal discharge may be modulated by medullary inspiratory and expiratory motoneurones, extramedullary factors such as vagal afferents may

also play a modulating influence on recurrent laryngeal discharge. There are three known lung receptors which have fibres in the vagus nerves, some of which may possibly modulate recurrent laryngeal discharge.

4.2 Vagal Modulation of Recurrent Laryngeal Motoneurone Activity: Evidence from Laryngeal Discharge Patterns

4.21 During Spontaneous Breathing

It is generally accepted that pulmonary stretch receptors (PSR) play an important role in the breath by breath regulation of breathing in some species. The effect of lung stretch receptor activity is to inhibit inspiration (see Bradley, 1977 for review). The decrease of firing rate of inspiratory laryngeal motoneurones as the inspiratory phase progressed, observed in this current study, could be due to this negative feedback mechanism. However, it is also possible that the decrease is the inherent pattern of the ILM discharge, or that it is due to the progressive increase in inspiratory terminating influence (notably lung volume) inhibiting inspiratory discharge shortly before the I-E transition. It is already known that with various inspiratory terminating inputs, such as lung volume (Clark and von Euler, 1972; Grunstein et al, 1973), the stimulus

strength needed to elicit I-E switching manifests substantial temporal progression spanning the entire inspiratory phase. When adequately activated, the "off-switch" inhibits the inspiratory discharge (Figure 80).

Similar effect of PSR input is believed to account for the inhibition of phrenic (PN) discharge near the end of the inspiratory phase, since during occlusion at minimum lung volume, it is reported that a moderate increase of PN activity may result during the last third of the inspiratory phase (Cohen, 1975). However, Cohen was recording from whole phrenic nerve and the changes he observed may have arisen from actions on discharge of "late" firing PN neurones whose activity starts well after the onset of the PN burst and which may be more susceptible to vagally mediated inhibition.

These changes are consistent with the analysis of the mechanism of the inflation reflex in the cat by Clark and von Euler (1972). The reflex has a volume "threshold" which when reached terminates inspiration and this threshold may be dependent on timing during inspiration and other influences including CO_2 tension. As lung volume is monitored by stretch receptors, this system depends on vagal integrity for its function.

Hence, during PSR block, the normally occurring PSR input is eliminated and the nature of its inhibitory effect is revealed by the pattern of disinhibition, which is manifested as an increase in frequency and duration of P-ILM discharge (Figures 81 and 82) but a decrease in frequency of T-ELM discharge (Figures 83 and 84). The disinhibition of P-ILM activity was apparent from the start of the inspiratory phase: the early attainment of peak activity was changed into one which showed an increasing frequency of discharge (Figure 85).

The PSR input seems to act by determining the level at which the P-ILM burst ceases. If this input is removed, the effect is to allow the discharge to continue for a longer time and at a higher frequency of firing.

Thus, normally PSR inhibitory effect on inspiratory laryngeal motoneurones is operative throughout the inspiratory phase and the effect seems to be a graded function of lung expansion. Presumably, as the inspiratory phase progresses, PSR input gradually increases the activity of inhibitory interneurones, for example the R beta neurones of the dorsal infrasolitary nucleus (von Euler et al, 1973a,b). However, the activity of this group must rise to a critical level in order to produce a shutoff of the recurrent laryngeal-driving neurones. This argument

is supported by the finding of this study that during lung inflation, there was a complete disappearance of ILM activity in 17.9% of the tests and a decrease in ILM discharge in another 67.9% of the tests.

Cohen (1975) used whole nerve recordings of recurrent laryngeal discharge and noted an increase of laryngeal activity during the inspiratory phase when the airway of decerebrate cats was occluded at minimum lung volume. He attributed it to the removal of PSR input and hence the recruitment of previously silent, "late" firing laryngeal neurones. However, there is a possibility that occlusion may have stimulated RAR or other lung receptors at the same time. Moreover, whole nerve recording in Cohen's study (1975) may actually be the summated laryngeal activity and so the increase in discharge frequency could be either an increase in inspiratory discharge of inspiratory fibres or an increase in tonic inspiratory activity of expiratory motoneurones.

That the effect of phasic pulmonary afferent input on laryngeal inspiratory activity is inhibitory is also shown by the marked increases of activity when this afferent input was removed by withholding inflation (Sica et al, 1985).

In this study, if an increase in frequency and duration of inspiratory discharge is observed

subsequent to bilateral intrathoracic vagotomy in SO_2-blocked animals, then it would suggest the existence of another vagal pathway other than that for the inflation reflex. The rapidly adapting receptors and /or J-receptors would be likely candidates in this pathway so that abolition of their activity should further increase frequency and duration of P-ILM activity.

4.22 During Lung Inflation

The inhibitory effect of PSR input on inspiratory laryngeal discharge also operates during the Hering-Breuer inflation reflex. Hering and Breuer stated in 1868 that occlusion of the trachea during lung inflation produces inspiratory-inhibitory and expiratory-excitatory effects which prolong the expiratory pause. The inhibitory effect of lung inflation is produced by an increase discharge of PSR carried to the respiratory centres via the vagi (Larrabee and Knowlton, 1946).

The responses to lung inflation observed in this study were varied. However, the majority of ILMs exhibited responses in the same direction as phrenic activity during the Hering-Breuer reflex. In many cases, not only was the phasic activity attenuated but a low frequency tonic discharge appeared. A minority of ILMs showed increased activity. The above observations were consistent with

the findings of previous workers (Barillot and Bianchi, 1971; Barillot and Dussardier, 1973). They explained that the vagal inputs from PSR may produce two opposite effects upon the ILMs:

i) they indirectly depress the discharge of ILMs as a consequence of the inhibition of the "inspiratory" centre;

ii) they simultaneously facilitate the discharge of ILMs through a polysynaptic pathway.

The finding of this study that ELM activity was inhibited during lung inflation is not in agreement with Green and Neil's (1955) findings. These researchers reported that lung inflation (50-100 mls of air from a syringe) had an excitatory effect on ELM. Eyzaguirre and Taylor (1963) on the other hand, found that inflation of the lungs (the volume used was unreported) did not affect the discharge of expiratory fibres. The difference in the results could be due to the different degree of inflation used, different anaesthetics and animal species.

The finding of this study, that lung inflation failed to inhibit the rhythmic discharge of P-ILMs after sulphur dioxide exposure, confirms that the reflex inhibition of phasic laryngeal discharge during the above manoeuvre is mediated by PSR. On the other hand, it also suggests the presence of a vagal

inspiratory initiation influence on RLN exposed by the block of PSR. As stretch receptors were inactivated, RAR or J-receptors are the most likely source of this effect. Past studies have established the involvement of vagal afferents by bilateral intrathoracic vagotomy (Eyzaguirre and Taylor, 1963). Since this manoeuvre also eliminates other receptors in the vagi, the extent to which PSR modulate RLM discharge could not be ascertained. Green and Neil (1955) recorded activity of RLN and PSR simultaneously. Nevertheless, it is still not conclusive that PSR mediates the effect of lung inflation on laryngeal activity since the simultaneous occurrence of RLN and PSR activity does not imply that they have any effect on each other. On the other hand, single fibre recordings show that sulphur dioxide can selectively abolish PSR activity in rabbits while leaving the rapidly adapting and J-receptors and their reflexes intact (Davies, 1976; Davies et al, 1978a).

Another finding of this study was that in some P-ILMs, lung inflation continued to inhibit phasic laryngeal discharge though PSR was blocked. This may be attributed to insufficient exposure to sulphur dioxide and/or to the degree of inflation of the lungs with a positive pressure of 20 cm H_2O.

The above results are similar to those obtained

by recording of electromyographic activity of intrinsic laryngeal muscles. Fukuda et al (1973) and Green and Neil (1955) reported that lung inflation inhibits inspiratory activity of the DCA muscles. Bartlett et al (1973) have also shown that not only was there an attenuation of phasic discharge of DCA muscle but also the appearance of tonic component during lung inflation. The effect of such a tonic discharge during the inflation apnoea is perhaps to maintain a patent airway.

4.231 During Artificial Ventilation

The hypothesis that PSR modulate inspiratory laryngeal activity is supported by the observation that the onset of recurrent laryngeal discharge occurs during the phase of lung deflation in artificially ventilated rabbits. During sulphur dioxide block with passive ventilation maintained at its previous control level, the most frequent pattern was one of total independence between pump and recurrent laryngeal bursts. This "free running" phenomenon indicates the absence of any sort of feedback: the inspiratory output is driven neither by any of the remaining vagal influences nor by extravagal inputs. On the other hand, these results may also suggest the presence of a vagal inspiratory initiation influence exposed by the block of PSR. The coincidence of RLN and PN discharges with either inflation or deflation or both

during stretch receptor block may be due to RAR which are still active at that time (Davies et al, 1978a). Though there is no evidence from this study to support this view, nevertheless this is compatible with the role of RAR in initiating inspiration and provoking augmented breaths (Davies and Roumy, 1978; Davies et al, 1981).

When stretch receptors were intact, the onset of recurrent laryngeal discharge during the inflation phase with reduced tidal volume indicates that the stretch receptor input was of insufficient stimulus to activate the "off-switch" mechanism and/or that some other mechanism may be modulating RLN.

The coincidence of recurrent laryngeal and phrenic discharge with either inflation or deflation, and the similar effects observed for both nerves during the manipulation of vagal input, suggests that PSR modulates recurrent laryngeal discharge and phrenic activity through a common intermediary of the "off-switch" mechanism. Failure of the PSR input to inhibit inspiratory laryngeal discharge through most of inspiration probably indicates that PSR inputs do not inhibit the motor output pool directly.

That rhythmical discharge of the RLN continues during PSR block, indicates that the activity of lung stretch receptors is not essential for rhythm,

but that the receptors modify the pattern of laryngeal motoneurone discharge.

The onset of recurrent laryngeal motoneurone discharge during the deflation phase at tidal volume 100% greater than eupnoeic level after SO_2 exposure may be due to the stretch receptor activity not completely inhibited by SO_2. It also suggests that RAR or J-receptors may also be involved in the modulation of RLN discharge. If this hypothesis is correct, it would follow that after bilateral intrathoracic vagotomy, RLN discharges would be "free-running" with no set phase relationship to lung inflations of any volume or frequency.

4.24 During Imposition of Added Dead Space :

The significant increase in discharge of P-ILMs, observed during inspiration when an additional dead space was imposed (Figures 81 and 82), is partly in agreement with Eyzaguirre and Taylor's (1963) findings. These researchers reported that during the administration of 6% CO_2 in air to cats deprived of chemoreceptor nerves, the activity of inspiratory fibres was increased during inspiration but decreased during expiration.

Dixon et al (1974) reported that hypercapnia (using rebreathing tube with capacity of 80ml) decreased laryngeal resistance to airflow in both the

inspiratory and expiratory phases. The increase in firing rate during inspiration and expiration found in this study could possibly account for the decreased laryngeal resistance during both phases of breathing reported by Dixon et al (1974). They, however, noted that breathing with a rebreathing tube increased expiratory resistance when both vagi had been cut in the chest.

The response of ILM to an added dead space, observed in this study, is consistent with the finding that the DCA (abductors) manifest an increase in activity during hypercapnia (Suzuki and Kirchner, 1969; Bartlett et al, 1973). However, the effect of hypercapnia on adductor muscle activity remains obscure (Sherrey and Megirian, 1975).

The response of ILM to an increase in dead space is probably not due to peripheral chemoreceptor stimulation (Eyzguirre and Taylor, 1963), since carbon dioxide acts mainly on respiratory centres (Heymans and Neil, 1958). The increase of discharge in inspiration and expiration of inspiratory fibres may be attributed to removal of PSR input, since it is known that carbon dioxide inhibits PSR activity (Bartlett and Sant'Ambrogio, 1976; Bradley et al, 1976).

This study revealed that during PSR block, there

was an increase in discharge of inspiratory laryngeal motoneurones during inspiration and expiration. However, the statistical significance of this finding could not be established due to the small samples used. Nevertheless, this suggests that RAR are excitatory if a subsequent bilateral intrathoracic vagotomy produces a decrease in recurrent laryngeal discharge.

4.25 During Augmented Breaths

From the preceding discussion, it appears that the frequency and duration of inspiratory laryngeal motoneurone discharge is governed by the activity of pulmonary stretch receptors during eupnoea. However, this may not be so during an augmented breath. Such reflex deep breaths are taken by mammals periodically (every 5-20 minutes in resting man). These breaths are thought to reverse the slow collapse of the lungs which gradually occurs in quiet breathing.

In the rabbits observed in this study, augmented breaths occurred frequently during spontaneous breathing, immediately following the release of lung deflation, and during lung inflation. Block of stretch receptors by sulphur dioxide did not suppress augmented breaths although they were less frequently produced.

Though there are various possible stimuli which

can cause augmented inspiration, the most common seems to be the decrease in compliance due to lung collapse (Reynolds, 1962; Bendixen et al, 1964) possibly mediated through RAR in the airway epithelium (Mills et al, 1969; Sellick and Widdicombe, 1970). Pulmonary stretch receptors seem unlikely to be the agent (Davies and Roumy, 1982).

There is very little published information regarding the behaviour of recurrent laryngeal motoneurones during an augmented breath. The increase in firing rate of ILM during inspiration and expiration during an augmented breath (Figures 81 and 82) can be attributed to the excitation of RAR sensitized by collapse of the lungs. The augmentation of ILM activity always occurred at the end of a normal inspiration. Since there is little difference in the rate of increase in ILM discharge between the spontaneous breath and the initial part of the augmented breath, perhaps the inspiratory-augmenting effect on ILM discharge is only initiated at the peak of a eupnoeic tidal volume.

It is possible that afferent activity from the RAR causes the "inspiratory centres" to continue firing beyond the time when lung volume information would normally halt them (Knowlton and Larrabee, 1946). The decrease in inspiratory laryngeal discharge during the rapid shallow inspirations

following an augmented breath suggests that the "inspiratory centres" are for a short while more sensitive to stretch receptor activity.

The decrease in T-ELM activity during an augmented breath (Figures 83 and 84) indicates that the activity of RAR may have an inhibitory effect on ELMs.

It is observed that inspiratory laryngeal discharge during spontaneous augmented breaths had similar characteristics to the triggered responses.

That the discharge pattern in the inspiratory laryngeal motoneurones corresponds to the biphasic characteristics of the augmented response indicates that the firing rate is modulated by the inspiratory rhythm. The reflexly induced discharge of ILMs during an augmented breath suggests that besides PSR, RAR may also play a physiological role by modulating the activity of recurrent laryngeal nerve. That the laryngeal response has similar characteristics to that of the phrenic activity in Glogowska et al (1972)'s study suggests that RAR input may be effected indirectly through the same source, namely the respiratory centre. It is interesting to note that P-ILMs increase their activity despite large increases in lung volume and an extended t_I, therefore suggesting the existence of an inhibitory influence

from RAR which could "overpower" PSR activity.

4.26 During Lung Deflation

It has been observed in this study that deflation of the lungs shortens expiratory duration (t_E) and speeds up breathing. The reflex action of deflation on breathing is by stimulation of RAR sensitized by collapse of the lungs. (Sellick and Widdicombe, 1969; Mills et al 1970, Knox; 1973) However, part of this response to deflation could be due to decreased PSR activity (Knox, 1973; Bartoli et al, 1973).

Little information exists to-date concerning the effect of lung deflation on recurrent laryngeal discharge. The increase in inspiratory discharge of ILMs during lung deflation and their brief intense bursts during expiration are reminiscent of RAR activity. It is of interest that within the current study, when the lungs were collapsed by removing air, the first inspiratory effort and discharge were greater than the subsequent ones. Furthermore, the increase of discharge of ILM was much greater during expiration than during inspiration. These effects may be explained by the stimulation of RAR which presumably induced the first inspiratory discharge by a positive feedback. However, the maintained inspiratory-excitatory effect may perhaps be the consequence of the reduction of PSR input. Blocking

PSR with sulphur dioxide altered but did not abolish this effect. This further establishes that inhibition of PSR discharge is not the main component of the deflation reflex but that other vagal afferents such as those from RAR or J-receptors may mediate the above response.

The observation that there are almost identical responses of P-ILMs to lung deflation before and during PSR block (Figures 81 and 82), suggests that RAR may have a primary role in the reflex response to lung deflation, but does not eliminate the involvement of other types of lung receptors.

The response of ILMs to lung deflation is consistent with studies on respiratory movements of the vocal cords. Murakami and Kirchner (1972) reported that deflation of the lung induced distinctive abduction of the vocal cord into an over-abducted position. These positions of the vocal cord were well explained by subsequent EMG studies. In the same study, the above workers found that lung deflation evoked a temporary activation of the DCA muscle and over-abduction of the vocal cord. Sherrey and Megirian (1975) also reported that lung deflation markedly increases cyclic activity of laryngeal abductor muscles.

The increase in inspiratory and expiratory

discharge of ILMs during lung deflation presumably brings about inspiratory dilatations of the larynx. Dixon et al (1974) reported that pneumothorax (which also stimulated RAR) caused inspiratory dilatations and expiratory constriction of the larynx, the responses of which were abolished by bilateral vagotomy below the origin of the RLN.

In this study, the T-ELMs showed a decrease in firing rate in the expiratory phase of the respiratory cycle following lung deflation (Figures 83 and 84). This is consistent with Murakami and Kirchner's (1972) findings in cats that deflation of the lungs (100cc room air) did not activate the adductors.

Under physiological conditions, RAR; activity exists at functional residual capacity while the tonic activity of stretch receptors at this level of lung inflation is low. Thus the onset of inspiratory discharge of ILM may not only depend on a waning central inhibition at end-expiratory level, but may also be influenced by an inspiratory initiating drive from RAR sensitized by the collapse of the lungs. This activating mechanism may account for the augmentation of inspiratory discharge of ILM during an augmented breath, as discussed earlier. This role is compatible with the inspiratory initiating role postulated by Davies et al (1981). It may also explain why the discharge of only the ILM was

significantly increased during expiration, but not the discharge of ELM.

Considering the mechanical effect of ILM discharge which is to open the glottis to minimize resistance to airflow, the increase in firing of ILM would facilitate hyperpnoea and hyperventilation, both of which are reflex actions of RAR. The close correlation between the response of ILM and the change in breathing pattern suggests that RAR inputs do not modulate RLN motor output pool directly, but rather, they may do so through the respiratory centre.

4.27 During Transient Changes in Lung Volume

Immediately following lung inflation or deflation and after its release, the ILMs and ELMs generally showed a brief intense and irregular discharge. That this prompt and vigorous response is more consistently elicited following lung inflation or deflation at early inspiratory phase or end-expiratory level, suggests the association of RLN with RAR which may be stimulated by these rapid changes in lung volume (Mills et al, 1970).

The early excitatory responses (lasting from 0.3 to 0.5 secs), appearing before the inhibitory responses in some ILMs at the onset of lung inflation, may be associated with the paradoxical reflex described by Head (1889). These responses were

more frequently seen during lung inflation with 20 cm H_2O positive pressure, than with 10cm H_2O positive pressure. RAR is also a likely candidate mediating this reflex (Sellick and Widdicombe, 1970; Fillenz and Widdicombe, 1972).

Davies and Roumy (personal communication) found rapid inflation of the lungs during inspiration and deflation during expiration to be the most potent stimulus to RAR in rabbits. This may possibly explain the sharper burst of ILM discharge when the application of inflation was made during early inspiration and deflation during expiration observed in this study.

The observed transient changes in laryngeal discharge after release of lung inflation are consistent with those of McCaffrey and Kern (1980), who noted that at the termination of lung inflation, the rapid deflation of the lungs produced a large increase in laryngeal resistance. They attributed this response either to the rapid inhibition of stretch receptor activity or to the stimulation of RAR by rapid lung deflation. As block of stretch receptors by sulphur dioxide did not suppress transient laryngeal discharge, pulmonary stretch endings seem unlikely to be mediating the above effect.

That ILMs and ELMs appear to be very rapidly adapting to volume changes of the air passages in most cases, suggests that RAR has an excitatory influence on recurrent laryngeal motoneurone activity.

4.3 Role of J-receptors

This study did not examine the roles of type J-receptors, previously studied by Paintal (1969), or the C fibre endings, studied by Coleridge and Coleridge (1977a), in the modulation of laryngeal motoneurone discharge. They may have contributed to the responses ascribed above to PSR or RAR. Furthermore, it is not known if SO_2 exposure may stimulate or sensitize J-receptors. Nevertheless, it is considered unlikely that the sensory endings of non-myelinated fibres can account for the results in this study. Guz and Trenchard (1971) have demonstrated that J-receptors have no tonic reflex effect on breathing in rabbits with healthy lungs. Coleridge and Coleridge (1977a) point out that these receptors may be a heterogeneous collection of endings and therefore be activated by a variety of stimuli. However, in cats, inflations of up to four times tidal volume fail to excite J-receptors (Paintal, 1969) and in rabbits, they are not stimulated, or only very weakly, by large maintained inflations or deflations (Sellick and Widdicombe, 1970). The natural stimulus suggested by Paintal (1973a) for J-receptors is

"pulmonary congestion". The degree of inflation needed to stimulate the receptors in cats (Armstrong and Luck, 1974), rabbits (Sellick and Widdicombe, 1970) and dogs (Coleridge et al, 1965) and the "sparse and irregular" activity of both bronchial and pulmonary C-fibres reported by Coleridge and Coleridge (1977a) for dogs, militates against, but does not exclude, the possibility of their modulating recurrent laryngeal motoneurone discharge under conditions of near eupnoeic tidal volume.

Having eliminated the possibility of J-receptor involvement, the most likely vagal afferents involved in the reflexes studied are those from the pulmonary stretch receptors and rapidly adapting receptors. There does, however, remain a possibility of the involvement of other unknown receptors with vagal fibres, which therefore warrants further investigations. A study of the physiologic role of J-receptors in modulating RLM discharge may be carried out using some chemical stimuli such as phenyl diguanide and capsaicin (Coleridge et al, 1965; Paintal, 1969).

4.4 Role of other Modulating Factors other than Vagal Inputs

As discussed in the preceding section, the inputs affecting recurrent laryngeal discharge may

arise in the lung and have their afferent pathways in the vagal nerves.

In order to abolish lung vagal inputs without interruption of the motor pathways to the larynx, bilateral intrathoracic vagotomy has to be performed in the chest just below the origins of the recurrent laryngeal nerves. It would be necessary to open the chest on both sides. In order to restore spontaneous breathing, the chest would have to be closed. In view of the complicated operative procedures involved and the likelihood of inducing pneumothorax, bilateral intrathoracic vagotomy was not carried out. Hence it was not possible to ascertain the degree of importance of RAR and other lung receptors in modulating RLM discharge and also to know if there are other influences other than those from PSR and RAR modulating RLM discharge. Thus it is not known if other inputs from peripheral chemoreceptors and intercostal afferents may interact with those from vagal afferent fibres in modulating RLM discharge.

From the rhythmic discharge of RLN, it is quite obvious that there is a respiratory modulation and that RLN may be secondarily driven by the respiratory centre. The experiments so far have also shown that RLN reacts in almost identical fashion to different experimental stimuli as the phrenic nerve (PN). It is possible that RLN and PN may have common pathways in

the medullary respiratory areas. Projections from the dorsal respiratory group (DRG) to the ventral respiratory group (site of laryngeal motoneurones) have been described (Merrill, 1974) (Figure 86). Conceivably, the DRG is also the site of laryngeal premotor neurones. However, Feldman and Speck (1983) did not find any case of a correlation peak in cross-correlations between DRG inspiratory neurones and ipsilateral RLN discharge. On the other hand, Cohen and Feldman (1984) found that some DRG inspiratory neurones have discharge patterns and inflation responses similar to those of laryngeal inspiratory neurones. It is therefore thought that such neurones may be part of a premotor system that projects to ILMs (Sica et al, 1985).

The ILMs may be modulated indirectly by vagal inputs through the intermediary of the respiratory centre. On the other hand, Richardson (1980) has suggested that there might be two central rhythm generators in the brainstem, with parallel pathways to the phrenic and the recurrent laryngeal motoneurones, in which case there might also be a possibility that the PSR and RAR input project to the central generator driving RLN and modulate it directly. Whether a separate respiratory generator exists for recurrent laryngeal motoneurones remains to be investigated.

The picture that has been created of the mechanisms modulating RLN activity is certainly over simplified. It must soon be modified or described in finer detail to include the mode of action of other vagal afferents and the effects of the central and peripheral chemoreceptors upon laryngeal motoneurone discharge.

4.5 Functional Advantage of Vagal Modulation

There is little question that the modulation of RLN discharge does not depend exclusively on the activity of pulmonary stretch receptors during inspiration. Nevertheless, PSR and RAR modulation, whether direct or indirect, may have an important influence on the pattern of laryngeal discharge during quiet breathing. It seems that vagal afferents not only influence regulation of phrenic activity corresponding to tidal volume and respiratory frequency, but also patterns of inspiratory and expiratory discharge in the RLN. This is not surprising in view of the role of the larynx as an effector respiratory organ, abducting during inspiration to facilitate in-flow of air, and adducting during expiration to retard expiratory flow. The outcome of this modulation is that it adjusts the pattern of breathing to make it most economical in terms of the work or force of breathing.

4.6 Laryngeal Discharge Pattern during Cough

It is known that the larynx plays an important role in cough. The cough observed in this study when the animals were first introduced to sulphur dioxide may be attributed to the activation of lung receptors. Widdicombe (1954) attributed the sulphur dioxide induced cough in his animals to irritant receptors (which in this study are referred to as RAR).

In the current study, sulphur dioxide was passed via the tracheal cannula, thereby bypassing the larynx. From this it can be seen that the role of laryngeal afferents mediating nociceptive and proprioceptive inputs from the larynx probably plays a small part. The animals stop coughing shortly after SO_2 inhalation. The cessation of coughing may be due to the blocking of the airway stretch receptors by sulphur dioxide. These receptors have been reported by Sant'Ambrogio et al (1984) to play a role in the genesis of coughing. However, there is insufficient evidence in this study to confirm their findings.

One of the striking features observed in the cough reflex was the short but powerful expiratory bursts elicited in the RLN immediately following sulphur dioxide administration. This can be correlated to studies carried out by Bucher (1958), Floersheim (1959) and Weber (1959) on the changes in vocal cords and glottic aperture based on three

phases of cough which have been distinguished as:

a) an initial inspiration phase

b) a compressive phase

c) an expulsive phase.

During the "initial inspiration" phase, it is documented that the glottis dilates to a greater degree than in resting respiration. The upper airway resistance drops sharply. When the inspiration is over, the glottis narrows again, just before the pleural and tracheal pressures begin to rise as a result of the contraction of the expiratory muscles. In cough, this expiratory narrowing of the glottal calibre is known to take place faster than in resting respiration and results in complete closure of the glottis. The strong expiratory bursts, observed during sulphur dioxide inhalation in this study, could probably account for the violent closure of the glottis. The glottal resistance created during the compressive phase helps to intensify the expiratory effort, both reflexly, via proprioceptive pathways, and mechanically. The mechanical effect of expiratory closure is probably to allow time for synchronization of the various groups of expiratory muscles and also to build up intrathoracic pressure so that the maximum effect can be obtained (in:Korpas and Tomori, 1979).

The strong and prolonged inspiratory bursts of RLN following the expiratory discharge noted in this

study may account for the events described in the expulsive phase of cough which consists of an abrupt widening of the glottal lumen beginning at the peak of tracheal and pleural pressure. The glottal lumen then remains opens for some time and afterwards narrows again. The role of the expulsive phase is believed to reduce the resistance in the glottal region against the air exhaled from the lungs, thereby speeding up its expulsion (Bucher, 1958; Floersheim, 1959; Yamashita and Urabe, 1960; Jimenes-Vargas et al 1962). This sudden opening of the glottis, and the resultant escape of air from the lungs, causes a sharp drop in tracheal pressure, but does not immediately affect the high pleural pressure.

The importance of the RLN in cough has been demonstrated as expiratory glottal resistance in cough disappeared after bilateral transection of this nerve (in Korpas and Tomori, 1979). It is also reported in the same book that following complete sensory and motor denervation of the larynx, cough efforts slow down and lose their sudden explosive character. The loss of explosivity is due mainly to the elimination of glottal closure which is dependent on activity of the intrinsic laryngeal muscles supplied by the recurrent laryngeal nerve.

The absence of similar response in PN during

cough indicates that RLN and PN are not inevitably linked and that lung reflexes may act on recurrent laryngeal nerve by pathways other than via the respiratory centres.

4.7 Role of Recurrent Laryngeal Nerve in Respiration

Eupnoeic breathing requires the temporal co-ordination of activity in different groups of respiratory motoneurones. The similarity in responses to different stimuli of phrenic and recurrent laryngeal nerves indicates that both populations of motoneurones receive inputs from the central inspiratory pattern generator(s). However, different patterns of activity emerge as outputs, a phenomenon which may be related to different functional roles of these nerves in breathing and also to the manner by which their activities are modulated by pulmonary afferent inputs. The effect of ILM discharge is to maintain laryngeal patency during inspiration by producing contraction of the glottic dilator muscles. The attainment of peak activity early in the inspiratory phase assures maximum patency in the larynx at a time when diaphragm contraction is still small and results in a similar pattern of glottic dilatation, which reaches a maximum in early inspiration (Bartlett et al, 1973; Sherrey and Megirian, 1975). This minimizes resistance to airflow during inspiration. On the other hand, the tonic

activity of T-ILMs during expiration, which has also been observed in the discharge of DCA muscle (a glottis dilator), increases expiratory airflow by producing glottis dilatation (Barillot and Bianchi, 1971; Bartlett et al, 1973; Sherrey and Megirian, 1975).

Hence, during an augmented breath, the glottis reaches maximum dilatation due to the increase in frequency and duration of P-ILM discharge in inspiration and a corresponding decrease in T-ELM discharge.

During the addition of an added dead space, the hypercapnic effect on P-ILMs promotes the egress of CO_2 by widening the glottis due to both facilitation of abduction and attenuation of adduction.

Though difficult to detect, the activity of ELMs produces contraction of glottis constrictor muscles, which prevents too sudden an increase of expiratory airflow at the transition from the inspiratory to the expiratory phase. Previous studies (Stransky et al, 1973; Szereda-Przestaszewska and Widdicombe, 1973; Dixon et al, 1974; Glogowska et al, 1974) have shown that changes in discharge of expiratory laryngeal motor fibres can be correlated well with changes in laryngeal resistance in the expiratory phase.

Furthermore, ELMs also have a protective

function. Thus, their activity which causes adduction of the vocal cords, prevents the aspiration of foreign bodies. Moreover, as discussed earlier, the contraction of the adductor muscles during the compressive stage of the cough reflex, enables a high intratracheal pressure to be developed.

4.8 Evaluation of Method

4.81 Data Collection and Processing

The methodology used in this study allowed quantitative measurements of laryngeal discharge and its variation in physiological conditions. However, the manual process of counting spikes was rather time-consuming and liable to human error. The sample size was small and as such, may not be representative of the population. The sample size may be seen as a limiting factor of this study.

The recurrent laryngeal motoneurones show considerable variation in spontaneous activity from animal to animal and from time to time within each fibre during the same experiment. As such, the results were not pooled for analysis as some means would be weighted by those animals with unusually high spike frequency.

As the population of the motoneurones does not appear to have normal distribution and therefore assumptions underlying parametric statistical testing

could not be fulfilled, a non-parametric statistical test, namely Mann-Whitney test was used to provide a means for analyzing the data. However, the Mann-Whitney test has major limitations when the total number of observations is less than six, for example, if five observations are made: three controls and two tests and u = 0 (i.e. there is no overlap between test and control observations), P = 0.1 which is non-significant.

There was no particular advantage in using airflow, instead of integrated phrenic discharge, to delineate inspiration and expiration in this study. In fact, airflow signal became very poor when the speed of the tape was reduced and that of paper increased in order to obtain individual spikes. Nevertheless, it was convenient for practical reasons to record single fibre activity from only one nerve at any one time. Moreover, while there are many studies which have recorded phrenic and recurrent laryngeal motoneurones simultaneously, only a few studies have been reported where airflow was recorded at the same time as the recurrent laryngeal motoneurone.

4.82 Histological Findings

The fibre types present in the nerve from which recordings were made in this study were similar to

those previously described by Evans and Murray (1954) for the recurrent laryngeal nerve of the rabbit and by Murray (1957) of the cat. These investigators had shown that as the recurrent laryngeal nerve ascends between the oesophagus and trachea, small diameter fibres were distributed in tracheal, oesophageal and other branches. Hence, the nerve sample at the level of its entry into the larynx consisted mainly of large diameter fibres (Plates 3a and 4a). Consideration was therefore given to the level of the nerve from which recordings were made. In order to avoid recording action potentials from fibres innervating the trachea and oesophagus, recordings were made from RLN cut close to its entry into the larynx.

The right laryngeal motoneurone was chosen for recording purposes for practical reasons, as it was found to be more convenient to locate and to set up for electroneurographic recording. Verification by histological examination had shown that there was no observable differences in structure between the left and right RLN at the level of their entry into the larynx (Plates 3a and 4a).

4.83 Difficulties Encountered in Obtaining a Satisfactory Record

Background noise and interference posed a problem to obtaining a clear record of recurrent laryngeal discharge. Whitfield (1959) defined "noise

level" as the degree of random movement of the baseline due to spontaneous fluctuations in the system.

Interference occurred as a result of the recording system picking up unwanted signals either from the preparation itself or the environment. This interference was not completely avoidable. It is not possible to generalise about the elimination of interference. A high signal to noise ratio was obtained by a system of trial and error in which the positions of the preparation, leads and components of the apparatus were altered until a satisfactory signal was obtained.

To obtain the most convenient records of TTL spikes and flow signal, a number of different combinations of paper and tape speeds were experimented with in the early stages of this investigation. Finally, a compromise of a tape speed of 3.75 cm/sec and a paper speed of 50 mm/sec was made.

4.84 Effect of Anaesthesia and other chemicals

In interpreting the findings, it has to be borne in mind that general anaesthetics modify both the central sensitivity to vagally mediated afferent information (Severinghaus and Larson, 1965), and the activity of the lung receptors themselves (Coleridge

et al, 1964). The exact pattern of the response depends on the type and depth of anaesthetics (Widdicombe, 1954). Frankstein (1970) reported that the apnoea resulting from vagal stimulation was consistently greater during sleep, than that obtained in the same animal during wakefulness. Some similar results were obtained by Bystrzycka et al (1970) in the rabbit, and by Bouverot et al (1970) in the dog.

Eyzaguirre and Taylor (1963) reported that expiratory units of RLN were sensitive to sodium pentobarbital. Sherrey and Megirian (1974) showed that intravenously injected pentobarbital (30mg/kg) abolished periodic on-going thyroarytenoid and lateral cricoarytenoid muscle activities. In addition, dorsal cricoarytenoid muscle activity was transformed from a tonically active muscle to a phasically active one but with retention of tonic activity during the expiratory phase. On the other hand, barbiturate anaesthetized (Martensson, 1963; Nakamura et al 1958) or unanaesthetized (Kawasaki et al, 1964, 1964a) dogs, and highly anaesthetized cats under barbiturate anaesthesia (Barillot and Bianchi, 1971), readily show thyroaytenoid (adductor) activity during expiration, whereas cats under relatively deep barbiturate anaesthesia (Bartlett et al, 1973; Eyzaguirre and Taylor, 1963; Murakami and Kirchner, 1971, 1972,; Nakamura et al, 1958; Remmers and Tsiaras, 1973) do

not manifest overt phasic adductor activity. On the other hand, cats anaesthetized with chloralose-urethane manifest thyroarytenoid muscle activity during expiration (Green and Neil, 1955; Sherrey and Megirian, 1974). Moreover, Sherrey and Megirian (1974) also showed that in normocapnic, decerebrate cats given ketamine hydrochloride, lateral crico-arytenoid muscle was active in phase with expiration. However, this activity was abolished by amounts of pentobarbital which when given alone to the cat would normally induce only light anaesthesia. In short, it appears that the species of the experimental animals, and the type and level of anaesthesia used, may determine if phasic adductor activity is detectable. In view of all these, relatively light sodium pentobarbital anaesthesia was used to maintain the animal in this study.

As regard the use of sulphur dioxide, Davies et al (1978a) have shown that inhalation of 200ppm sulphur dioxide abolished activity in PSR. The mechanism of this inhibition has not yet been clearly elucidated but from their histological findings, it seems unlikely to be due to frank damage to the airways. Moreover, in their studies, both the receptors and the Hering-Breuer reflex recovered in 20-60 minutes and it was possible to repeat the procedure several times without the loss of its

effectiveness. Whether exposure to sulphur dioxide changes the sensitivity of rapidly adapting and J-receptors is not known (Davies et al, 1978a).

As to the use of gallamine, it is known to act on the central nervous system (in Koelle, 1975), and could depress chemoreflexes.

4.85 Effect of Tracheostomy

In this study, tracheostomy was adopted as a routine surgical procedure. The trachea was cannulated and so the glottis and upper respiratory tract did not play a part in respiration.

Breathing through tracheal cannula bypassed the larynx and so any change in laryngeal calibre which would otherwise impose a strong mechanical influence on breathing would not have this effect. Moreover, any alterations in upper airway resistance produced by the nose, pharynx and larynx would not affect airflow. Thus recordings of RLN discharge with the larynx "out of circuit" may alter the pattern of breathing and hence may not show the actual response of RLN when breathing with larynx "in circuit". According to Bianconi and Raschi (1964), respiratory abductor function of the larynx appears to be more susceptible to central alterations when the upper airway is bypassed through tracheostomy. Sasaki et al (1973) demonstrated from electromyographic measurement of the

DCA activity following tracheostomy that a significant alteration in central processing of its regulatory function was obtained. The efferent contribution from the brainstem received by DCA established the appropriate vocal cord position to maintain normal physiologic airway resistance within the respiratory cycle.

Mathew et al (1984) reported that when breathing was diverted from tracheostomy to the upper airway in dogs, the peak frequency and duration of the superior laryngeal activity increased, reflecting the introduction of pressure and flow stimuli to the larynx as well as an increase in the activation of laryngeal muscles ("drive stimuli"). This increase in laryngeal muscle activity is presumably dependent on the increase in total airway resistance (Sasaki et al, 1973), as well as the introduction of negative pressure into the larynx. Laryngeal negative pressure is known to activate laryngeal abductor muscles (Mathew,1984) which in turn may stimulate the "drive" receptors (Sant'Ambrogio et al, 1983). In fact, in the presence of a tracheostomy, which Mathew et al (1984) considered as a partial functional de-afferentation of the upper airway, there is an impairment of the upper airway muscle activity which compromises airway patency (Sasaki et al, 1973; Buckwalter and Sazaki, 1984). Moreover, the longer

the duration of the tracheostomy, the greater is the difficulty encountered in re-establishing the lost function (Sasaki et al 1973; Buckwalter and Sasaki, 1984).

In the dog, during spontaneous breathing when ventilatory resistance is maximal, DCA activity appears to be maximal (Buckwalter and Sasaki, 1984). Following a change to mouth breathing, these workers noted that DCA activity decreased, corresponding to a decrease in intratracheal pressure. When spontaneous breathing is shunted through a tracheostomy, abductor activity gradually diminished and disappeared. When the tracheostomy cannula was partially occluded and breathing was in part resumed through the upper air passages, DCA activity slowly returned. These findings may account for the lack of laryngeal motoneurone discharge observed in some fibres at some stages of this study. During those occasions, an added dead space was imposed to drive the recurrent laryngeal motoneurones.

4.9 Conclusions and Implications

When assumptions that

i) SO_2 exposure blocks the activity of PSR, and

ii) during PSR block, RAR are the only receptors spontaneously active,

are accepted, the analysis of the results of this study leads to the following conclusions:

I. The motoneurones in the recurrent laryngeal nerve fire with different discharge patterns and the modulation of their firing rate follows the respiratory rhythm.

II. The responses of RLMs during PSR block, lung inflation, and added dead space, all support the view that PSR activity inhibits ILM discharge whereas RAR activity initiates ILM discharge. The negative feedback mechanism from PSR input and the inspiratory initiating drive from RAR which exists at functional residual capacity results in RLN discharge starting during the phase of lung deflation in artificially ventilated animals. The activity of PSR is not essential for rhythmical discharge of RLMs.

III. The responses of RLMs during augmented breaths, lung deflation, and transient changes in lung volume, are consistent with the view that RAR inputs initiate and augment ILM discharge, but shorten ELM activity.

IV. The modulation by PSR and RAR may be effected indirectly through the respiratory centre, even though an additional and direct modulation cannot be ruled out.

V. Although J-receptors show little response to volume changes of the lungs (Paintal, 1973a; Sellick and Widdicombe, 1970), and have no tonic reflex effect on breathing in rabbits with healthy lungs (Guz and Trenchard, 1971), they may also have contributed to the responses ascribed above to PSR and RAR.

IV. The patterns of RLM discharge and their responses to changes in vagal afferent inputs are probably related to the functional role of different motoneurone populations in the control of airway resistance.

What contribution might the present study makes to the existing knowledge in the physiology of the RLN? The findings of this study support the current belief that the rhythmic discharge of RLM has respiratory significance and that this discharge is modified by pulmonary afferent inputs. However, the current study has further shown that PSR and RAR are the likely candidates reflexly modulating RLM discharge during eupnoeic breathing. The influence of RAR is further demonstrated by their initiating and accelerating effects on RLM activity during a differential block of PSR.

Furthermore, this study also validates the usefulness and specificity of SO_2-differential block which inhibits the activity of PSR while leaving RAR

and J-receptors and their reflexes intact. However, it is still not known if SO_2-exposure may also act to stimulate or sensitize J-receptors.

Summary of Findings

RESULTS OF INVESTIGATIONS ON SPONTANEOUSLY BREATHING RABBITS

1. Action potentials were recorded from single laryngeal motor fibres in 9 rabbits anaesthetized with pentobarbitone and breathing through a tracheal cannula.

2. Four types of recurrent laryngeal motoneurones (RLMs) were observed: i) phasic-inspiratory (P-ILMs); ii) tonic-inspiratory (T-ILMs); iii) phasic-expiratory (P-ELM); iv) tonic-expiratory (T-ELMs).

3. During pulmonary stretch receptor (PSR) block with sulphur dioxide, P-ILMs showed an increase in duration and frequency of inspiratory discharge.

4. Breathing through an added dead space increased the frequency of inspiratory discharge of P-ILMs.

5. The response to lung inflation varied. However, phasic recurrent laryngeal motoneurone activity was always inhibited. When stretch receptors were blocked, lung inflation failed to inhibit phasic recurrent laryngeal discharge.

6. Lung deflation increased the activity of ILMs but decreased that of ELMs. These responses persisted in a modified form after the inhibition of PSR activity.

7. During augmented breaths, P-ILM activity was two-phased. The duration and frequency of P-ILM discharge greatly exceeded that during normal breaths while that of T-ELM decreased.

8. RLMs showed intense and irregular discharge during the first 0.2 seconds following inflation or deflation and immediately after their release. The transient responses depended on the position of the respiratory cycle at which inflation or deflation occurred.

RESULTS OF INVESTIGATIONS ON ARTIFICIALLY VENTILATED RABBITS

1. Simultaneous recordings of recurrent laryngeal nerve (RLN) and phrenic nerve (PN) were carried out on 5 rabbits anaesthetized, paralysed and artificially ventilated with a tidal volume and frequency identical to their spontaneous breathing.

2. With stretch receptors intact, the onset of both RLN and phrenic discharge occurred during the deflation phase of ventilation.

3. Decreasing the tidal volume by 50% or increasing it by 100% of the spontaneous eupnoeic value could not unlink RLN and PN bursts from the deflation phase of the pump cycle.

4. During PSR block, the most frequent pattern was one of total independence between pump and RLN/PN cycles. The free-running condition was converted to one in which RLN and PN bursts were initiated by either of the pump's phases, more frequently by deflation. These timings were changed into a free-running pattern by decreasing the tidal volume to half the eupnoeic value.

5. Short but powerful expiratory laryngeal bursts were elicited during cough at the beginning of exposure to

sulphur dioxide. There was an increase in the frequency and duration of laryngeal and phrenic discharges following such expiratory bursts.

6. That selective block of PSR could not unlink the synchronization of RLN and PN activity indicates that both nerves may be driven by common structures in the respiratory centre.

The above results suggest that the activity of lung stretch receptors has an inhibitory effect on ILM discharge whereas that of RAR an activating and accelerating effect. RAR may also play a part in shortening ELM discharge, an effect compatible with the view that RAR may initiate ILM discharge.

That RLN and PN reacted in almost identical fashion to changes in vagal afferent inputs suggests that PSR and RAR may operate via the same central mechanisms to modulate both neural outputs.

References

"..... of making many books, there is no end,
and much study is a weariness of the flesh."

Ecclesiastes XII: 12

ADRIAN, E.D.: Afferent impulses in the vagus and their effect on respiration. J. Physiol. 79: 332-358 (1933).

AGOSTONI, E.; CITTERIO, G.; PICCOLI, S.: Reflex partitioning of inputs from stretch receptors of bronchi and thoracic trachea. Respir. Physiol. 60: 311-328 (1985).

ANDREW, B.L.: Proprioception at the joint of the epiglottis of the rat. J. Physiol. 126: 507-523 (1954).

ANDREW, B.L.: The respiratory displacement of the larynx: A study of the innervation of accessory respiratory muscles. J. Physiol. 130:474-487 (1955).

ANDREW, B.L.: A functional analysis of the myelinated fibres of the superior laryngeal nerve of the rat. J. Physiol. 133:420-432 (1956).

ARISTOTLE: Historia Animalium. Translated by A.L. PEEK (1965) Cambridge : Harvard University Press; cited by FINK, B.R. in "The Human Larynx: A

Functional Study". Raven Press (1975).

ARMSTRONG, D.J.; LUCK, J.C.: A comparative study of irritant and type-J receptors in the cat. Resp. Physiol. 21: 47-60 (1974).

BAIER, H.; WANNER, A.; ZARZECKI, S.; SACKNER, M.A.: Relationships among glottis opening, respiratory flow, and upper airway resistance in humans. J. Appl. Physiol. 43: 603-611 (1977).

BARILLOT, J.C.; BIANCHI, A.L.: Activite des motoneurones larynges pendant les reflexes de Hering-Breuer. J. Physiol. (Paris) 63: 783-792 (1971).

BARILLOT, J.C.; DUSSARDIER, M.: Modalites de decharge des motoneurones larynges inspiratoires dans diveres conditions experimentales. J. Physiol.(Paris) 66:593-629 (1973).

BARILLOT, J.C.; DUSSARDIER, M.: Activite des motoneurones larynges expiratoires. J. Physiol. (Paris) 72: 311-343 (1976).

BARONE, R.: "Anatomie Comparee Des Mammiferes Domestiques". p.713 (1976).

BARTLETT, D.: Origin and regulation of spontaneous deep breaths. Respir. Physiol. 12: 230-238 (1971).

BARTLETT, D.,JR.: Effects of hypercapnia and hypoxia on laryngeal resistance to airflow. Respir. Physiol., 37:293-302 (1979).

BARTLETT, D.,JR.: Effects of vagal afferents on laryngeal responses to hypercapnia and hypoxia. Respir. Physiol. 42: 189-198 (1980).

BARTLETT, D.JR.; KNUTH, S.L.: Human Vocal cord movements during voluntary hyperventilation. Resp. Physiol. 58: 289-294 (1984).

BARTLETT, D.,JR.; REMMERS, J.E.; GAUTIER, H.: Laryngeal regulation of respiratory airflow. Resp. Physiol. 18: 194-204 (1973).

BARTLETT, D.; SANT'AMBROGIO, G.: Effects of local and systemic hypercapnia on the discharge of stretch receptors in the airways of the dog. Resp. Physiol. 26: 91-99 (1976).

BARTOLI, A.; BYSTRYZCKA, E.; GUZ, A.; JAIN, S.K.; NOBLE, M.I.M.; TRENCHARD, D.P.: Studies of the pulmonary vagal control of central respiratory rhythm in the absence of breathing movements. J. Physiol. 230: 449-465 (1973).

BAUMGARTEN, R.V.: Koordinationsformen einzelner Ganglienzellen der rhombencephalen Atemzentren. Pflug. Arch. ges. Physiol., 262:573-594 (1956)

cited by EYZAGUIRRE, C; TAYLOR, J.R.: Respiratory discharge of some vagal motoneurones. J. Neurophysiol. 26: 61-78 (1963).

BENDIXEN, H.H.; SMITH, G.M.; MEAD, J.: Pattern of ventilation in young adults. J. Appl. Physiol. 19:195-198(1964).

BIANCONI, R.; RASCHI, F.: Respiratory control of motoneurons of the recurrent laryngeal nerve and hypocapnic apnoea. Arch. Ital. Biol. 102:56-73(1964).

BIRTLES, M.: Personal communication.

BLIDE, R.W.; KERR, H.D.; SPICER, W.S, JR.: Measurement of upper and lower airway resistance and conductance in man. J. Appl. Physiol. 19: 1059-1069 (1964).

BOUHUYS, A.: "The Physiology of Breathing". p.236. Academic Press (1977).

BOUVEROT, P.; CRANCE, J.P.; DEJOURS, P.: Factors influencing the intensity of the Breuer-Hering inspiration-inhibiting reflex. Respir. Physiol. 8: 376-384 (1970).

BRADLEY, G.W.: Control of breathing pattern. In Int. Rev. Physiol., Respir. Physiol. II, Ed. J.G. Widdicombe, p.185-217. Baltimore:

University Park Press (1977).

BRADLEY, G.W.; NOBLE, M.I.M.; TRENCHARD, D.: The direct effect on pulmonary stretch receptor discharge produced by changing lung carbon dioxide concentration in dogs on cardiopulmonary bypass and its action on breathing. J. Physiol. 261:359-373(1976).

BRANCATISANO, T.; COLLETT, P.W.; ENGEL, L.A.: Respiratory movements of the vocal cords. J. Appl. Physiol. 54:1269-1276(1983).

BRANCATISANO, T.P.; DODD, D.S.; COLLETT, P.W.; ENGEL, L.A.: Effect of expiratory loading on glottic dimensions in humans. J. Appl. Physiol. 58(2):605-611(1985).

BREUR, J.: Self-steering of respiration through the nervus vagus (English translation by E. Ullmann). In: Breathing: Hering-Breuer Centenary Symposium, edited by R.Porter: London: Churchill, p.365-394 (1970).

BRODY, A.W.: Mechanical compliance and resistance of the lung-thorax calculated from the flow recorded during passive expiration. Am. J.Physiol. 178: 189-196 (1954).

BRONK, D.W.; FERGUSON, L.K.: The nervous control of

intercostal respiration. Amer. J. Physiol., 110:700-707 (1935).

BUCHER, K.: Pathophysiology and pharmacology of cough. Pharm. Rev. 10: 43-58 (1958).

BUCKWALTER, J.A.; SASAKI, C.T.: Effect of tracheotomy on laryngeal function. Otolary. Clinics of N. Amer. 17(1):41(1984).

BURNS, B.D.; SALMOIRAGHI, G. C.: Repetitive firing of respiratory neurones during their burst activity. J. Neurophysiol. 23: 27-46 (1960).

BYSTRZYCKA, E.; HUSZCZUK, A.; KARCZEWSKI, W.; GROMYSZ, H.: Proc. III Europ. Congress of Anaesthesiology, Prague (w druku) (1970).

CALLANAN, D.; DIXON, M.; WIDDICOMBE, J.G.W.: The acute effects of sulphur dioxide on pulmonary mechanics, breathing patterns and pulmonary vagal afferent receptors in the rabbit. J. Physiol. 247:23-24P (1975).

CAMPBELL, C.J.; MURTAGH, J.A.; RABER, C.F.: Chemical agents and reflex control of laryngeal glottis. Ann. Otol. Rhinol. Laryngol. 72: 589-604 (1963).

CITTERIO, G.; AGOSTONI, E.: Inspiratory facilitation and inhibition from pulmonary stretch receptors in rabbits. Respir. Physiol. 53: 307-323 (1983).

CITTERIO, G.; PICCOLI, S.; AGOSTONI, E.: Breathing pattern and diaphragm EMG after sulphur dioxide in rabbit intra- or extrathoracic airways. Respir. Physiol. 59: 169-183 (1985).

CLARK, F.J.; VON EULER, C.: On the regulation of depth and rate of breathing. J. Physiol. 222: 267-295(1972).

COHEN M.I.: Phrenic and recurrent laryngeal discharge patterns and the Hering-Breuer reflex. Am. J. Physiol. 228(5):1489-1496 (1975).

COHEN, M.I.; FELDMAN, J.L.P: Discharge properties of dorsal medullary inspiratory neurons: relation to pulmonary afferent and phrenic efferent discharge. J. Neurophysiol. 51: 753-766 (1984).

COLERIDGE, H.M.; COLERIDGE, J.C.G.: Impulse activity in afferent vagal C-fibres with endings in the intrapulmonary airways of dogs. Resp. Physiol. 29:125-142 (1977a).

COLERIDGE, H.M.; COLERIDGE, J.C.G.; BANZETT, R.B.: Effect of CO_2 on afferent vagal endings in the canine lung. Resp. Physiol. 34:135-151 (1978).

COLERIDGE, H.M.; COLERIDGE, J.C.G..; KIDD, C.: Role of the pulmonary arterial baroreceptors in the effects produced by capsaicin in the dog. J.

Physiol. 170:272-85 (1964).

COLERIDGE, H.M.; COLERIDGE, J.C.G.; LUCK, J.C.: Pulmonary afferent fibres of small diameter stimulated by capsaicin and by hyperinflation of the lungs. J. Physiol. 179:248-262 (1965).

COLERIDGE, J.C.G.; COLERIDGE, H.M.: Afferent C-fibers and cardiorespiratory chemoreflexes. Am. Rev. Resp. Dis., 115:251-260 (1977b).

CZERMAK, J.N.: On the Laryngoscope and its Employment in Physiology and Medicine. London: New Syndenham Society (1861) cited by FINK, B.R.: Respiration. In "The Human Larynx: A Functional Study". p.56-84. New York: Raven, 1975.

DAVENPORT, P.W.; FREED, A.N.; REX, K.A.: The effect of sulphur dioxide on the response of rabbits to expiratory loads. Respir. Physiol. 56: 359-368 (1984).

DAVIES, A.: Respiratory reflexes in rabbits without pulmonary stretch receptor activity. In Respiratory Centres and Afferent Systems, Vol. 59, ed. DURON, B. p.253-262. Paris:INSERM (1976).

DAVIES, A.; DIXON, M.; CALLANAN, D.; HUSCZUK, A.; WIDDICOME, J.G.; WISE J.C.M.: Lung reflexes in rabbits during pulmonary stretch receptor block by sulphur dioxide. Resp. Physiol. 34:83-101

(1978a).

DAVIES, A.; DIXON, M.; PENMAN, R.; WIDDICOMBE, J.G.; WISE J.C.M.: Effect of repeated exposures to high concentration of sulphur dioxide on respiratory reflexes in rabbits. Bull. Eur. Physio-path. resp. 14:41-52(1978b).

DAVIES, A.; NADAL, J.A.; WEINMANN, G.: Effect of changes in airway pressure on the breathing pattern in conscious dogs. Q. J. Exp. Physiol. 69: 145-153 (1984).

DAVIES, A.; ROUMY, M.: Changes in the pattern of breathing provoked by irritant receptors. J. Physiol., 272:78-79P (1977).

DAVIES, A.; ROUMY, M.: The inspiratory augmenting effect of lung irritant receptor activity. J. Physiol. 275: 14P (1978).

DAVIES, A.; ROUMY, M.: The effect of transient stimulation of lung irritant receptors on the pattern of breathing in rabbits. J. Physiol. 324:389-401 (1982).

DAVIES, A.; SANT'AMBROGIO, F.; G.SANT'AMBROGIO: Onset of inspiration in rabbits during artificial ventilation. J. Physiol. 318:17-23(1981).

DEDO, H.H.: The paralyzed larynx: an electromyographic study in dogs and human. Laryngoscope 80:1455-1517(1970).

DEDO, H.H.; OGURA, J,H.: Vocal Cord Electromyography in the Dog. The Laryngoscope, 75:201-211 (1965).

DESSY, G.: Movementi respiration del laringe in condizioni normali e pathologiche. Riv. Patol. Clin. Tuberculosi, 12:791-806 (1938) cited by FINK, B.R. in "The Human Larynx: A Functional Study"; p.56-84. New York: Raven Press (1975).

DIXON M.; WIDDICOMBE J.G.; WISE J.C.M.: Laryngeal calibre during hyperthermia in cats. Resp. Physiol. 20:371-377(1974).

ENGLAND, S.J.; BARTLETT, D.JR.: Changes in respiratory movements of the human vocal cords during hyperpnea. J. Appl. Physiol. 52:780-785(1982).

ENGLAND, S.J.; BARTLETT, D.JR.; DAUBENSPECK, J.A.: Influence of human vocal cord movements on airflow and resistance during eupnea. J. Appl. Physiol. 52:773-779(1982a).

ENGLAND, S.J.; BARTLETT, D.JR.; KNUTH, S.L.: Comparison of human vocal cord movements during isocapnic hypoxia and hypercapnia. J. Appl.

Physiol. 53:81-86(1982b).

EULER, C.V.: The functional organisation of the respiratory phase switching mechanisms. Fed. Proc., 36:2375-2380 (1977).

EULER, C.V.; HAYWARD, J.N.; MARTTILA, I.; WYMAN, R.J.: The spinal connections of the inspiratory neurones of the ventro-lateral nucleus of the cat's tractus solitarius. Brain Res. 61: 23-33 (1973a).

EULER, C.V.; HAYWARD, J.N.; MARTTILA, I.; WYMAN, R.J.: Respiratory neurones of the ventrolateral nucleus of the solitary tract of cat: vagal input, spinal connections and morphological identifications. Brain Res. 61, 1-22 (1973b).

EULER, C.V.; HERRERO, F.; WEXLER, I.: Control mechanisms determining rate and depth of respiratory movements. Resp. physiol.10:93-108 (1970).

EVANS, D.H.L.; MURRAY, J.G.: Histological and functional studies on the fibre composition of the vagus nerve of the rabbit. J. Anat. 88:320-340 (1954).

EXNER, S.: Sitzoer. Akad. Wiss. Wien 89: 63 (1884) cited by HARREVELD, A.V.; TACHIBANA, S.: Innervation and reinnnervation of cricothyroid

muscle in the rabbit. Am. J. Physiol. 201(6): 1199-1202 (1961).

EYZAGUIRRE, C.; TAYLOR, J.R.: Respiratory discharge of some vagal motoneurons. J. Neurophysiol. 26:61-78 (1963).

FAABORG-ANDERSEN, K.: Electromyographic investigation of intrinsic laryngeal muscles in human. Acta. Physiol. Scand. 41:Suppl. 140(1957).

FABRICIUS, H.: De visione voce auditu. Venice (1600); cited by FINK, B.R.: Respiration. In "The Human Larynx: a Functional Study". p.56-84. New York: Raven Press, 1975.

FELDMAN, J.L.; SPECK, D.M.: Interactions among inspiratory neurons in dorsal and ventral respiratory groups in cat medulla. J.Neurophysiol. 49: 472-490 (1983).

FERRER, P.; KOLLER, E.A.: Uber die Vagusafferenzen des Meerschweinchens und ihre Bedeutung fur die Spontanatmung. Helv. Physiol. Pharmacol. Acta, 26:365-387 (1968).

FERRIS, B.G.; JR.; MEAD, J.; OPIE, L.H.: Partitioning of Respiratory Flow Resistance in Man. J. Appl. Physiol. 19:653-658(1964).

FILLENZ, M.; WIDDICOMBE, J.G.: Receptors of the lungs

and airways, p.81-112; in AUTRUM, H. et al.: Handbook of Sensory Physiology, vol. 3, p.233 (Springer-Verlag, Berlin-Heidelberg-New York 1971).

FILLENZ, M.; WIDDICOMBE, J.G.: Receptors of the lungs and airways. In: Handbook of Sensory Physiology. Vol. III/I, Enteroceptors. Berlin, Springer-Verlag, p.81-112 (1972).

FINK, B.R.: Respiration. In: The Human Larynx: A Functional Study. p.56-84. New York: Raven, 1975.

FLOERSHEIM, G.L.: Uber die Rolle der Glottis beim Husten. Helv. physiol. pharmacol. Acta 17:153-165 (1959) cited by KORPAS, J.; TOMORI, Z. In "Cough and Other Respiratory Reflexes"; p.41-63. Karger, Basel (1979).

FRANKSTEIN, S.I.: Neural control of respiration, p.56-57; in PORTER, R.:Ciba Foundation Hering-Breuer Centenary Symposium, Breathing (J. et A. Churchill, London 1970).

FRANKSTEIN S.I.; SERGEEVA Z.N.: Tonic activity of lung receptors in normal and pathological states. Nature 210:1054-1055 (1966).

FUKUDA, H.; SASAKI, C.T.; KIRCHNER, J.A.: Vagal afferent influences on phasic activity of the

posterior cricoarytenoid muscle. Acta Otolaryng. 75:112-118(1973).

GALEN: On the Usefulness of the parts of the Body. Translated by MAY, M.T.(1966). Ithaca: Corbell University Press cited by FINK, B.R.: Respiration. In "The Human Larynx: A Functional Study". p.56-84. New York: Raven Press, 1975.

GARCIA, M.: Observations on the Human Voice. Proc. Roy. Soc. (London) 7:399-420 (1855) cited by Fink, B.R.: Respiration. In "The Human Larynx: a Functional Study". p.56-84. New York: Raven Press, 1975.

GAUTIER, H.: Pattern of breathing during hypoxia or hypercapnia of the awake or anesthetized cat. Resp. Physiol. 27:193-206(1976).

GAUTIER, H.; REMMERS, J.E.; BARTLETT, D.: Control of the duration of expiration. Respir. Physiol. 18: 205-221 (1973).

GILCHRIST, A.G.: Rehabilitation after Laryngectomy. Acta Otolaryngol. 75:511-518 (1973).

GLOGOWSKA, M.; RICHARDSON, P.S.; WIDDICOMBE, J.G.; WINNING, A.J.: The role of the vagus nerves, peripheral chemoreceptors and other afferent pathways in the genesis of augmented breaths in cats and rabbits. Respir. Physiol. 16: 179-196

(1972).

GLOGOWSKA, M.; STRANSKY, A.; WIDDICOMBE, J.G.: Reflex control of discharge in motor fibres to the larynx. J. Physiol. 239:365-379 (1974).

GREEN, J.H.; NEIL, E.: The respiratory function of the laryngeal muscles. J. Physiol.,129: 134-141 (1955).

GRUNSTEIN, M.M.; YOUNES, M.; MILIC-EMILI, J.: Control of tidal volume and respiratory frequency in anaesthetized cats. J.Appl. Physiol. 35:463-476 (1973).

GUZ, A.; TRENCHARD, D.: The role of non-myelinated vagal afferent fibres from the lungs in the genesis of tachypnoea in the rabbit. J. Physiol. 213: 345-371 (1971).

HABER, E.; KOHN, K.W.; NGAI, S.H.; HOLIADAY, D.A.; WANG, S.C.: Localization of spontaneous respiratory neuronal activities in the medulla oblongata of the cat: a new location of the expiratory center. Amer. J. Physiol. 190:350-355 (1957).

HARDING, R.: State related and development changes in laryngeal function. Sleep 3:307-322 (1980).

HARREVELD, A.V.; TACHIBANA, S.: Innervation and re-

innervation of cricothyroid muscle in the rabbit. Am. J. Physiol. 201(6):1199-1202 (1961).

HEAD, H.: On the regulation of respiration. J. Physiol. 10: 1-70 (1889).

HENDERSON-SMART, G.J.; JOHNSON, P.; AND MCCLELLAND, M.E.: Asynchronous respiratory activity of the diaphragm during spontaneous breathing in the lamb. J. Physiol. 327: 377-391 (1982).

HERING, E.: Die Selbststeurerung der Atmung duch den Nervus Vagus. Sitzber Akad Wiss Wien 57:672-677 (1868).

HEYMANS, C.; NEIL, E.: Reflexogenic . Areas of the Cardiovascular system. London, Churchill (1958).

HOMBERGER, A.C.: Beitrag zum Nachweis von Kollapsafferenzen im lungenvagus des kaninchens. Helv. Physiol. Pharmacol. Acta. 26:97-118 (1968).

HYATT, R.E.; WILCOX, R.E.: Extrathoracic airway resistance in man. J. Appl. Physiol. 16: 326-330 (1961).

JIMENEZ-VARGAS, J.; MIRANDA, J.; MOURIZ, A.: Physiology of the cough. Differentiation of constrictor and dilatatory reflexes of the laryngeal sphincter. Rev. resp. Fisiol. 18: 7-21 (1962).

KAWASAKI, M.; OGURA, J.H.; TAKENOUCHI, S.: Neurophysiologic observations of normal deglutition. I.Its relationship to the respiratory cycle. Laryngoscope 74: 1747-1765 (1964).

KAWASAKI, M.; OGURA, J.H.; TAKENOUCHI, S. Neurophysiologic observations of normal deglutition. II. Its relationship to allied phenomena. Laryngoscope 74: 1766-1780 (1964a).

KEELE, C.A.; NEIL, E.; JOELS, N.: Samson Wright's Applied Physiology. p.169-171. Oxford University Press (1984).

KELLER, G.J.; LOESER, A.: Der zentripetale Lungenvagus. Z. Biol. 89: 373-395 (1926) cited by WIDDICOMBE, J.G.: Pulmonary and respiratory tract receptors. J. Exp. Biol. 100: 41-57 (1982).

KNOWLTON, G.C.; LARRABEE, M.G.: A unitary analysis of pulmonary volume receptor. Am. J. Physiol. 147: 100-114 (1946).

KNOX, C.K.: Characteristics of the inflation and deflation reflexes during expiration in the cat. J. Neurophysiol., 36: 284-295 (1973).

KOELLE, B.G.: Drugs acting at synaptic and neuroeffector junctional sites: Neuromuscular blocking agents. p.575. In "The Pharmacological

Basis of Therapeutics". Edited by GOODMAN, L.S.; GILMAN, A. Macmillan Publishing Co., Inc, 1975.

KOLLER, E.A.: Afferent vagal impulses in anaphylactic bronchial asthma. Acta Neurobiol. Exp. 33: 51-56 (1973).

KORPAS, J.; TOMORI, Z.: Cough and other respiratory reflexes. In: Progress in respiratory research, Vol. 12. Series Ed: Herzog, H. Karger, Basel, p 47-63 (1979).

LARRABEE, M.G.; KNOWLTON, G.C.: Excitation and inhibition of phrenic motoneurones by inflation of the lungs. Am. J. Physiol. 147:90-99 (1946).

LAST, R.J.: Anatomy, Regional and Applied, 5th edition. London: Churchill-Livingstone. (1972).

MARTENSSON, A.: Reflex responses and recurrent discharges evoked by stimulation of laryngeal nerves. Acta Physiol. Scand. 57: 248-269 (1963).

MATHEW, O.P.: Upper airway negative pressure effects on respiratory activity of upper airway muscles. J. Appl. Physiol. 56:500-505 (1984).

MATHEW, O.P.; SANT'AMBROGIO, G.; FISHER, J.T.; SANT'AMBROGIO, F.B.: Laryngeal pressure receptors. Respir. Physiol. 57: 113-122 (1984).

MCCAFFREY T.V.; KERN E.B.: Laryngeal regulation of airway resistance. II : Pulmonary receptor reflexes. Ann. Otol. 89:462-466 (1980).

MCCLURE, R.C: The Cranial Nerves. p.544. In "Anatomy of the Dog". Edited by Miller, M.E; Christensen, G.C; Evans, H.E. Saunders (1964).

MEAD, J.: Control of respiratory frequency. J. Appl. Physiol. 15:325 - 336 (1960).

MERRILL, E.G.: Preliminary studies on nucleus retroambigualis-nucleus of the solitary tract interactions in cats. J.Physiol. 244: 54-55P (1974).

MILLER, M.E.: Guide to dissection of the dog. 3rd Ed. p.276, Ithaca, New York (1952).

MILLS, J.E.; SELLICK, H.; WIDDICOMBE, J.G.: Activity of lung irritant receptors in pulmonary microembolism, anaphylaxis and drug-induced bronchoconstrictions. J. Physiol. 203: 337-357 (1969).

MILLS, J.E.; SELLICK, H.; WIDDICOMBE, J.G.: Epithelial irritant receptors in the lungs, p.77-99; in PORTER, R.: Ciba Foundation Hering-Breuer Centenary Symposium, Breathing (J. et A. Churchill, London 1970).

MINK, P.J.: Die respiratorischen Bewegungen des Kehlkopfes. Arch. Laryngol. Rhinol. 30:391-412 (1916) cited by FINK, B.R. In "The Human Larynx: A Functional Study", p.56-84. Raven Press (1975).

MISEROCCHI, G.; MORTOLA, J.; AND SANT'AMBROGIO, G.: Localization of pulmonary stretch receptors in the airways of the dog. J. Physiol. 235:775-782 (1973).

MITCHINSON, A.G.; YOFFEY, J.M.: Respiratory displacement of the larynx, hyoid bone and tongue. J. Anat. 81:118-120 (1947).

MORTOLA J.; SANT'AMBROGIO, G.; CLEMENT M.G.: Localization of irritant receptors in the airways of the dog. Respir. Physiol, 24: 107-14 (1975).

MURAKAMI, Y.; AND KIRCHNER, J.A.: Electrophysiological properties of laryngeal reflex closure. Acta Otolaryngol. 71: 416-425 (1971).

MURAKAMI, Y.; KIRCHNER, J.A.: Respiratory movements of the vocal cords. An electromyographic study in the cat. Laryngoscope 82: 454-467 (1972).

MURRAY, J.G.: Innervation of the intrinsic muscles of the cat's larynx by the recurrent laryngeal nerve: A unimodal nerve. J. Physiol. 135:206-212

(1957).

MURTAGH, J.A.; CAMPBELL, C.J.: The respiratory function of the larynx. Laryngoscope 61: 581 (1951).

NAKAMURA, F.; UYEDA, Y.; SONODA, Y.: Electromyographic study on respiratory movements of the intrinsic laryngeal muscles. Laryngoscope 68:109-119 (1958).

NATTIE, E.E.; TENNEY, S. M.: The ventilatory response to resistance unloading during muscular exercise. Respir. Physiol. 10: 249-262 (1970).

NEGUS, V.E.: Certain Anatomical and Physiological Considerations in Paralysis of the Larynx. Proc. Roy. Soc. Med. 40:849-853 (1947).

NEGUS, V.E.: "The comparative anatomy and physiology of the larynx". Heineman, London (1949).

OGURA, J.H.; LAM, R.L.: Anatomical and physiological correlations on stimulating the human superior laryngeal nerve. Laryngoscope, 63, 947-959 (1953).

PAINTAL A.S.: The response of pulmonary and cardiovascular vagal receptors to certain drugs. J.Physiol. 121:182-190 (1953).

PAINTAL, A.S.: Impulses in vagal afferent fibres from

specific pulmonary deflation receptors. The response of these receptors to phenyl diguanide, potato starch, 5-hydroxytryptamine and nicotine, and their role in respiratory and cardiovascular reflexes. Quart. J. Exp. Physiol. 40: 89-111 (1955).

PAINTAL, A.S.: The location and excitation of pulmonary deflation receptors by chemical substances. Quart. J. Exp. Physiol. 42: 56-71 (1957).

PAINTAL, A.S.: Mechanisms of stimulation of type J pulmonary receptors. J. Physiol. 203:511 (1969).

PAINTAL A.S.: The mechanism of excitation of type J-receptors and the J reflex, in: Breathing: Hering-Breuer Centenary synposium, ed. R. Porter (J and A. Churchill Co., London) (1970).

PAINTAL, A.S.: Sensory mechanism involved in the Benzold-Jarisch effect. Aust. J. Exp. Biol. Med Sci:51: 3-15 (1973).

PAINTAL A.S.: Vagal sensory receptors and their reflex effects. Physiological Reviews 53: 159-227 (1973a).

PETIT, J.M.; MILIC-EMILE, G.; DELHEZ, L.: Role of the

diaphragm in breathing in conscious normal men: an electromyographic study. J. Appl. Physiol. 15: 1101-1106 (1960).

PRESSMAN, J.J.: Physiology of the vocal cords in phonation and respiration. Arch. Otolaryngol. 35:355-398 (1942).

PRESSMAN, J.J.; KELEMEN, G.: Physiology of the larynx. Physiol. Rev. 35, 506-554 (1955).

RATTENBORG, C.: Laryngeal regulation of respiration. Acta Anaesthesiol. Scand. 5:129-140 (1961).

REMMERS, J.E.: Extra-segmental reflexes derived from intercostal afferents: phrenic and laryngeal responses. J.Physiol. 233: 45-62 (1973).

REMMERS, J.E.; BARTLETT, D.JR.: Reflex control of expiratory airflow and duration. J. Appl. Physiol. 42 : 80-87 (1977).

REMMERS, J.E.; TSIARAS, W.G.: Effect of lateral cervical cord lesions on the respiratory rhythm of anaesthetized, decerebrate cats after vagotomy. J. Physiol. 233: 63-74 (1973).

REX, M.A.E.: Laryngospasm during general anaesthesia in the cat. Ph.D Thesis. Massey University (1970).

REYNOLDS, L.B.: Characteristics of an inspiration

augmenting reflex in anaesthetized cat. J. Appl. Physiol. 17: 683-688 (1962),

RICHARDSON, C.A. JR.: Power spectral analysis of inspiratory activity of the phrenic and recurrent laryngeal nerves of the decerebrate cat: evidence for two central respiration pattern generators. Dissertation Abstracts International Vol. 40 (12), p.5562-B (1980).

RICHARDSON, P.S.; SANT'AMBROGIO, G; MORTOLA, J; BIANCONI, R.: The activity of lung afferent nerve during tracheal occlusion. Resp. Physiol., 18:273-283 (1973).

ROSENTHAL, J.: Die Physiologie der Atembewegungen und der Innervation derselben. In: Handuch der Physiologie, Vol. 4, part 2, edited by L. Hermann, p.163-286. Leipzig: Vogel (1882) cited by FINK, B.R.: Respiration. In "The Human Larynx: a Functional Study". p.56-84. New York: Raven Press, 1975.

SAMPSON, S.R.; VIDRUK, E.H.: Properties of "irritant" receptors in canine lung. Respir. Physiol: 25:9-22 (1975).

SANT'AMBROGIO, G.; MATHEW, O.P.; FISHER, J,T.; SANT'AMBROGIO, F.B.: Laryngeal receptors responding to transmural pressure, airflow and

local muscle activity. Respir. Physiol. 54: 317-330 (1983).

SANT'AMBROGIO, G.; AND MISEROCCHI, G.: Functional localization of pulmonary stretch receptors in the airways of the cat. Arch. Fisiol. 70: 189-195 (1973).

SANT'AMBROGIO, G.; SANT'AMBROGIO, F.B.; DAVIES, A.: Airway receptors in cough. Bull. Eur. Physiopathol. Respir. 20: 43-47 (1984).

SASAKI, C.T.; FUKUDA, H.; KIRCHNER, J.A.: Laryngeal abductor activity in response to varying ventilatory resistance. Trans. Am. Acad. Ophthalmol. Otolaryngol. 77:403-410 (1973).

SCHAEFER, E.A.: Textbook of Physiology, Vol. 2, p.279; Edinburgh: Y.J. Pentland (1900) cited by FINK, B.R. in "The Human Larynx: A Functional Study", p.56-84. New York:Raven Press (1975).

SCHIRATZKI, H.: Upper airway resistance in normal man during mouth breathing. Acta Otolaryngol. 58:535-554 (1964).

SCHIRATZKI, H.: The oral and laryngeal components of the upper airway resistance during mouth breathing. Acta Oto-laryngol. 60: 71-82 (1965).

SELLICK, H.; WIDDICOMBE, J.G.: The activity of lung

irritant receptors during pneumothorax, hyperpnoea and pulmonary vascular congestion. J. Physiol. 203, 359-382 (1969).

SELLICK H.; WIDDICOMBE J.G.: Vagal deflation and inflation reflexes mediated by lung irritant receptors. Q. J. Exp. Physiol. 55:153-163 (1970).

SELLICK, H.; WIDDICOMBE, J.G.: Stimulation of lung irritant receptors by cigarette smoke, carbon dust and histamine aerosol. J. Appl. Physiol. 31, 15-19 (1971).

SEVERINGHAUS, J.W.; LARSON, C.P.: Respiration in anesthesia, p.1219-1264; in FENN, W.O.; RAHN, H.: Handbook of Physiology. Respiration, vol.2, sect. 3, p.925-1698 (American Physiological Society, Washington 1965).

SHERREY, J.H.; MEGIRIAN, D.: Spontaneous and reflexly evoked laryngeal abductor and adductor muscle activity of cat. Exp. Neurol. 43 :487-498 (1974).

SHERREY J.H.; MEGIRIAN, D.: Analysis of the respiratory role of intrinsic laryngeal motoneurons of cat. Exp. Neurol : 49:456-465 (1975).

SICA, A.L.; COHEN, M.I.; DONNELLY, D.F.; ZHANG, H.:

Responses of recurrent laryngeal motoneurones to changes of pulmonary afferent inputs. Respir. Physiol. 62: 153-168 (1985).

SPANN, R.W.; HYATT, R.E.: Factors affecting upper airway resistance in conscious man. J. Appl. Physiol. 31:708-12 (1971).

STRANSKY, A.; SZEREDA-PRZESTASZEWSKA, M.; WIDDICOMBE J.G.: The effects of lung reflexes on laryngeal resistance and motoneurone discharge. J. Physiol. 231:417-438 (1973).

SUZUKI, M.; KIRCHNER, J.A.: The posterior cricoarytenoid as an inspiratory muscle. Am. Otol. Rhinol. Laryngol. 78: 849-864 (1969).

SZEREDA-PRZESTASZEWSKA, M.: Activity of vagal respiratory motoneurones in anaphylactic shock in rabbits. Acta allerg. 26, 17-38 (1971).

SZEREDA-PRZESTASZEWSKA, M.; WIDDICOMBE, J.G.: The effect of intravascular injections of veratrine on laryngeal resistance to airflow in cats. Q. Jl. Exp. Physiol. 58:379-385 (1973).

TONKIN, J.P.; AND HARRISON, G.A.: The Surgical Management of the Laryngeal Complications of Prolonged Intubation. Laryngoscope 81:297-307 (1971).

TRENCHARD, D.; GARDENER, D.; GUZ, A.: Role of pulmonary vagal afferent nerve fibres in the development of rapid shallow breathing in lung inflammation. Clin. Sci, 42:251-263 (1972).

TRIPPENBACH, T.; MILIC-EMILI, J.: Vagal contribution to the inspiratory "off-switch" mechanism. Federation Proc. 36 (10): 2375-2380 (1977).

WEBER, H.H.: Radiologische Exploration des Hustenaktes. I. Fortschr. Rontgenstr. 90: 452-474 (1959) cited by KORPAS, J.; TOMORI, Z. In "Cough and Other Respiratory Reflexes". p.47-63. Basel: Karger, 1979.

WHITFIELD, I.C.: An introduction to electronics for physiological workers. 2nd ed. p.118-125. Macmillan Co. Ltd., London (1959).

WIDDICOMBE, J.G.: Respiratory reflexes from the trachea and bronchi of the cat. J. Physiol. 123: 55-70 (1954).

WIDDICOMBE, J.G.: Receptors in the trachea and bronchi of the cat. J. Physiol. 123:71-104 (1954a).

WIDDICOMBE, J.G.: Respiratory reflexes excited by inflation of the lungs. J. Physiol. 123:105-115 (1954b).

WIDDICOMBE, J.G.: Respiratory reflexes in man and other mammalian species. Clin. Sci. 21:163-170 (1961).

WIDDICOMBE, J.G.: Respiratory reflexes. In Handbook of Physiology, Section 3:"Respiration". Fen W.O; Rahn H. eds; Vol 1, p.583-630. Am. Physiol. Soc., Washington D.C (1964).

WIDDICOMBE, J.G.: Reflex control of breathing, p.274-301; in WIDDICOMBE, J.G. (Ed.): Respiratory physiology, vol.2, p.381 (Butterworths, London; University Park Press, Baltimore 1974).

WIDDICOMBE, J.G.; DAVIES, A.: Respiratory physiology. Physiological Principles in Medicine series, Arnold (1983).

WRIGHT, P.G.: An analysis of the central and peripheral components of respiratory failure produced by anticholinesterase poisoning in the rabbit. J. Physiol. 126: 52-70 (1954).

YAMASHITA, T.; URABE, K.: Glottisschlupreflex und M. cricoarytaenoideus posterior. Arch. Ohr.-, Nas.-, Kehlk.-Heilk. 177: 39-44 (1960) cited by KORPAS, J.; TOMORI, Z. In "Cough and Other Respiratory Reflexes". p.47-63. Karger, Basel (1979).

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Vagal Modulation of Recurrent Laryngeal Motoneurone Discharge

Volume II

Fig. 1 The Cartilages of the Rabbit's Larynx (After Barone, 1976)

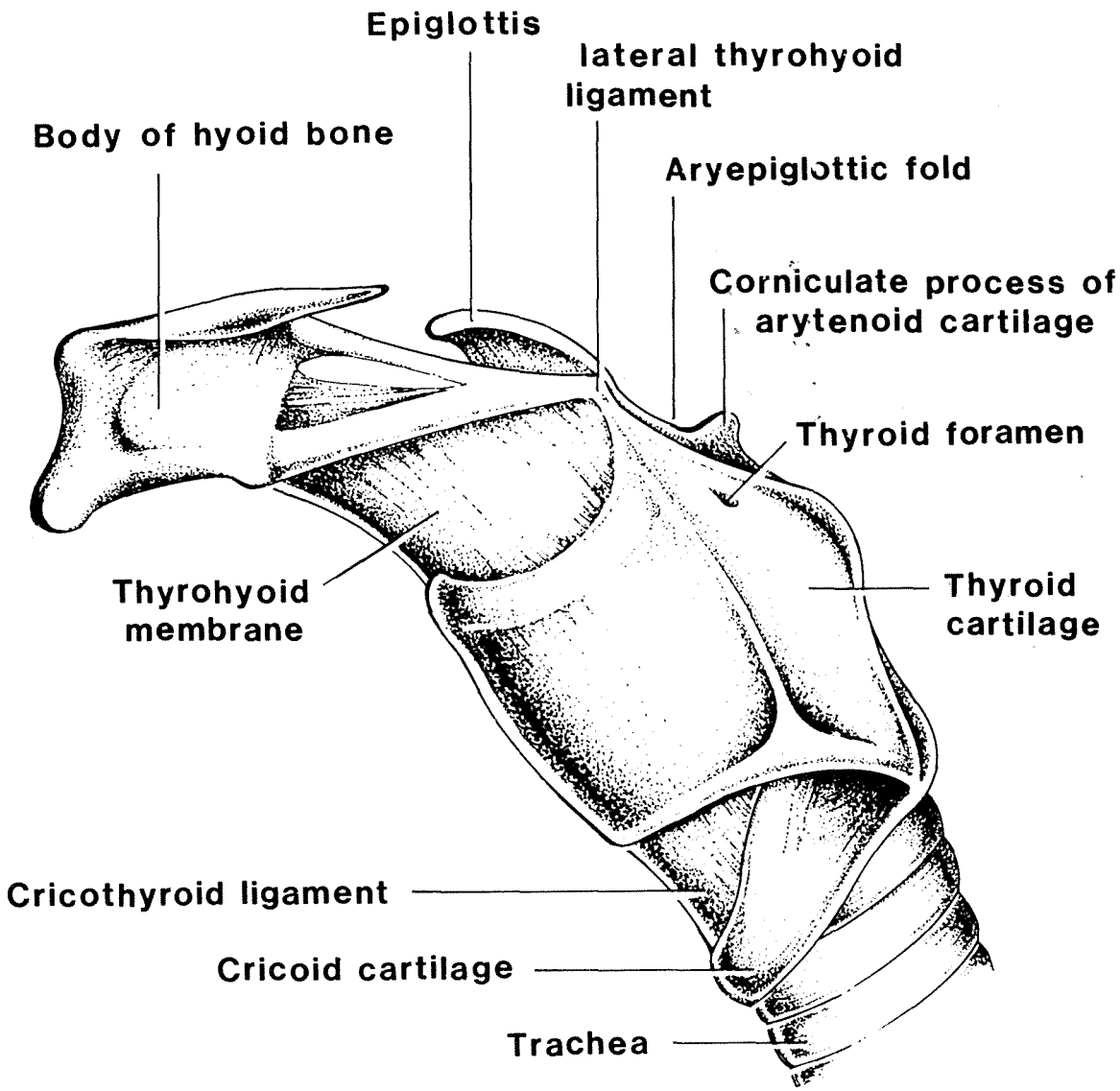

Epiglottis
lateral thyrohyoid ligament
Body of hyoid bone
Aryepiglottic fold
Corniculate process of arytenoid cartilage
Thyroid foramen
Thyrohyoid membrane
Thyroid cartilage
Cricothyroid ligament
Cricoid cartilage
Trachea

Fig. 2 The Intrinsic Laryngeal Muscles of the Rabbit (After Barone, 1976)

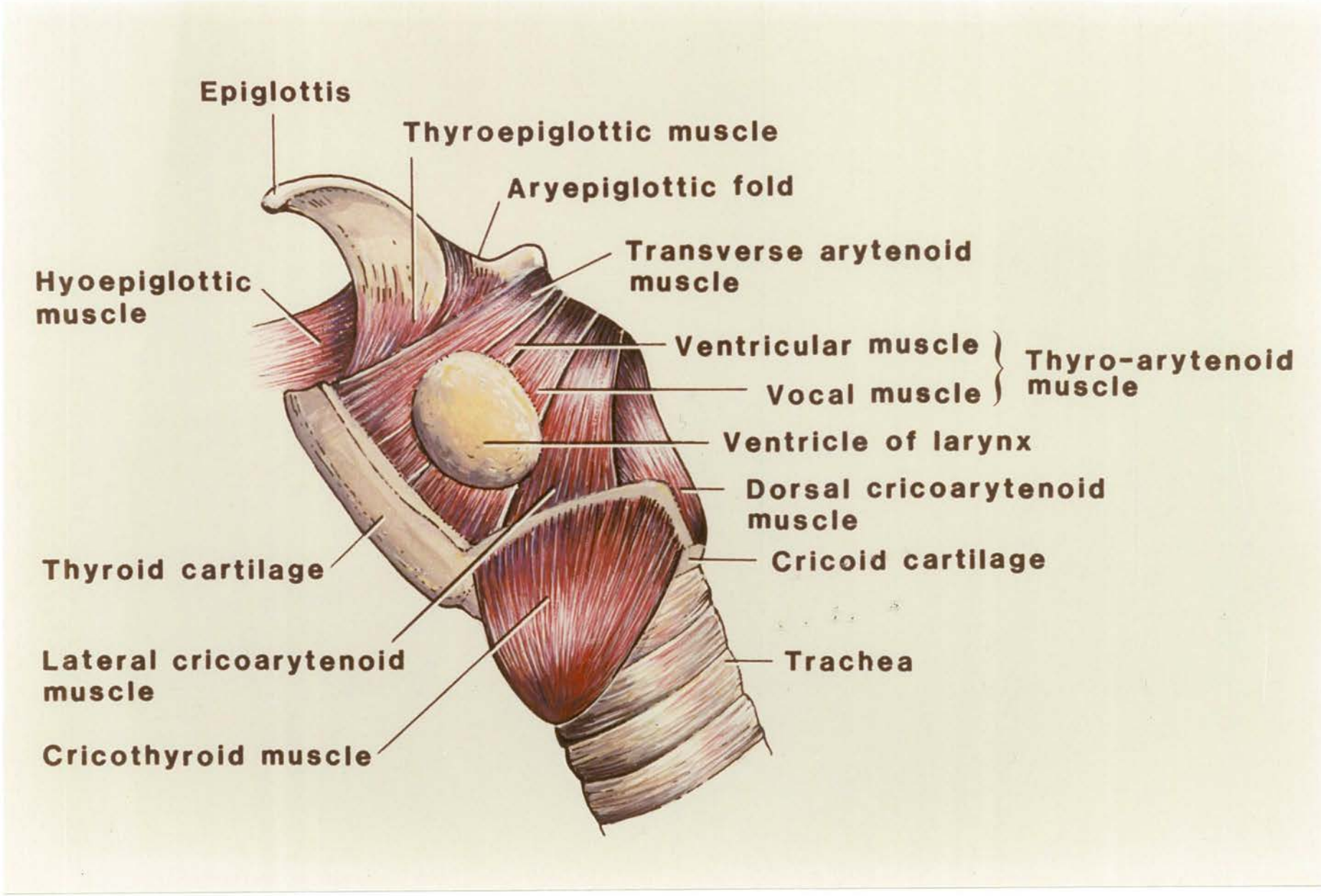
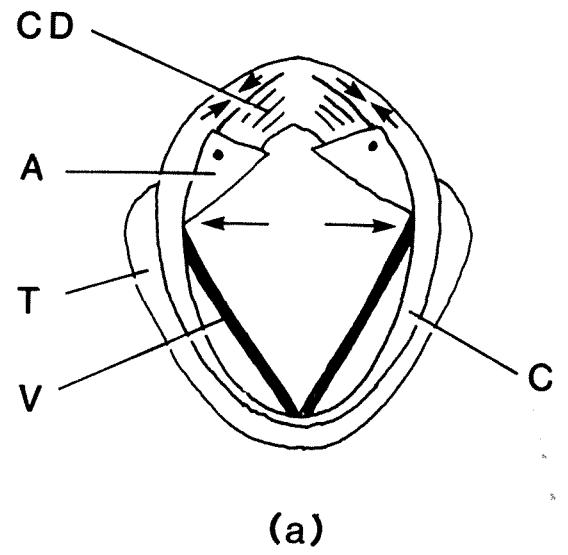
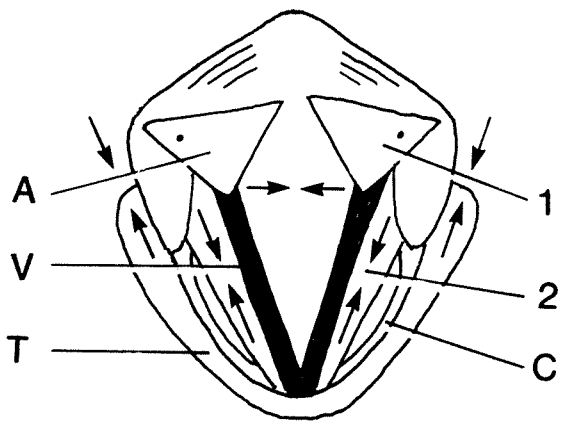

Epiglottis
Thyroepiglottic muscle
Aryepiglottic fold
Transverse arytenoid muscle
Hyoepiglottic muscle
Ventricular muscle
Vocal muscle
Thyro-arytenoid muscle
Ventricle of larynx
Dorsal cricoarytenoid muscle
Cricoid cartilage
Thyroid cartilage
Lateral cricoarytenoid muscle
Trachea
Cricothyroid muscle

Fig. 3 Scheme of action of laryngeal muscles on vocal cords



(a)



(b)

After Miller (1952)

a Action of the dorsal cricoarytenoid muscles as abductors of the vocal cords

b Action of the lateral intrinsic muscles as adductors of the vocal cords

A Arytenoid cartilage
C Cricoid cartilage
T Thyroid cartilage
V Vocal cord

CD Dorsal cricoarytenoid muscle
1 Lateral cricoarytenoid muscle
2 Thyroarytenoid muscle

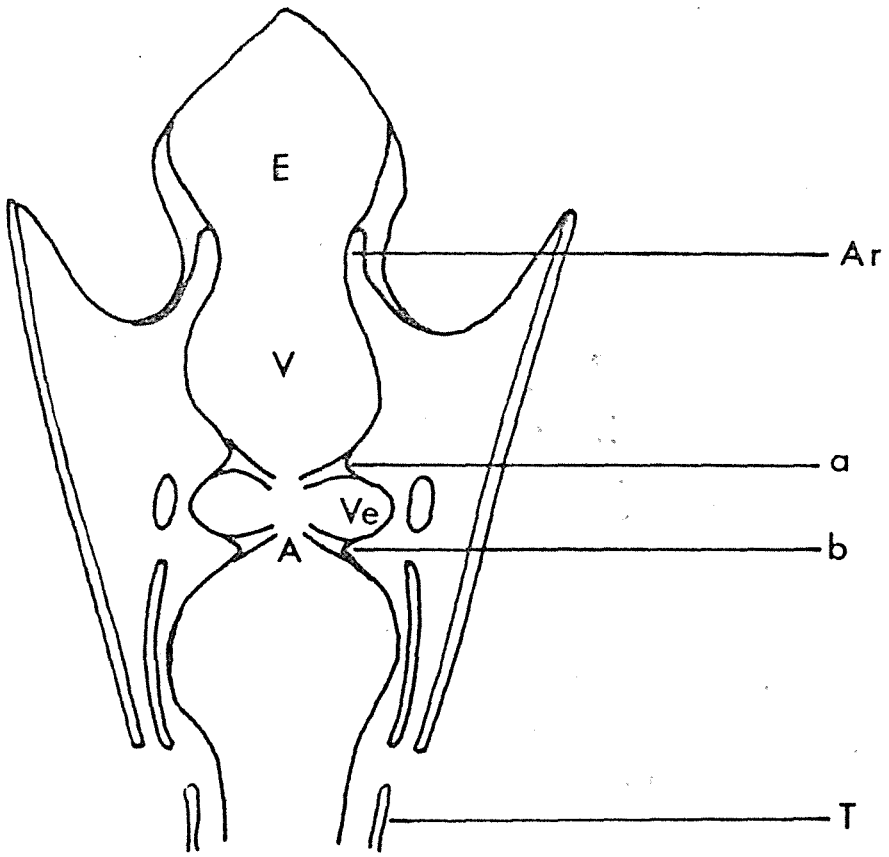


Fig. 4

A schematic diagram of a frontal section of composite mammalian larynx to show the main features (After Rex, 1970)

A Glottis
Ar Aryepiglottic folds
T First tracheal cartilage
Ve Ventricle
a False cords
b True vocal cords
V Vestibule
E Epiglottis

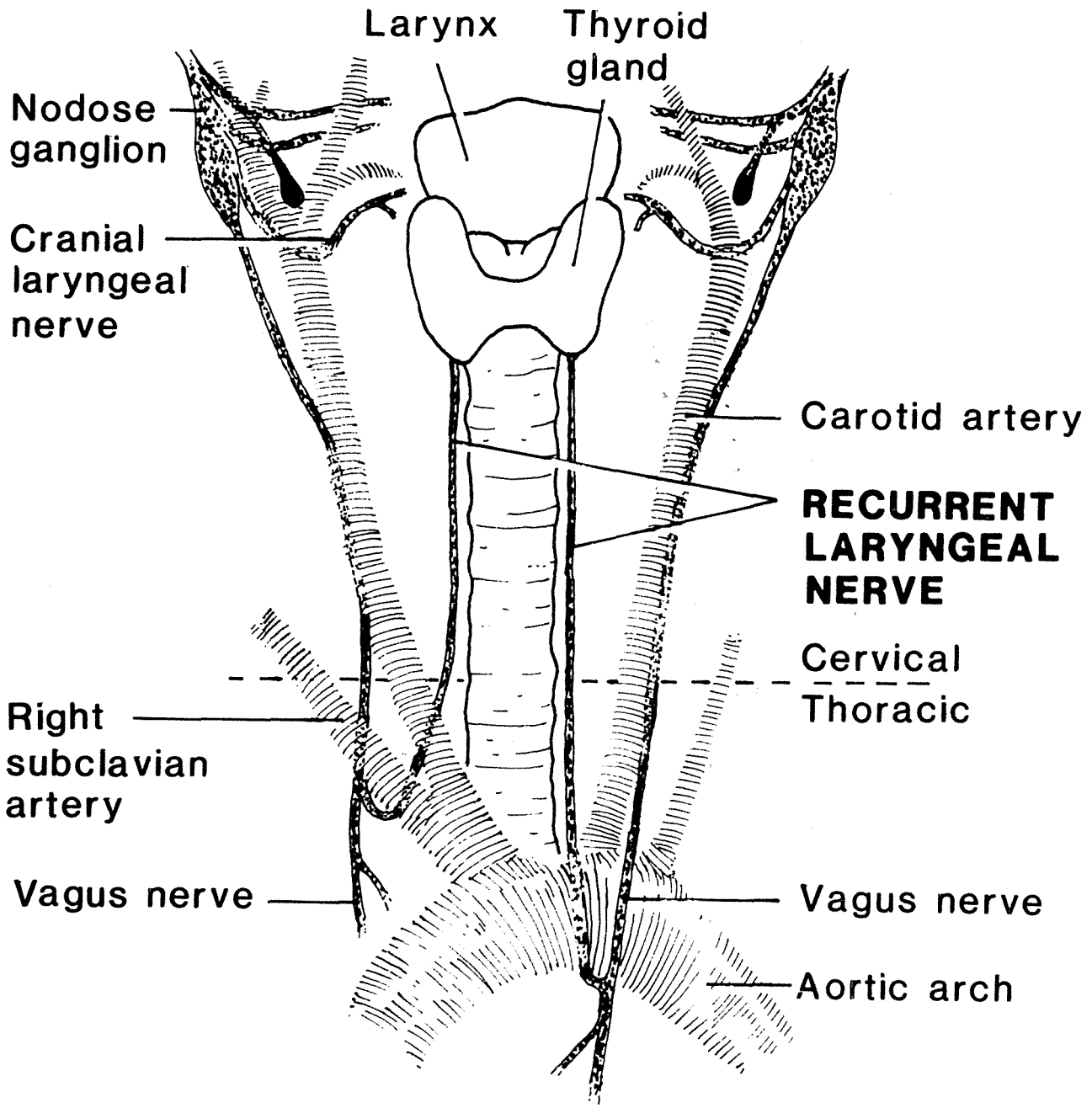


Fig. 5 Schematic drawing demonstrating course of right and left recurrent laryngeal nerves

Fig. 6 Distribution of the recurrent laryngeal and cranial laryngeal nerves. Lamina of the thyroid cartilage was cut and reflected (After McClure, 1964).

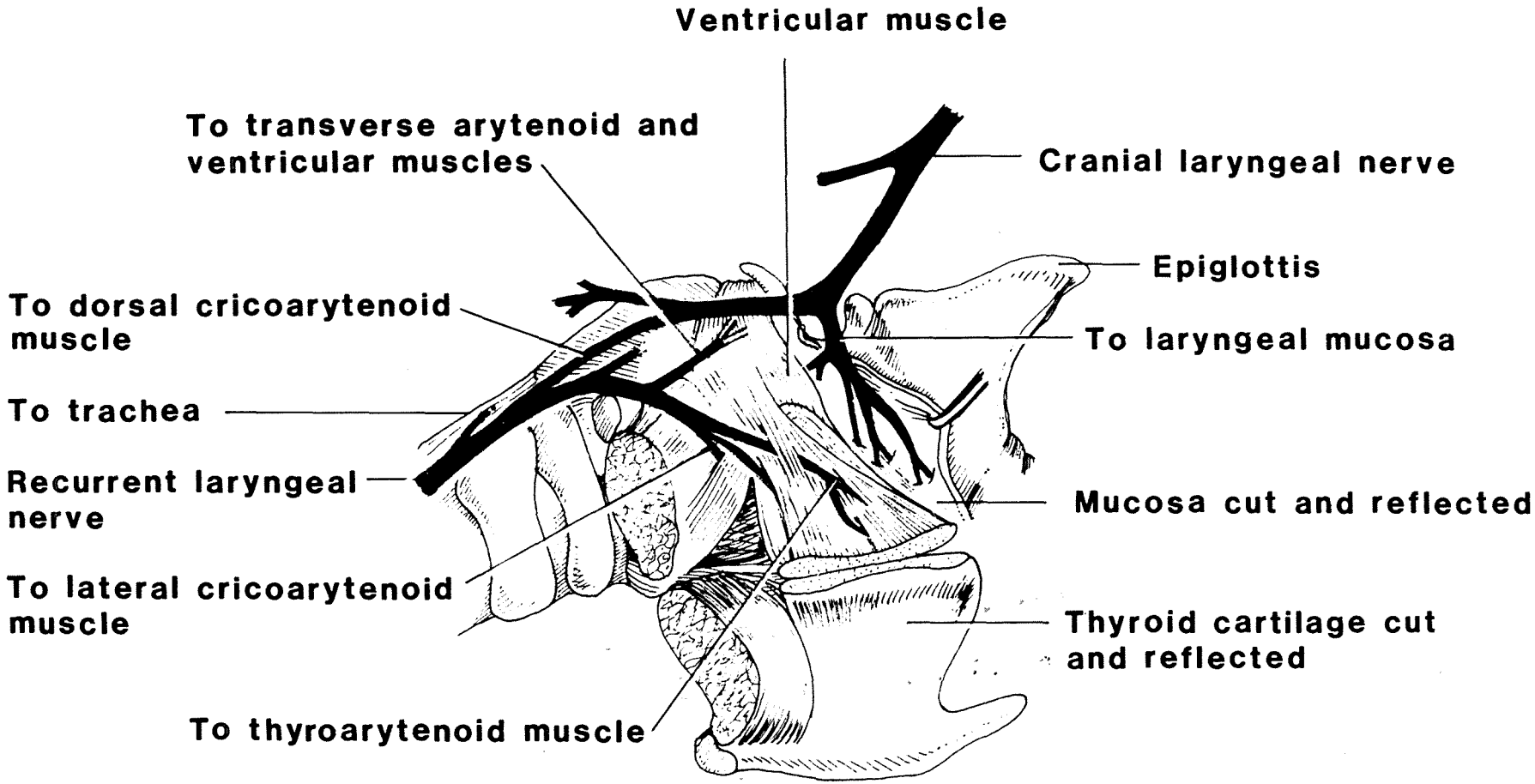
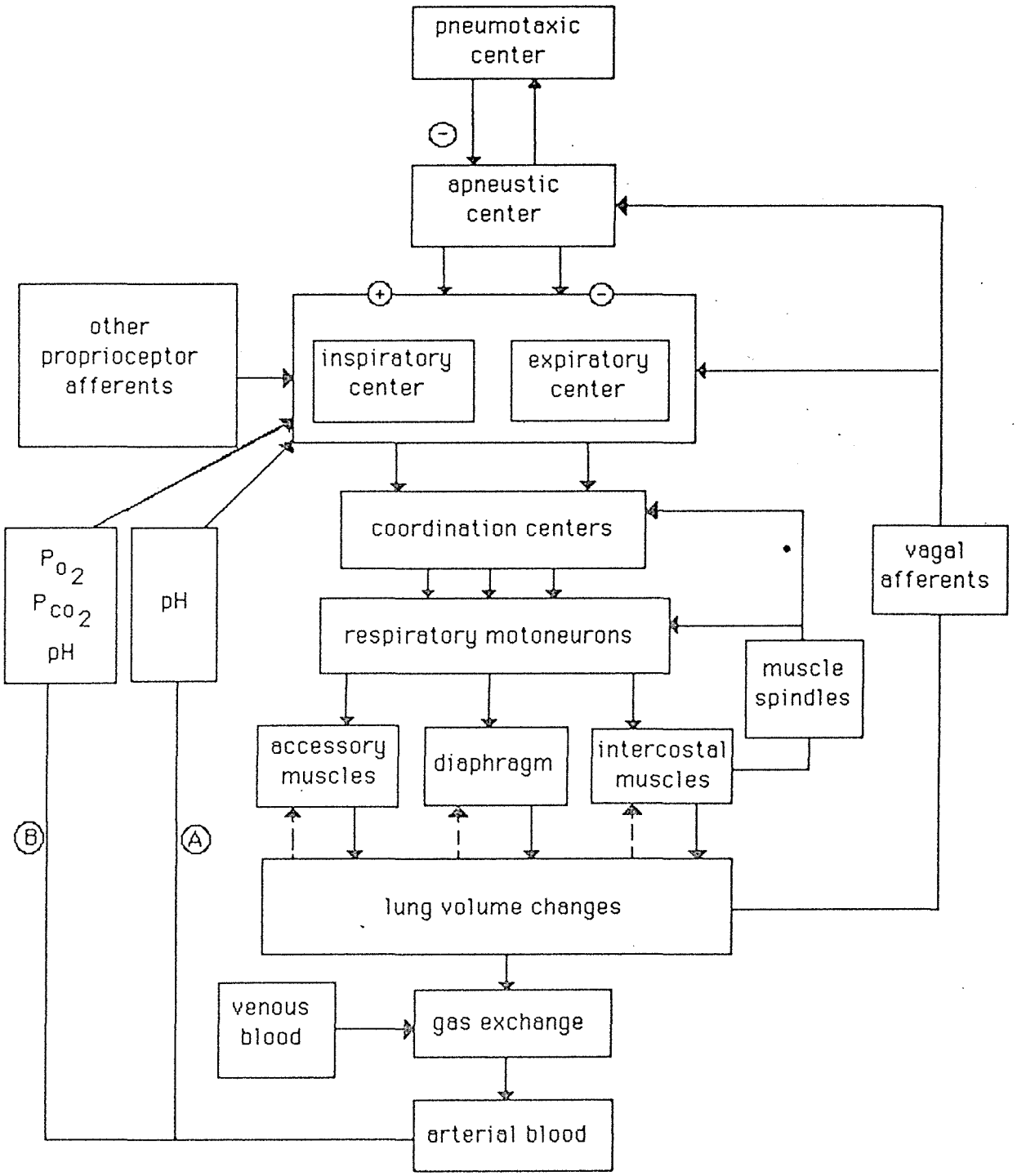

Ventricular muscle
To transverse arytenoid and ventricular muscles
Cranial laryngeal nerve
Epiglottis
To dorsal cricoarytenoid muscle
To laryngeal mucosa
To trachea
Recurrent laryngeal nerve
Mucosa cut and reflected
To lateral cricoarytenoid muscle
Thyroid cartilage cut and reflected
To thyroarytenoid muscle

Fig. 7 Main functional components of the control system and the breathing apparatus. Facilitatory (+) and inhibitory (-) effects in the brainstem shown where appropriate. Dashed arrows indicate mechanical effect of lung volume changes on muscles. A : central chemoreceptors; B : peripheral chemoreceptors. (After Bouhuys, 1977)


pneumotaxic center
apneustic center
other proprioceptor afferents
inspiratory center
expiratory center
coordination centers
vagal afferents
P_{O_2}
P_{CO_2}
pH
pH
respiratory motoneurons
muscle spindles
accessory muscles
diaphragm
intercostal muscles
B
A
lung volume changes
venous blood
gas exchange
arterial blood

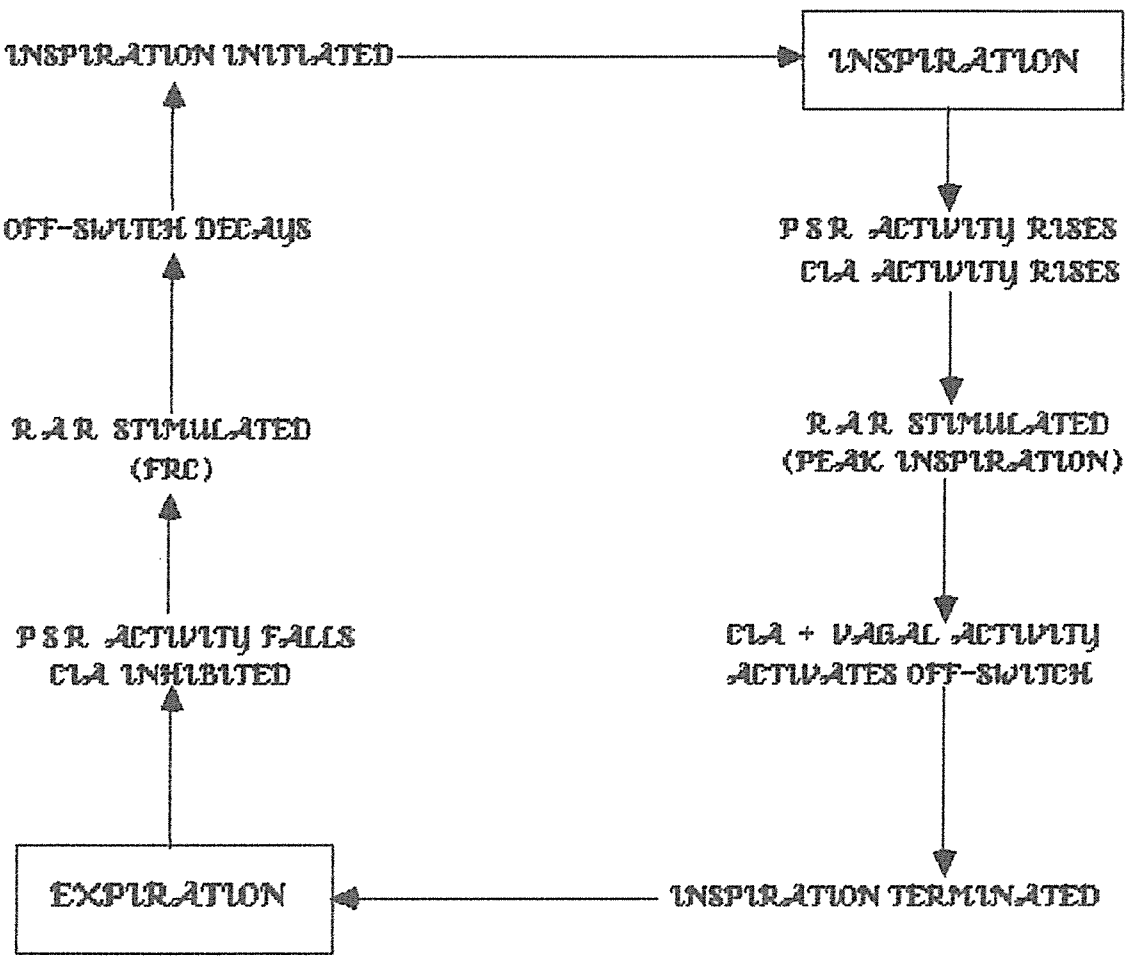


Fig. 8 Receptor influence on the phases of breathing. Alteration of receptor activity changes duration of inspiration or expiration.

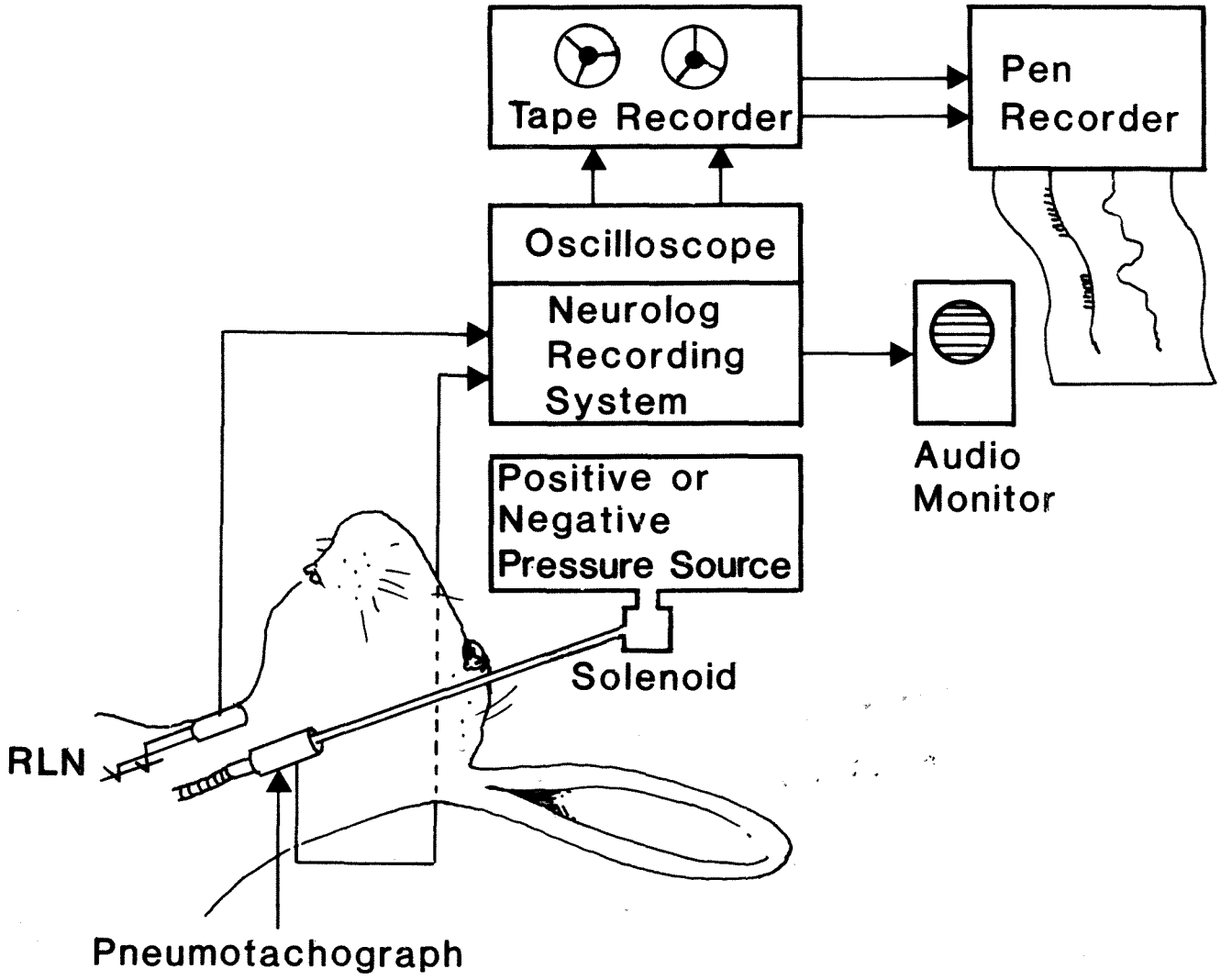


Fig. 9 Schematic outline of experimental setup. RLN: recurrent laryngeal nerve.

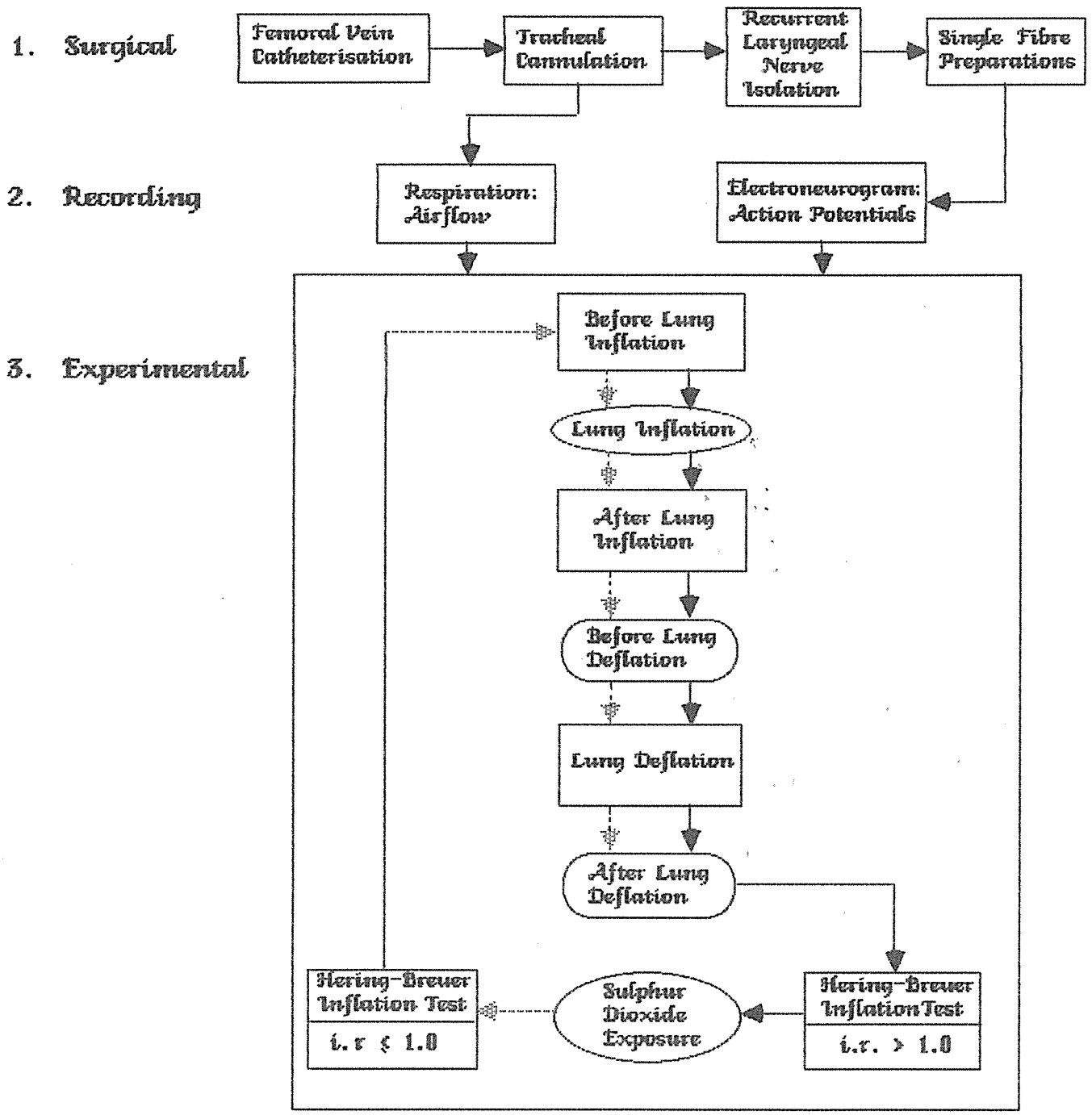


Fig. 10 Experimental protocol. i.r. = Inhibitory Ratio

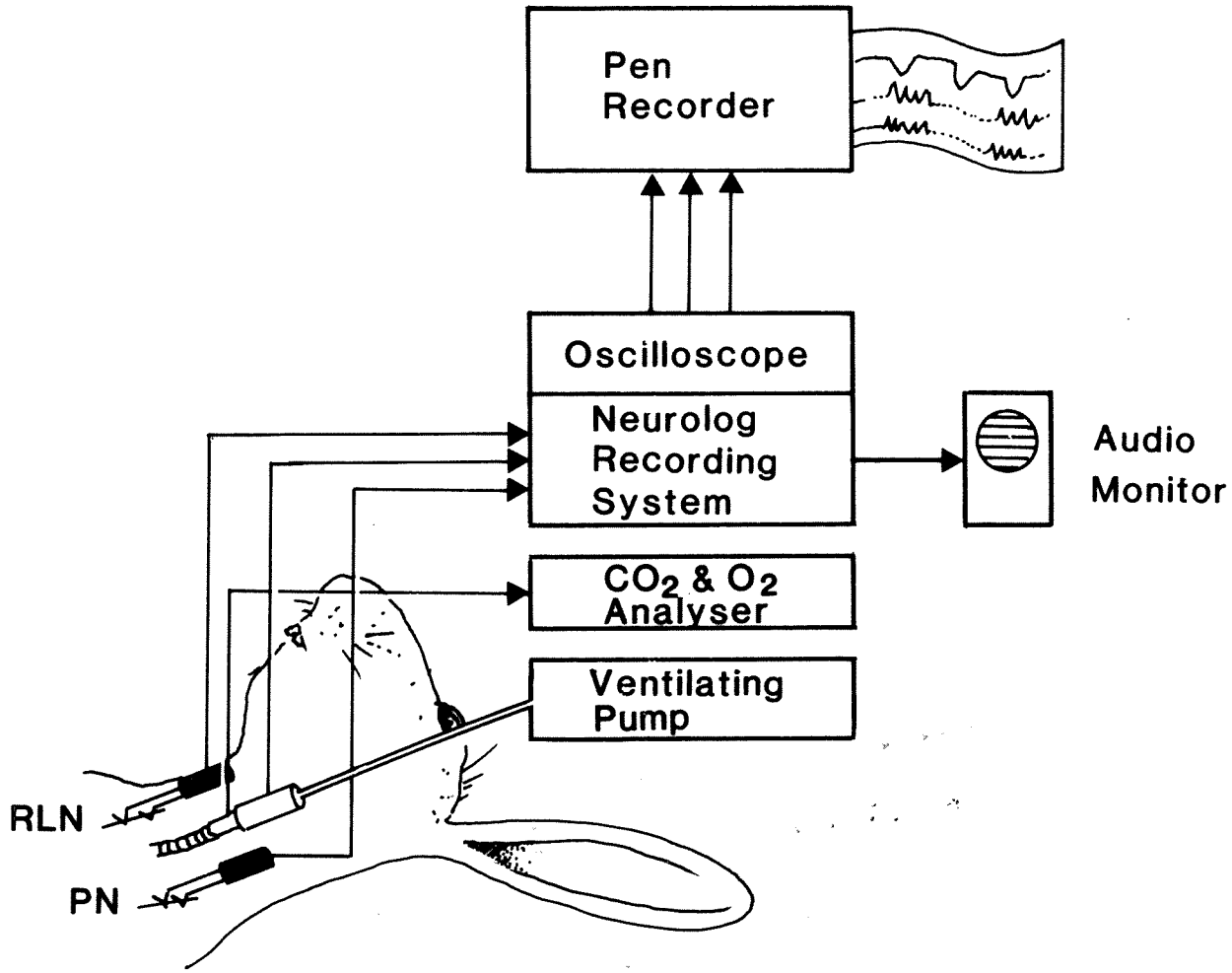


Fig. 11 Neuromuscular junction block series. Diagram of experimental set up. RLN: recurrent laryngeal nerve, PN: Phrenic nerve.

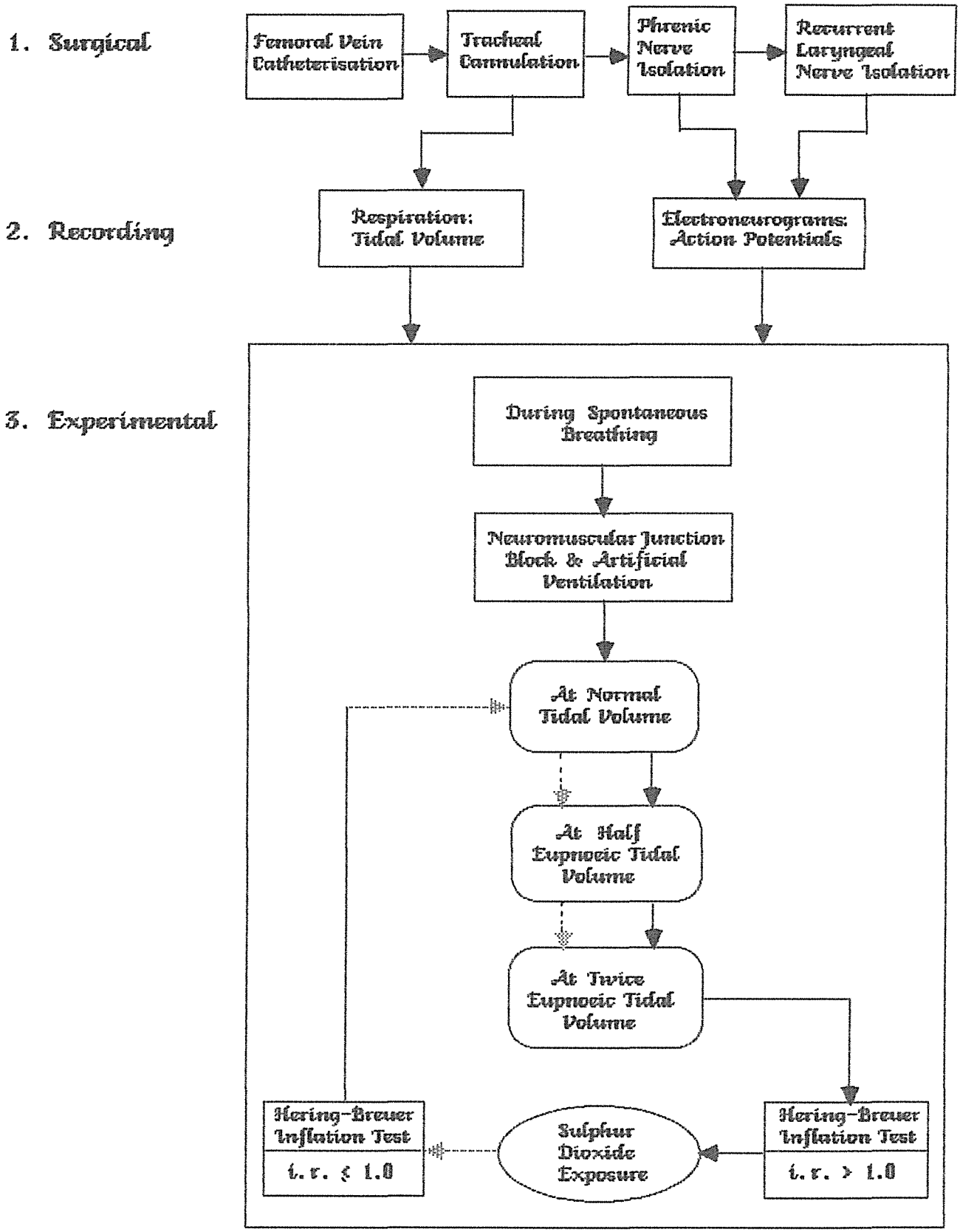


Fig. 12 Experimental Protocol (Neuromuscular Junction Block Series). i.r. = Inhibitory Ratio.

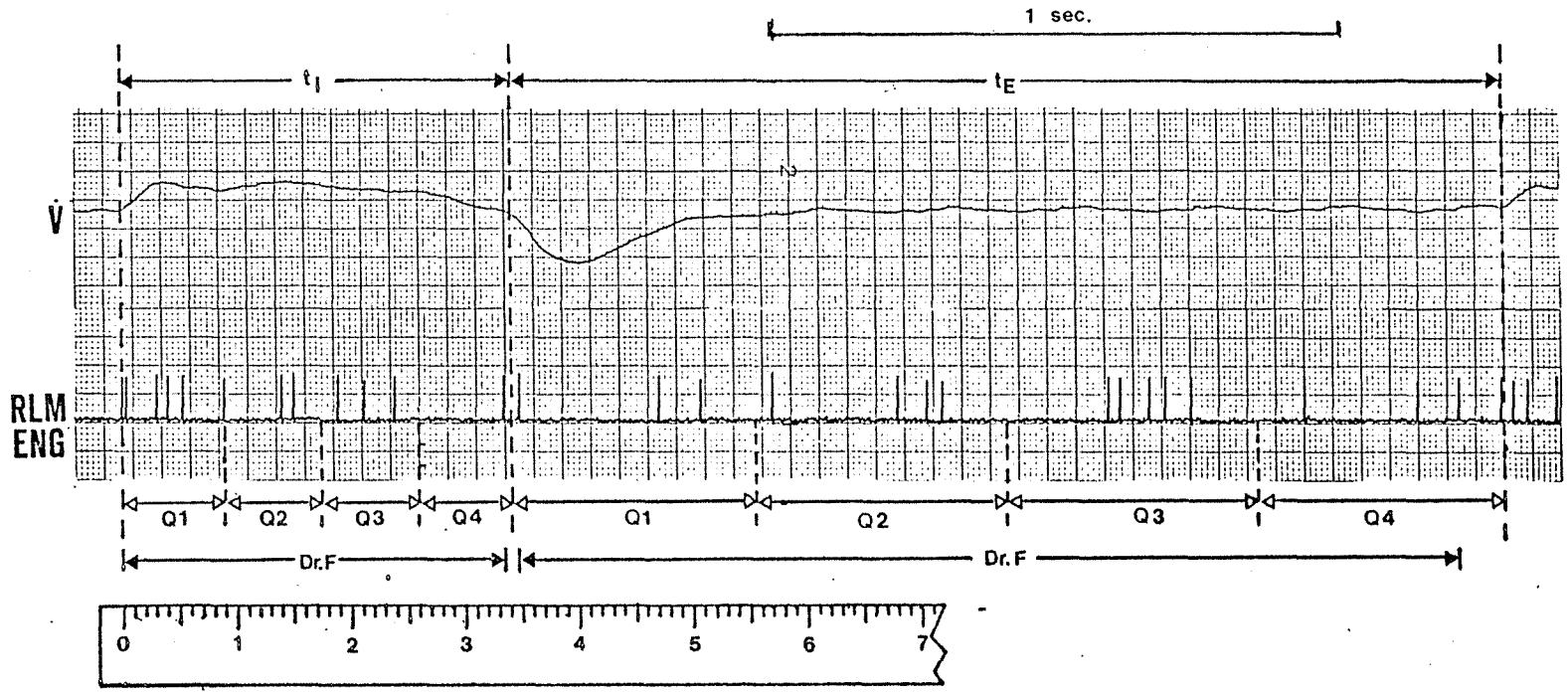


Fig. 13 Schematic diagram showing how the duration of inspiration (tI) and expiration (tE), number of spikes within each quartile (Q1 – Q4) of both respiratory phases and duration of firing (Dr.F) were measured from the airflow ($\dot{V}$) signal (upper trace) and recurrent laryngeal motoneurone (RLM) neurogram (lower trace) records.

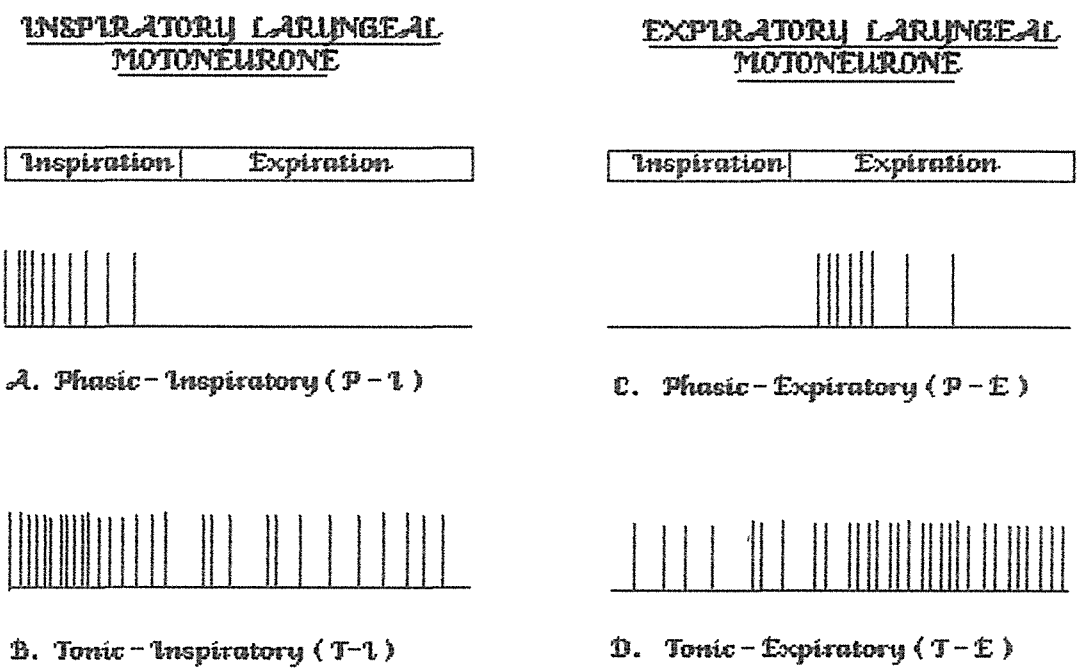


Fig. 14 Schematic classification of major discharge patterns of recurrent laryngeal motoneurones. Each pattern is derived from spike counts of a representative motoneurone.

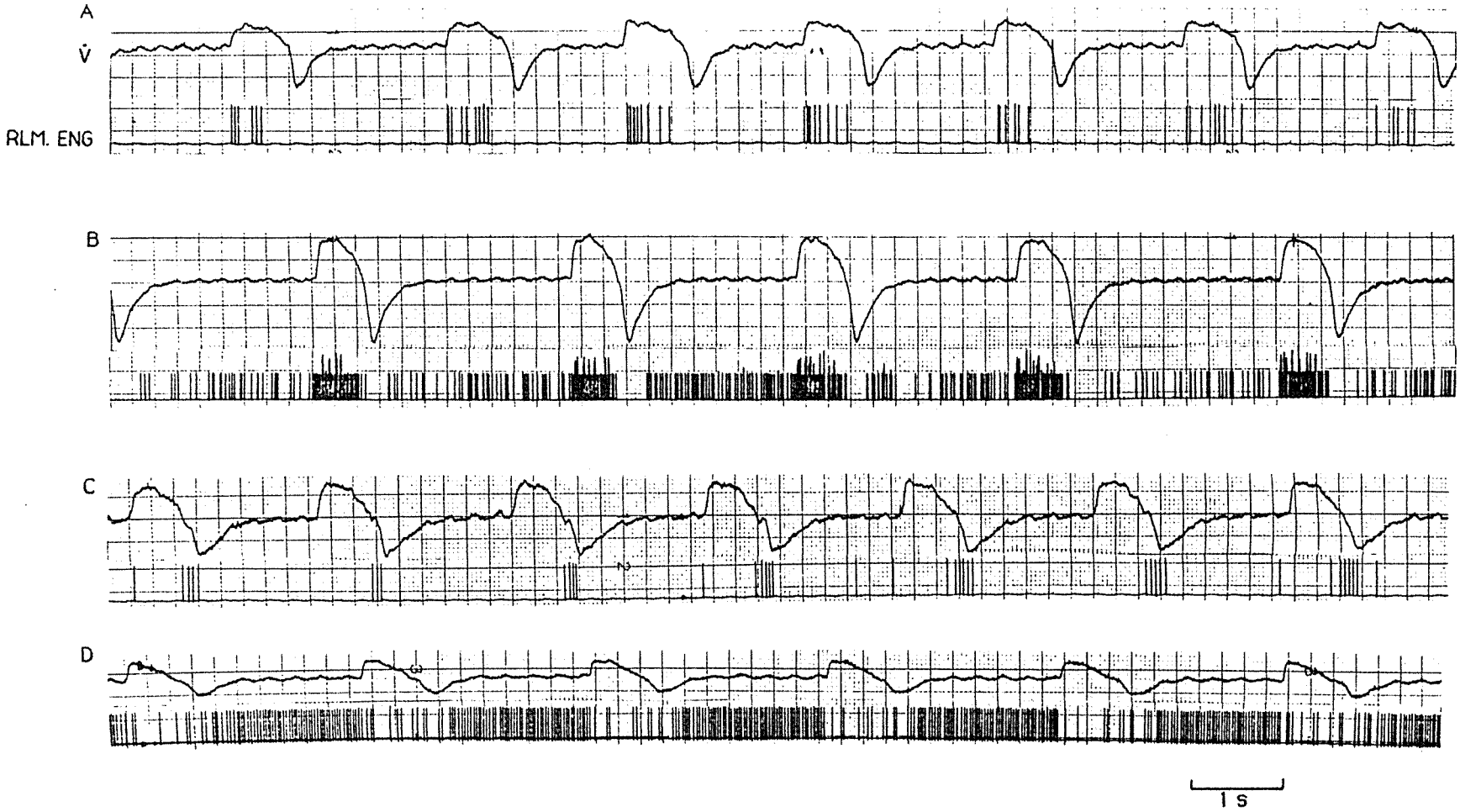


Fig. 15 Spontaneous activity of recurrent laryngeal motoneurones with different patterns of discharge. A: Phasic-inspiratory, B: Tonic-inspiratory, C: Phasic-expiratory, D: Tonic-expiratory. Rabbits under pentobarbitone anaesthesia. In each record, upper trace: airflow ($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM. ENG).

Fig. 16 Histogram showing correlation between impulse frequency of a phasic-inspiratory laryngeal motoneurone and the inspiratory (I.P) and expiratory (E.P) phases of the respiratory cycle. Each of the two phases is divided into 4 equal intervals (quartiles).
Upper trace : Airflow signal
Lower trace : Recurrent laryngeal motoneurone electroneurogram

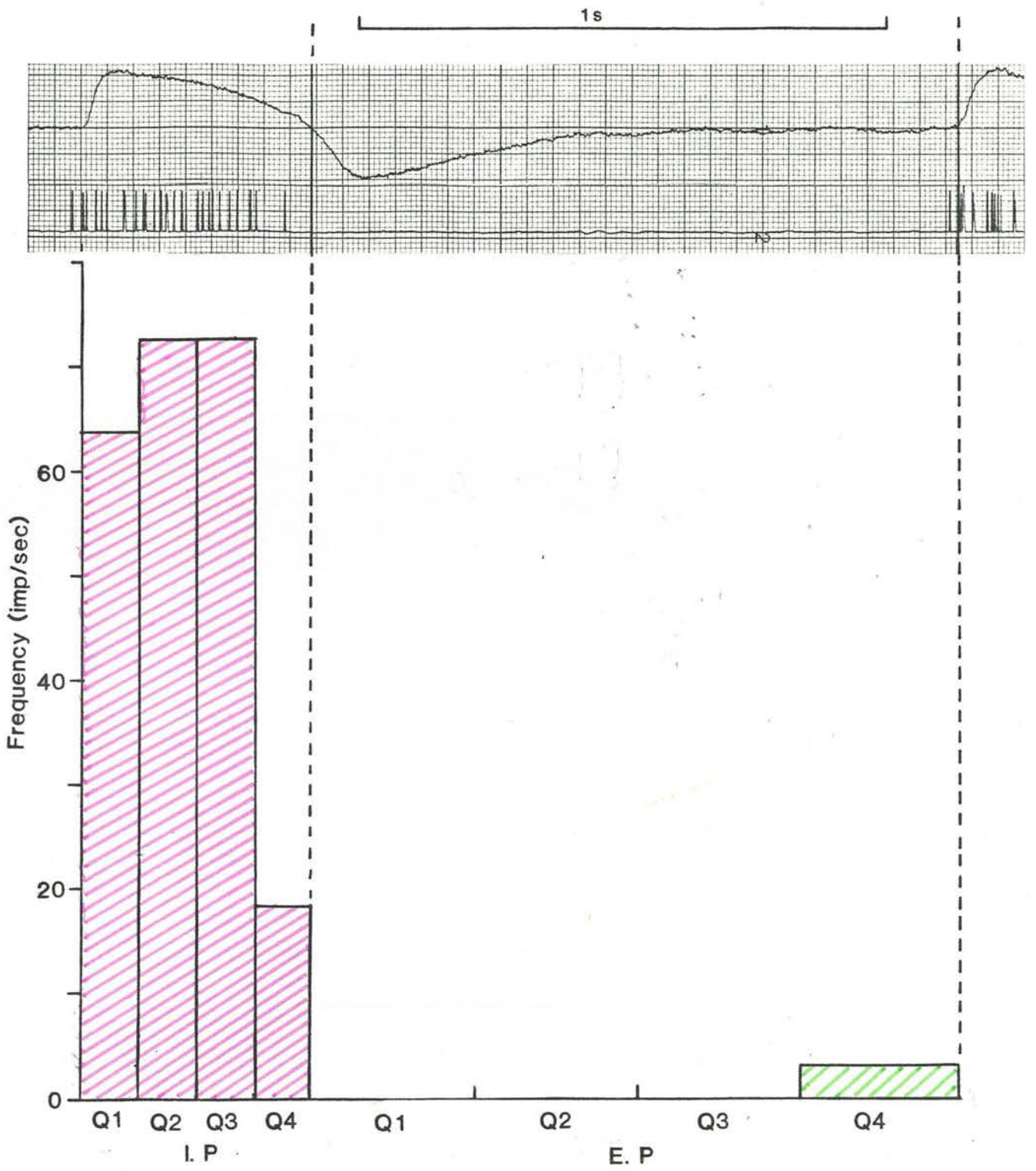

1s
Frequency (imp/sec)
60
40
20
0
Q1
Q2
Q3
Q4
I. P
Q1
Q2
Q3
Q4
E. P

Fig. 17 Histogram showing correlation between the impulse frequency of a tonic - inspiratory laryngeal motoneurone and the inspiratory (I.P) and expiratory (E.P) phases of the respiratory cycle. Each of the two phases is divided into 4 equal intervals (quartiles).
Upper trace : Airflow signal
Lower trace : Recurrent laryngeal moto-neurone electroneurogram

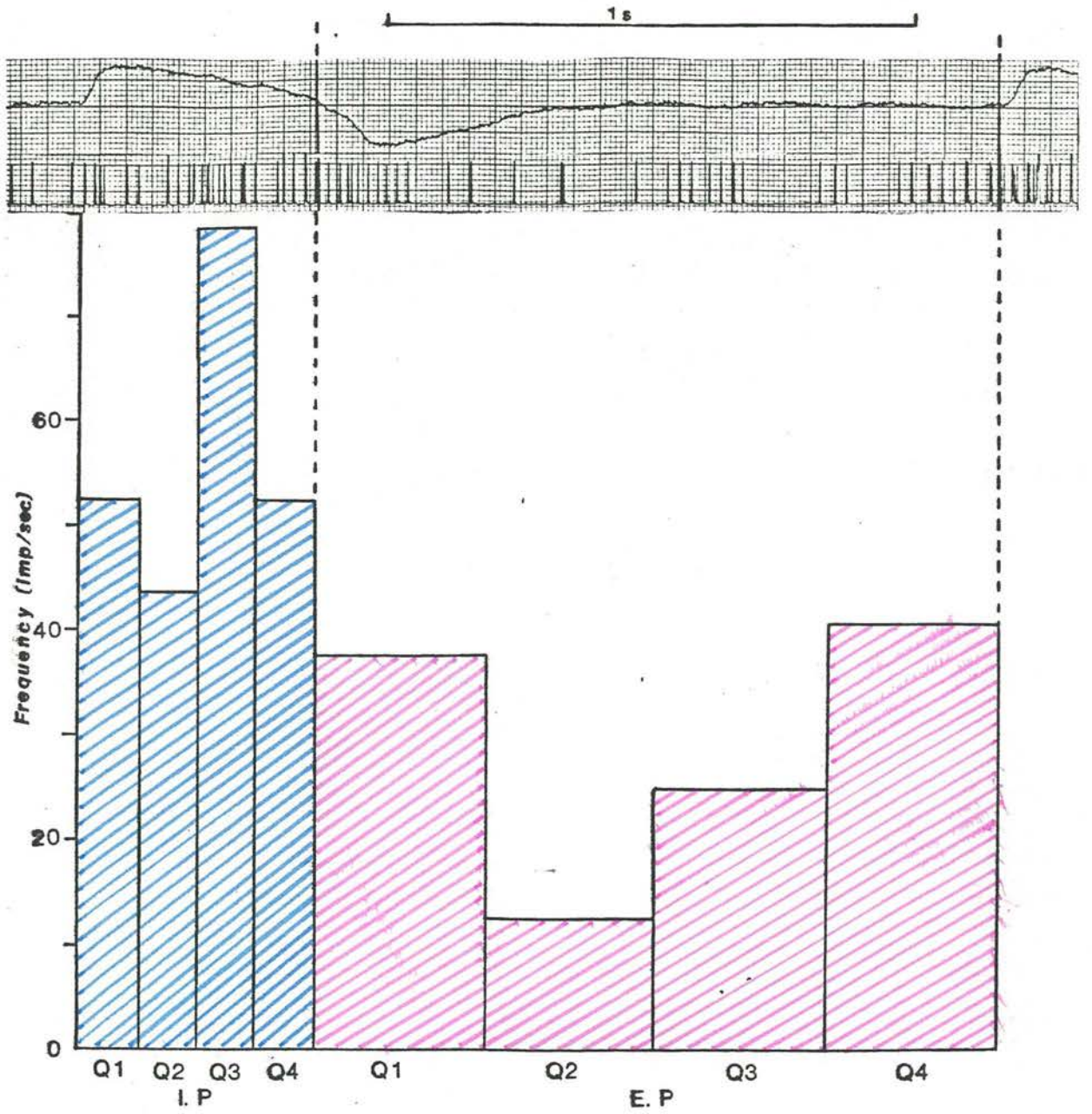

1 s
Frequency (Imp/sec)
60
40
20
0
Q1
Q2
Q3
Q4
I. P
Q1
Q2
Q3
Q4
E. P

Fig. 18 Histogram showing correlation between impulse frequency of a phasic-expiratory laryngeal motoneurone and the inspiratory (I.P) and expiratory (E.P) phases of the respiratory cycle. Each of the two phases is divided into 4 equal intervals (quartiles).
Upper trace : Airflow signal
Lower trace : Recurrent laryngeal motoneurone electroneurogram

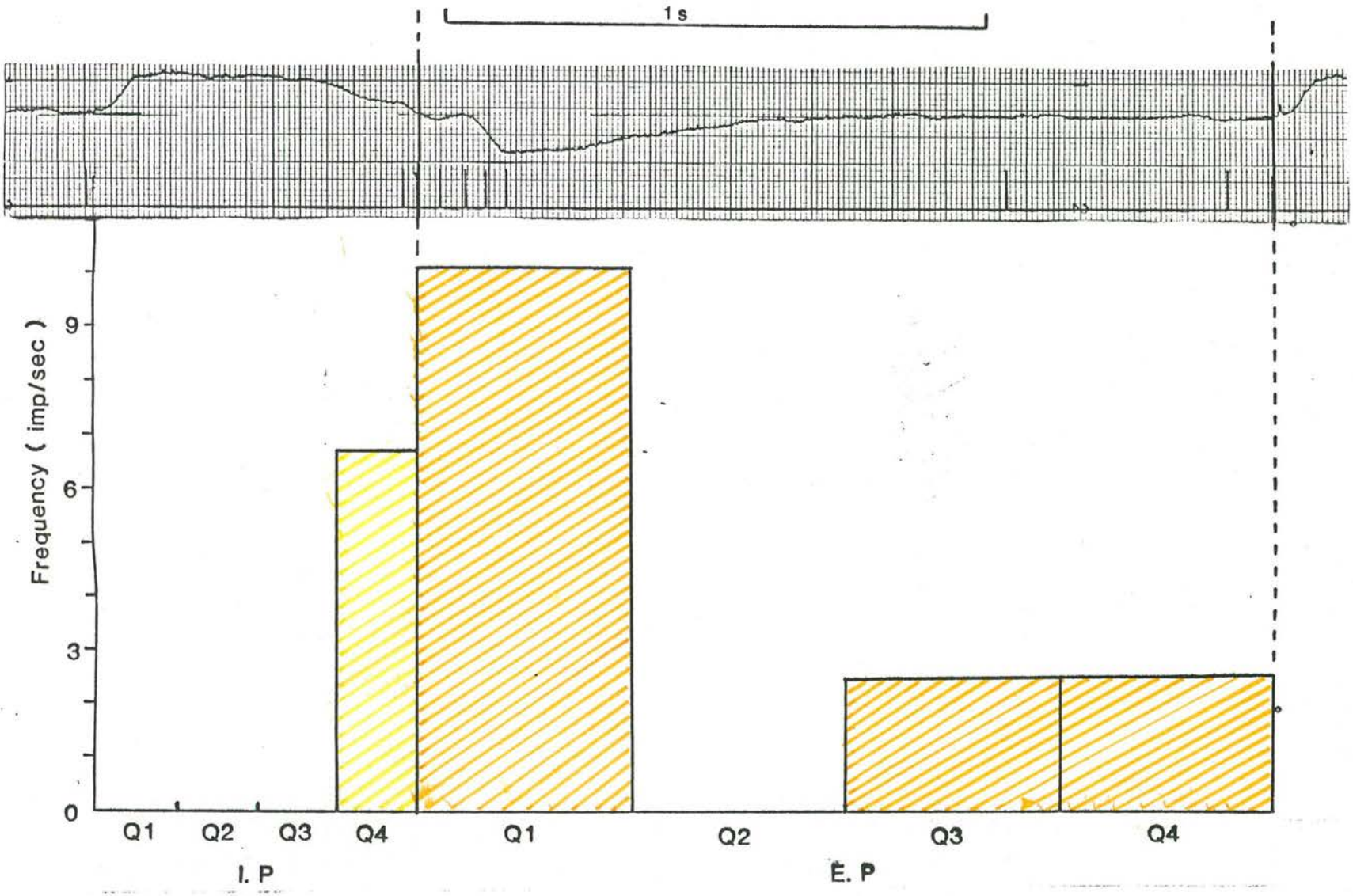

1 s
Frequency (imp/sec)
9
6
3
0
Q1
Q2
Q3
Q4
Q1
Q2
Q3
Q4
I. P
E. P

Fig. 19 Histogram showing correlation between the impulse frequency of a tonic - expiratory laryngeal motoneurone and the inspiratory (I.P) and expiratory (E.P) phases of the respiratory cycle. Each of the two phases is divided into 4 equal intervals (quartiles).
Upper trace : Airflow signal
Lower trace : Recurrent laryngeal motoneurone electroneurogram

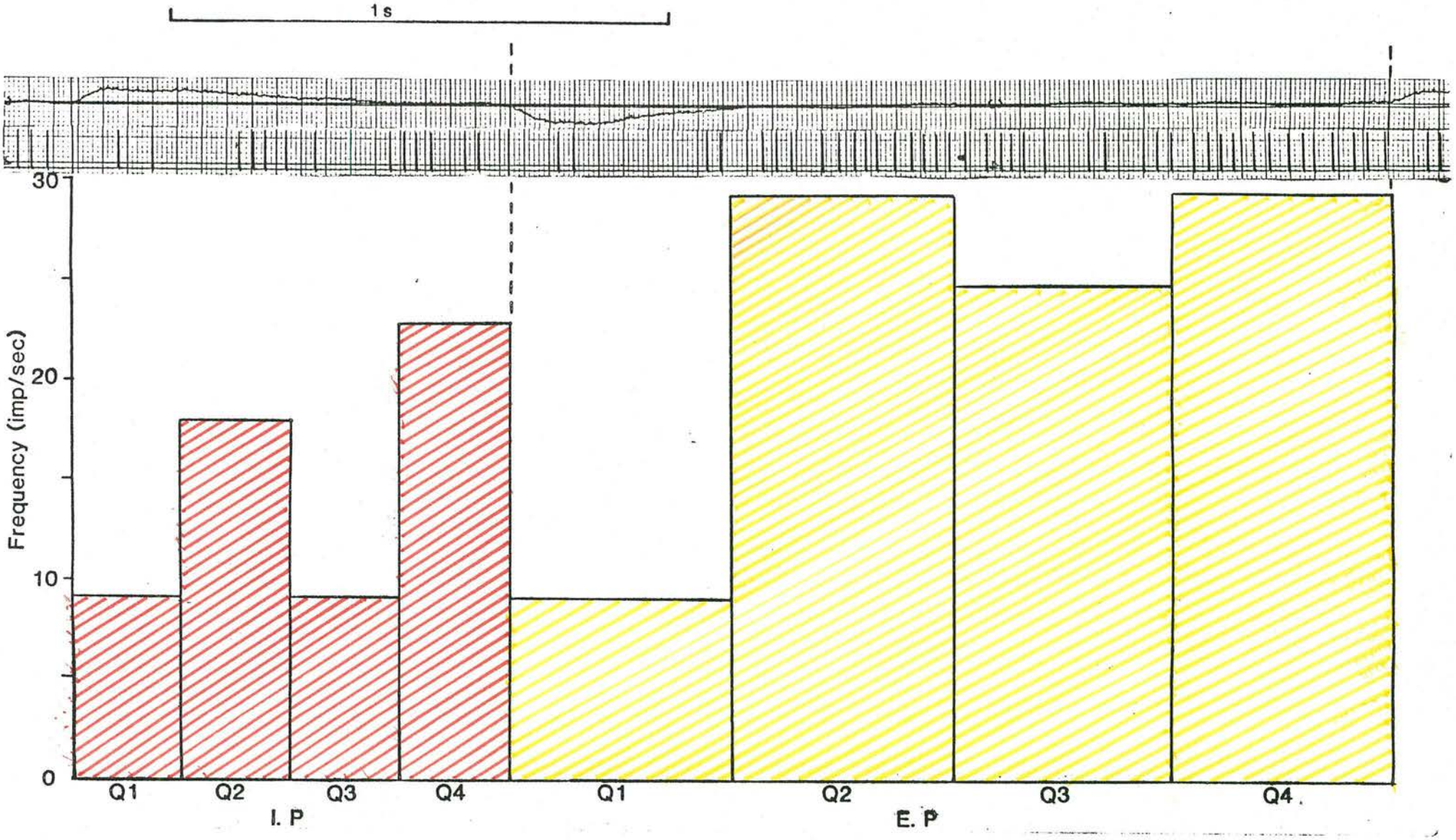
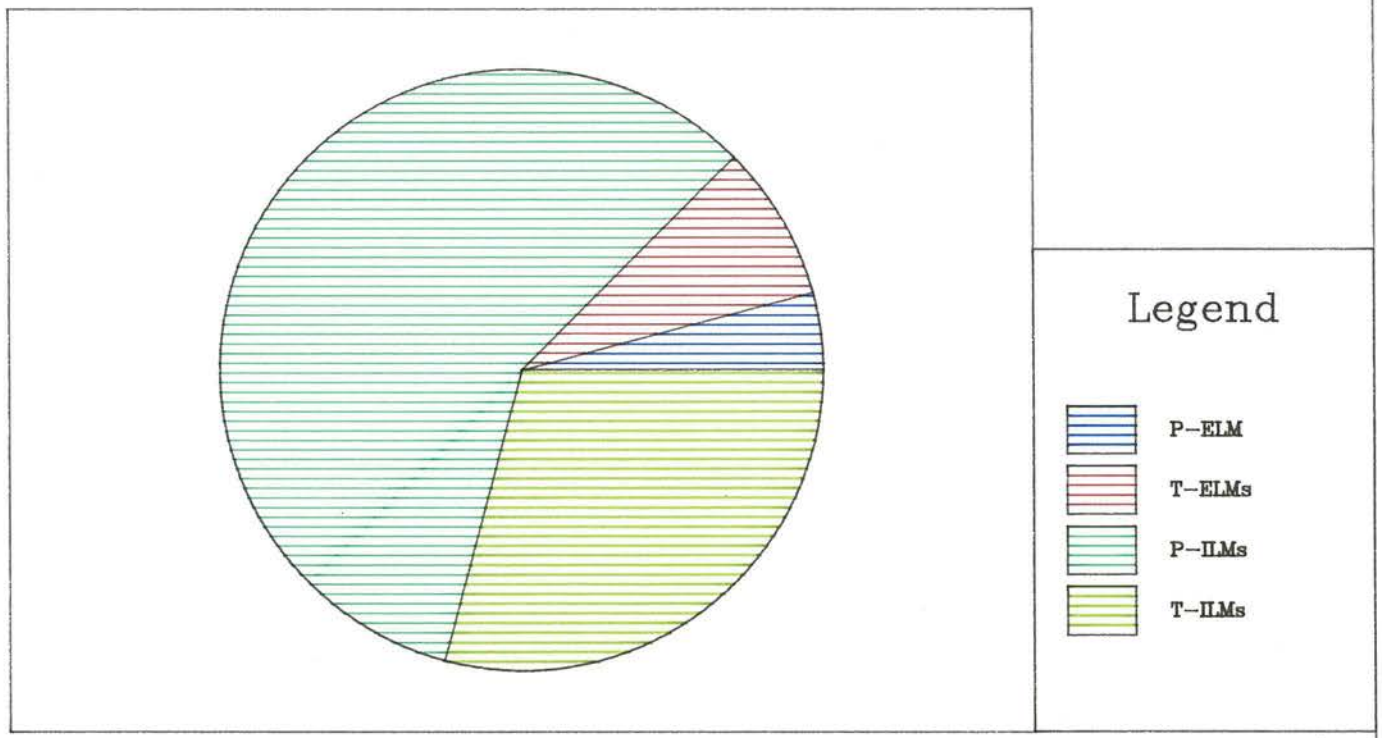

1 s
30
20
10
0
Frequency (imp/sec)
Q1
Q2
Q3
Q4
I. P
Q1
Q2
Q3
Q4
E. P

FIG. 20 FIBRE TYPES IN THE RECURRENT LARYNGEAL NERVE SAMPLES



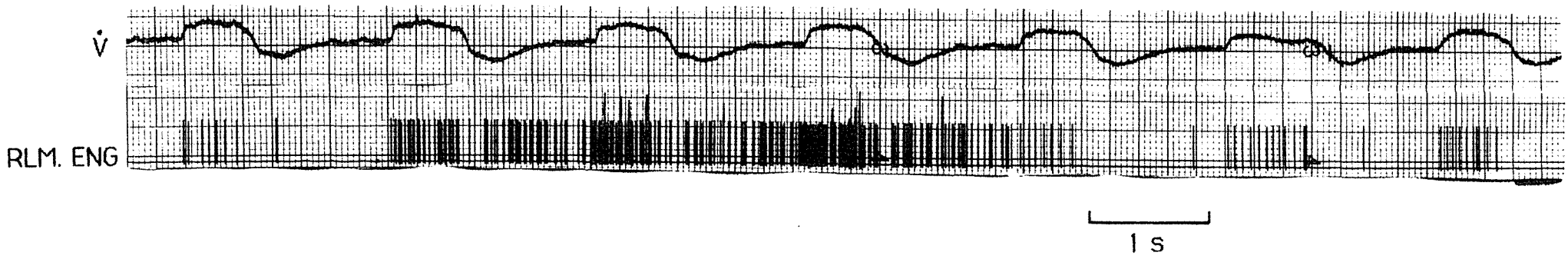


Fig. 21 Record showing the change in discharge pattern of a phasic-inspiratory laryngeal motoneurone during eupnoeic breathing: from a phasic-inspiratory pattern of discharge into a tonic-inspiratory pattern and back to phasic-inspiratory again. Rabbit under pentobarbitone anaesthesia. Upper trace: airflow ($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM.ENG).

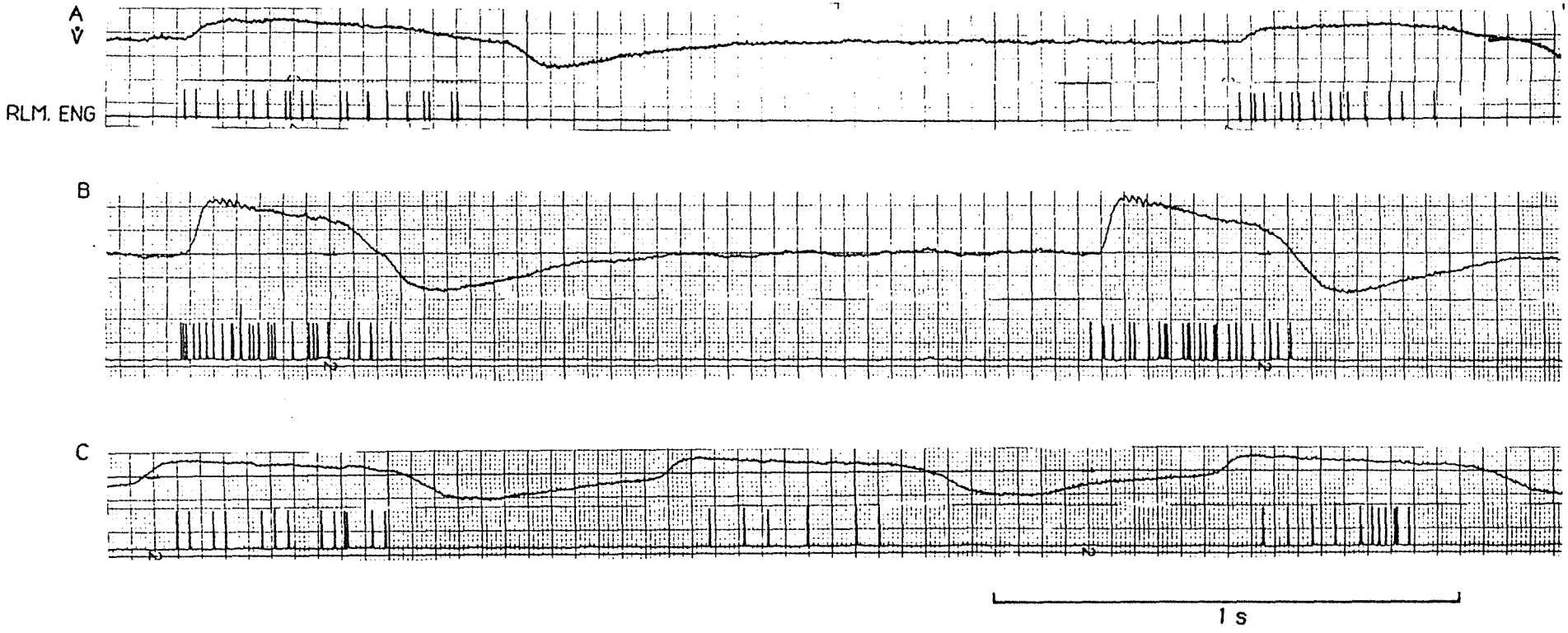


Fig. 22 Records from 3 phasic-inspiratory laryngeal motoneurones showing different times of onset of motoneurone discharge during eupnoeic breathing. A. The onset of motoneurone activity was synchronous with the onset of inspiration. B. The onset of motoneurone activity was earlier than the start of the inspiratory phase. C. The onset of activity was later than the onset of inspiratory phase. In each record, upper trace: airflow ($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM. ENG). Rabbits under pentobarbitone anaesthesia.

Fig. 23 Mean frequency of P-ULM discharge in different quartiles of the inspiratory (I.P) and expiratory (E.P) phases of the respiratory cycle during eupnoeic breathing (N=14 fibres). Vertical bars represent standard errors.

Inspiratory discharge

Expiratory discharge

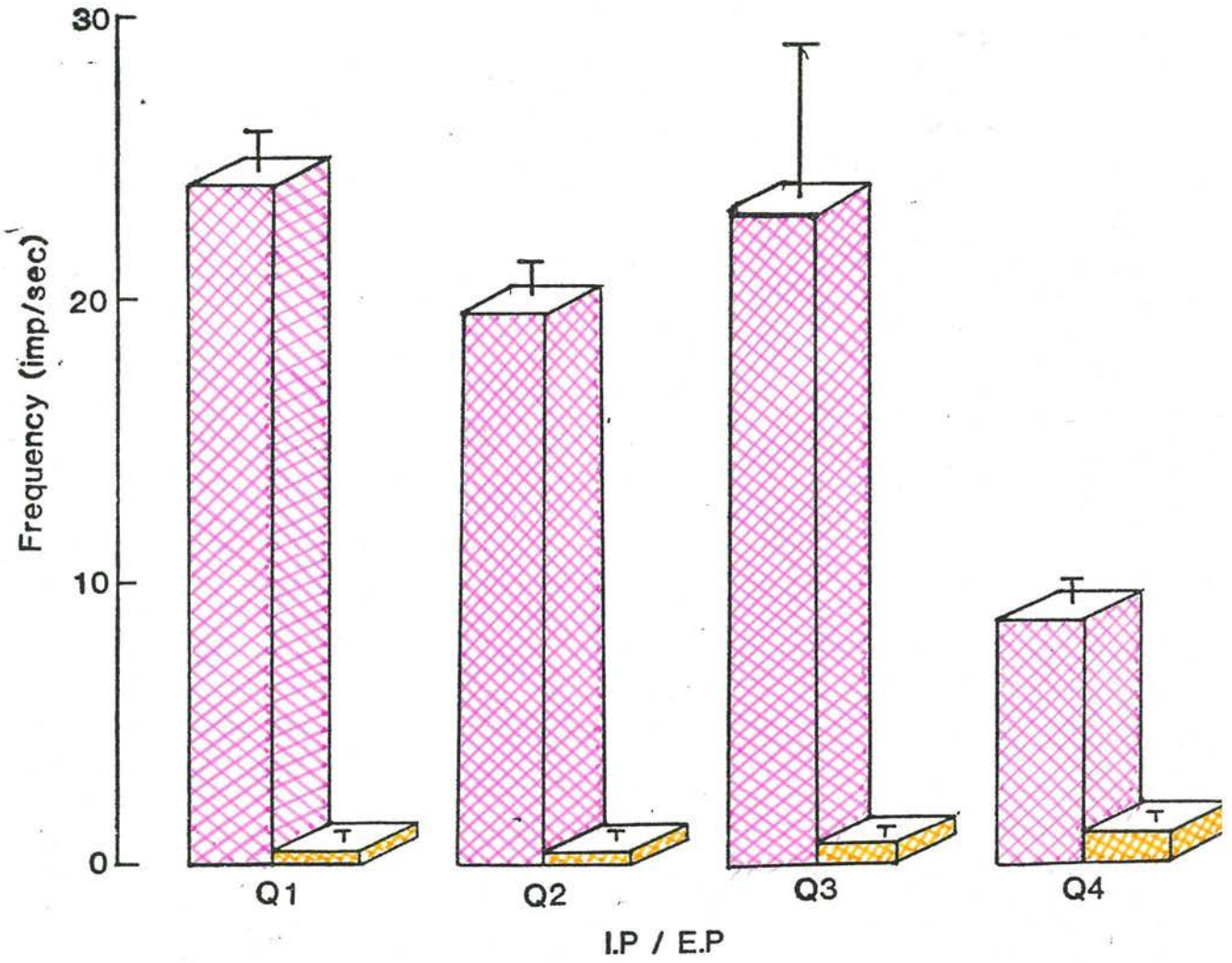

30
20
10
0
Frequency (imp/sec)
Q1
Q2
Q3
Q4
I.P / E.P

Fig. 24 Mean frequency of T-ILM discharge in different quartiles of the inspiratory (I.P) and expiratory (E.P) phases of the respiratory cycle during eupnoeic breathing (N=7 fibres).

Inspiratory discharge

Expiratory discharge

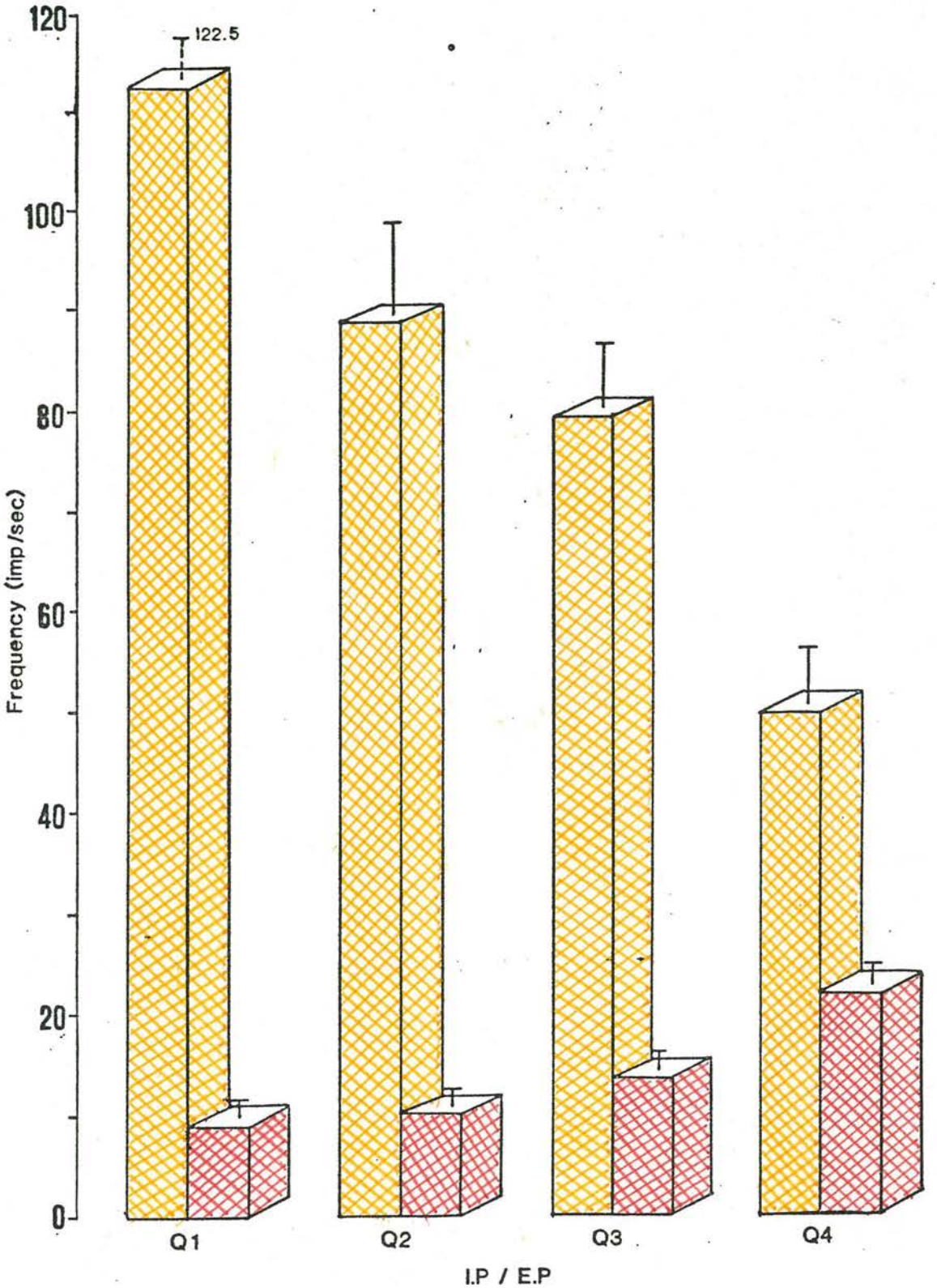

120
100
80
60
40
20
0
122.5
Frequency (imp/sec)
Q1
Q2
Q3
Q4
I.P / E.P

Fig. 25 Mean frequency of tonic-expiratory laryngeal motoneurone discharge in different quartiles of the inspiratory (I.P) and expiratory (E.P) phases of the respiratory cycle during eupnoeic breathing.

Inspiratory discharge

Expiratory discharge

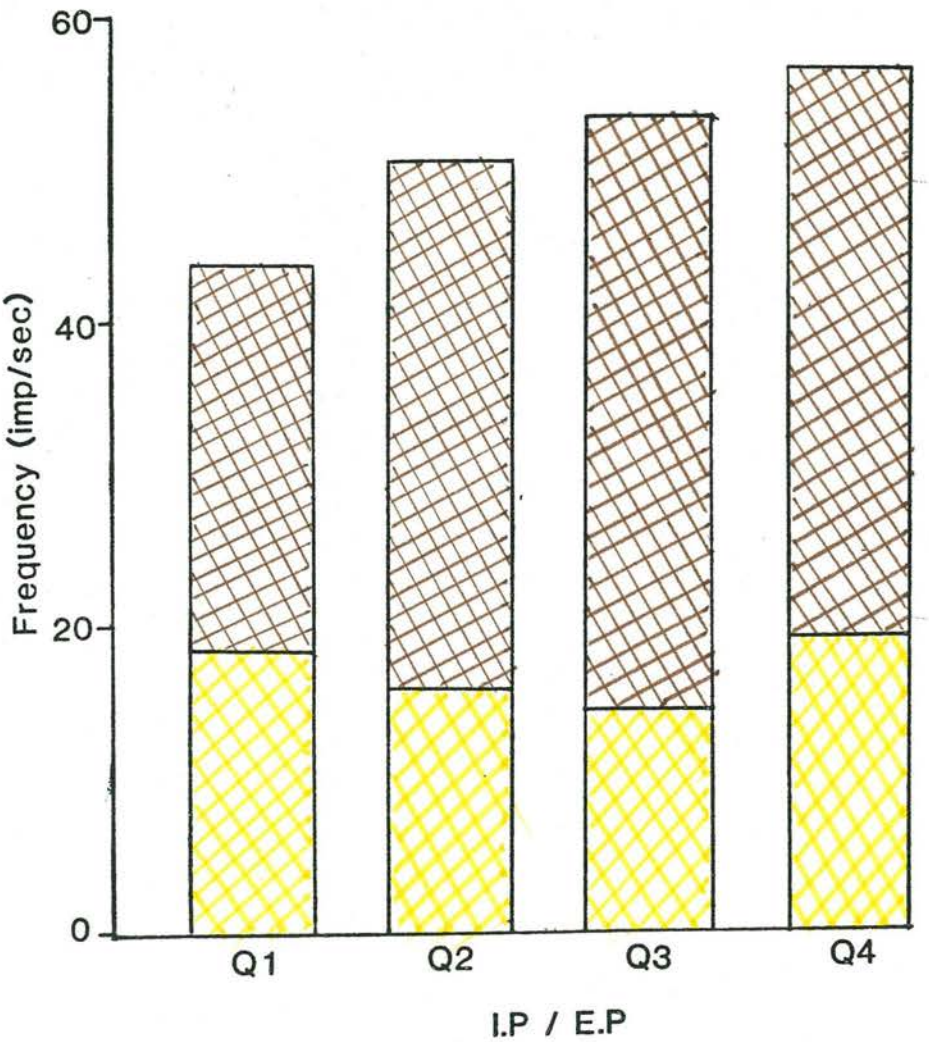
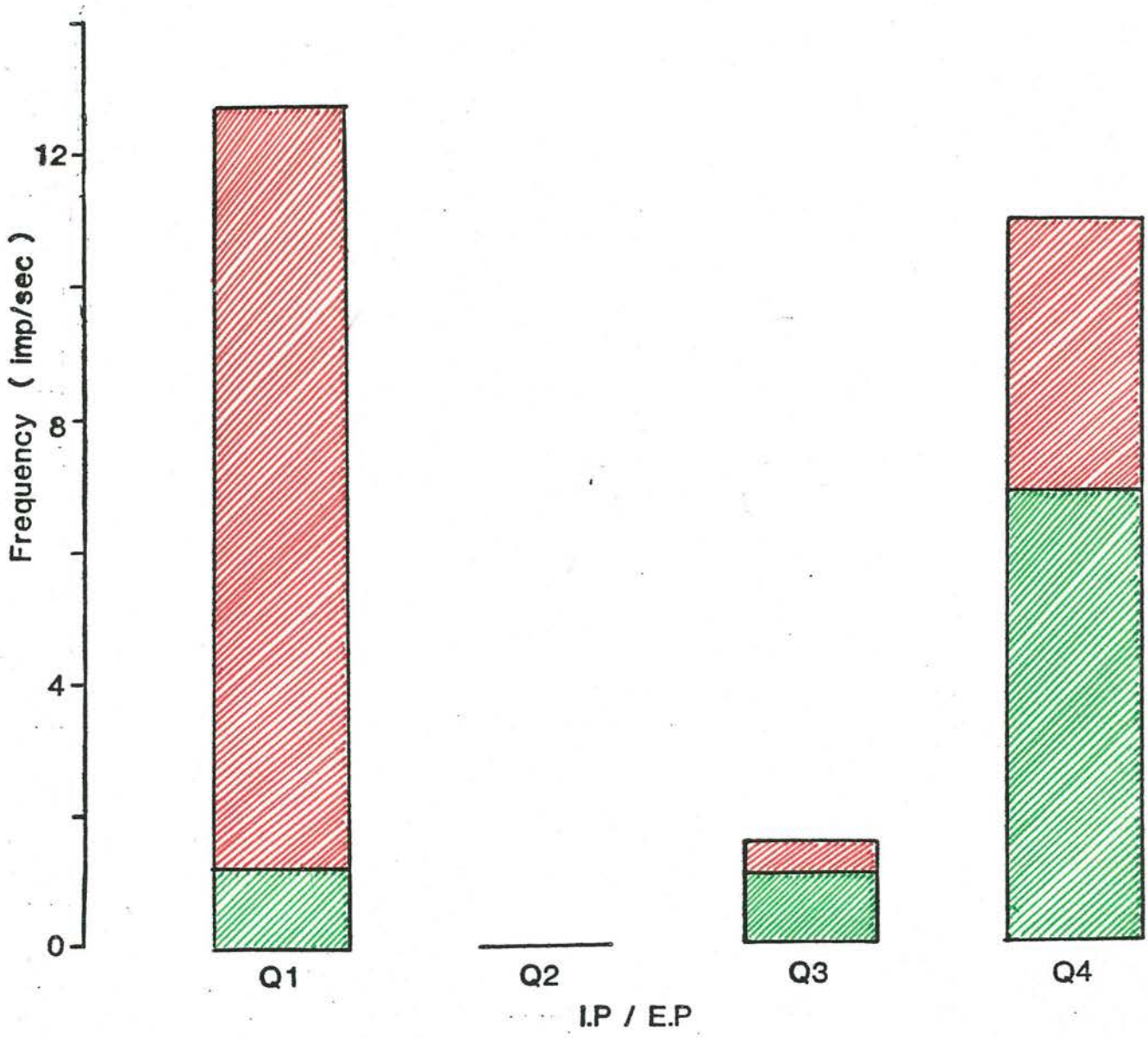

60
40
20
0
Frequency (imp/sec)
Q1
Q2
Q3
Q4
I.P / E.P

Fig. 26 Mean frequency of P-ELM discharge in different quartiles of the inspiratory (I.P) and expiratory (E.P) phases of the respiratory cycle during eupnoeic breathing.

Inspiratory discharge

Expiratory discharge


Frequency (imp/sec)
12
8
4
0
Q1
Q2
Q3
Q4
I.P / E.P

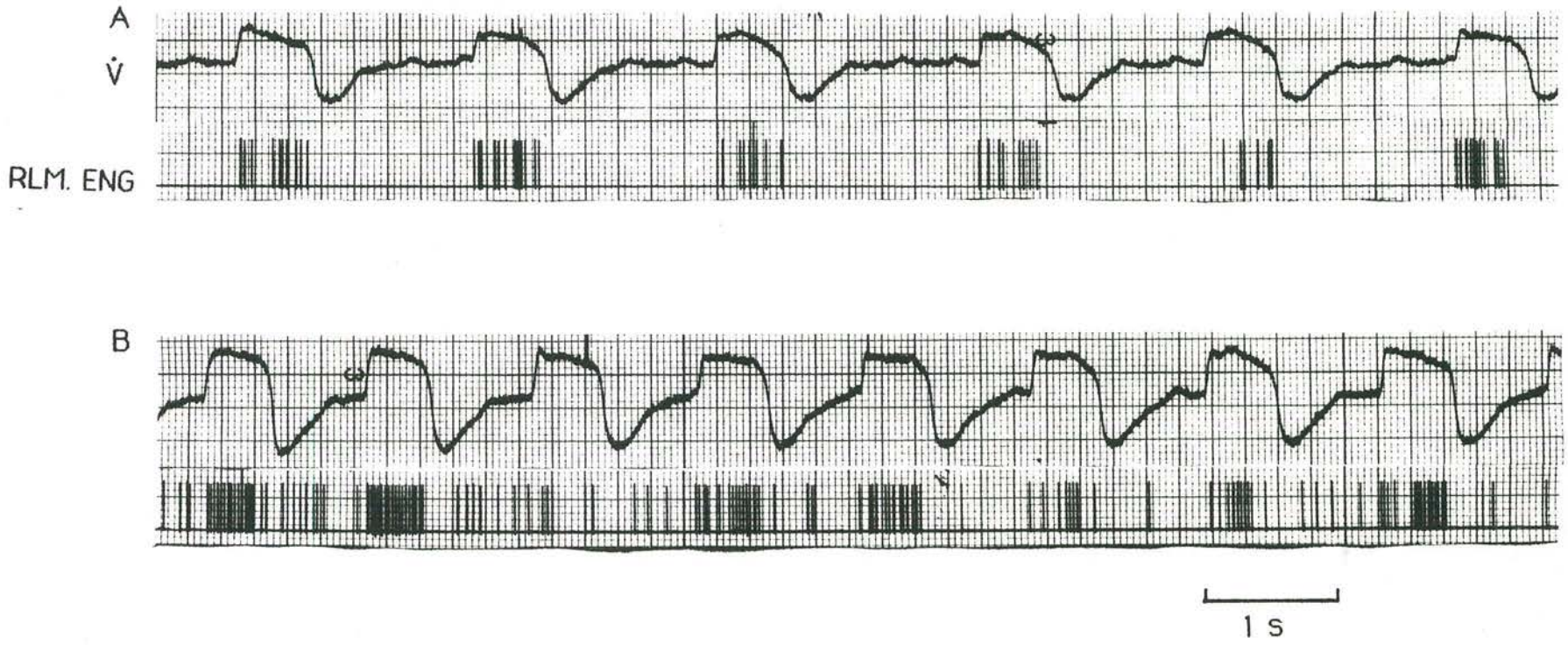
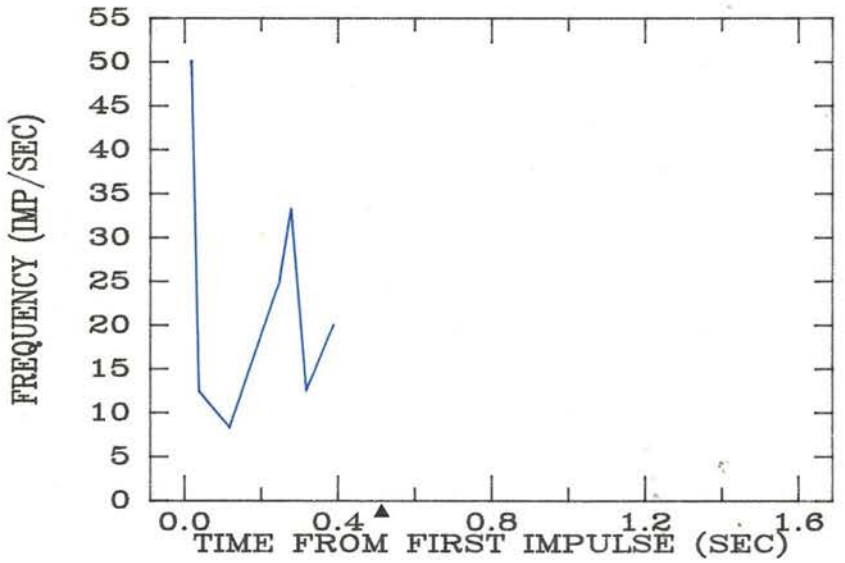


Fig. 27 Effect of breathing through an added dead space on phasic-inspiratory laryngeal motoneurone discharge. A: During control breathing, B: Breathing through an added dead space. Rabbit under pentobarbitone anaesthesia. In each record, upper trace: airflow ($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM. ENG).

Fig. 28 Instantaneous frequency plot of the phasic-inspiratory laryngeal motoneurone in Fig. 27, showing how the impulse frequency changed during hypercapnia (caused by breathing through an added dead space). Arrow indicates the end of the inspiratory phase of the respiratory cycle.

INSTANTANEOUS IMPULSE FREQUENCY IN A CONTROL BREATH



INSTANTANEOUS IMPULSE FREQUENCY DURING HYPERCAPNIA

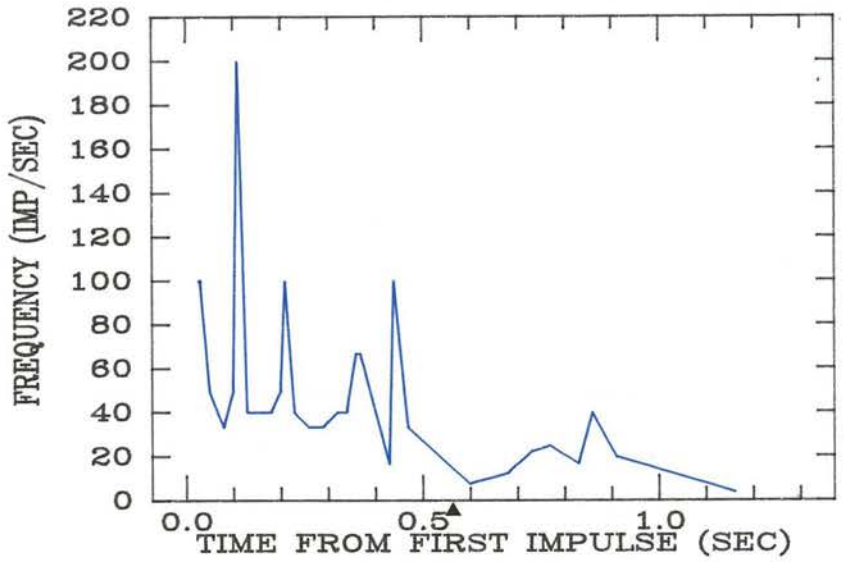
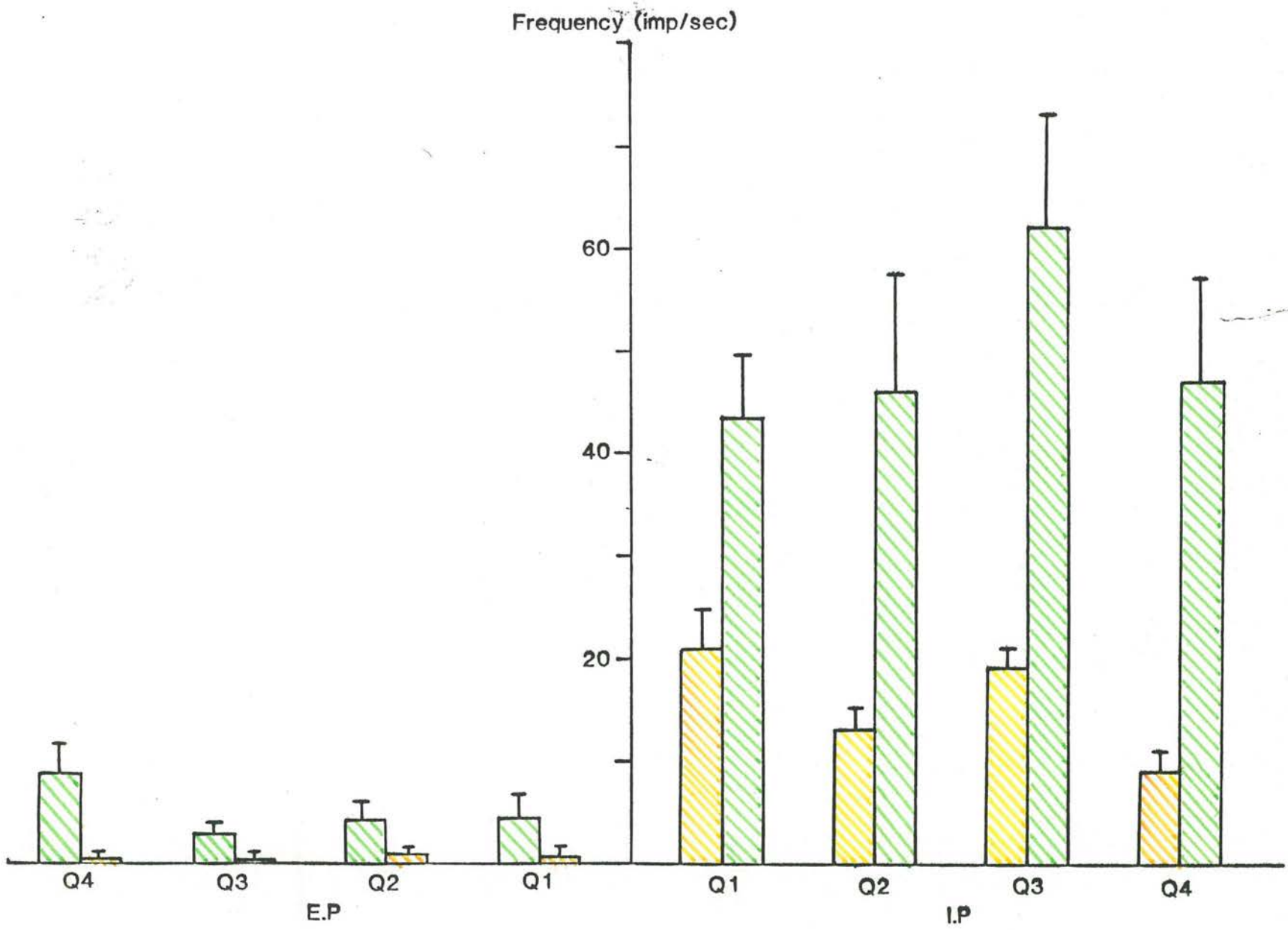


Fig. 29 Effect of breathing through an added dead space on the mean frequency of phasic-inspiratory laryngeal motoneurone discharge in different quartiles of the inspiratory (I.P) and expiratory (EP) phases of the respiratory cycle. Vertical bars represent standard errors.

During air breathing

During hypercapnia


Frequency (imp/sec)
60
40
20
Q4
Q3
Q2
Q1
E.P
Q1
Q2
Q3
Q4
I.P

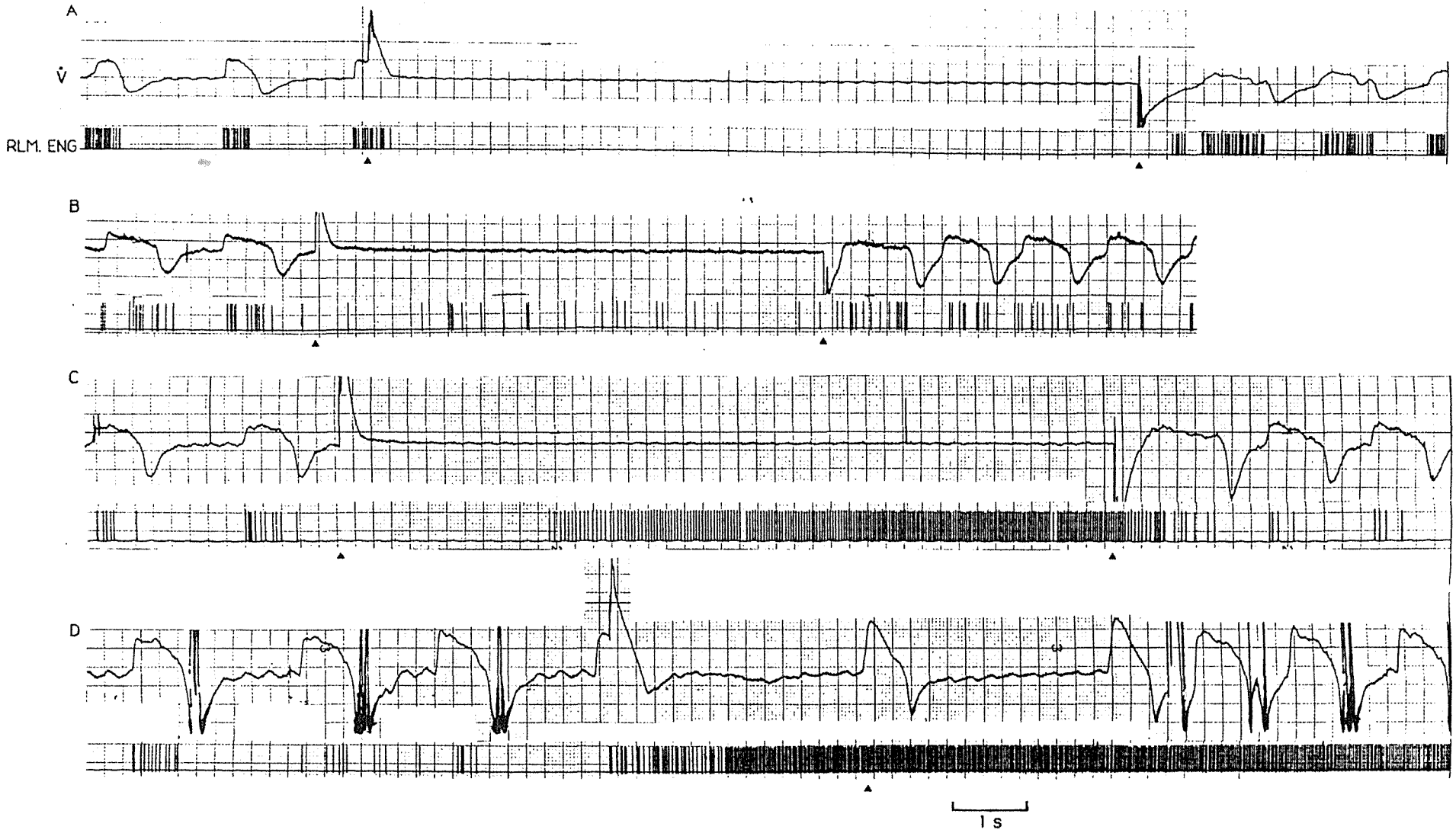


Fig. 30 Responses of phasic-inspiratory laryngeal motoneurones to lung inflation. A. Type A response, B. Type B response, C. Type C response, D. Type D response. Arrows indicate the onset and the end of lung inflation. Rabbits under pentobarbitone anaesthesia. The artifact produced by the opening and closing of the magnetic valves is clearly seen on the airflow record. In each record, upper trace: airflow ($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM. ENG).

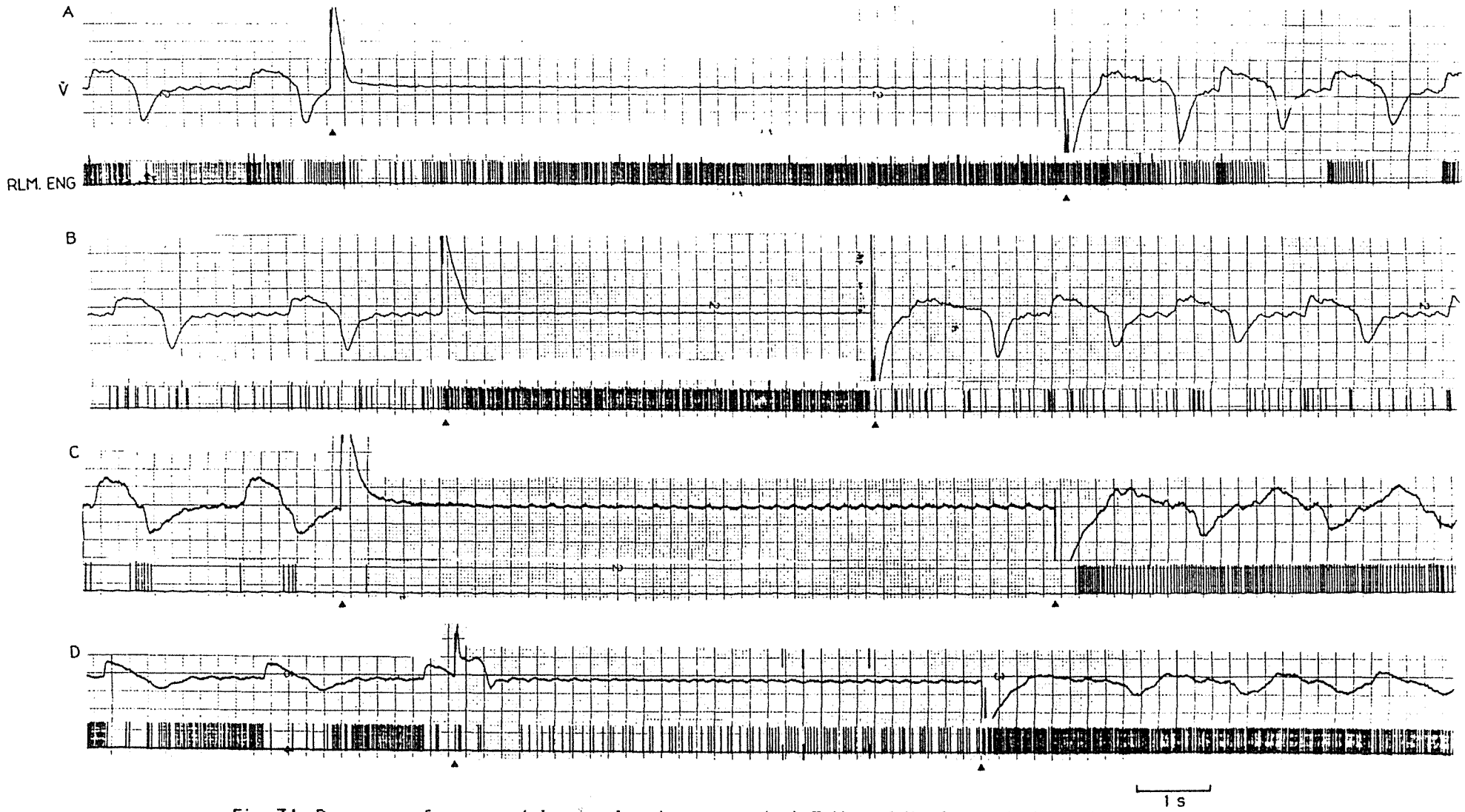


Fig. 31 Responses of recurrent laryngeal motoneurones to inflation of the lungs. A: A type B response of a tonic-inspiratory laryngeal motoneurone, B: A type D response of a tonic-inspiratory laryngeal motoneurone, C: A type A response of a phasic-expiratory laryngeal motoneurone, D: A type B response of a tonic-expiratory laryngeal motoneurone. The artifact produced by the opening and closing of the magnetic valves is clearly seen on the airflow record. Rabbits under pentobarbitone anaesthesia. In each record, upper trace: airflow($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM.ENG).

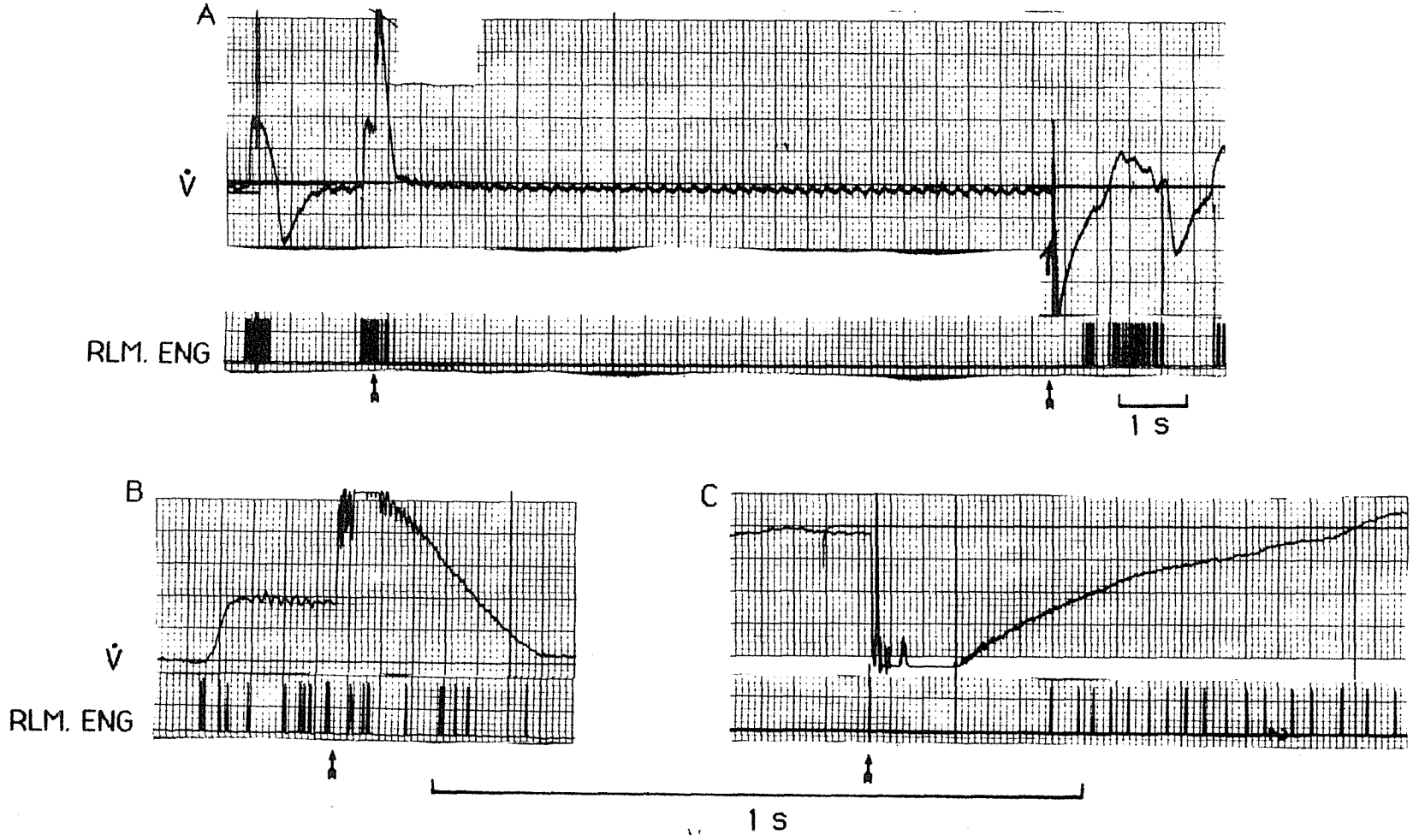


Fig. 32 Typical transient response of a phasic-inspiratory laryngeal motoneurone to lung inflation (between the arrows). Records B and C from the same motoneurone in record A, show transient response immediately following lung inflation and after the release of lung inflation. Rabbit under pentobarbitone anaesthesia. In each record, upper trace: airflow ($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM.ENG).

Fig. 33 Changes in frequency of inspiratory laryngeal motoneurone discharge relative to control inspiratory discharge (Ftr/Fc) elicited by lung inflation and deflation at different positions of the inspiratory phase (I.P) of the respiratory cycle.

□ During lung inflation (PSR intact)

■ During lung inflation (PSR blocked)

○ During lung deflation (PSR intact)

● During lung deflation (PSR blocked)

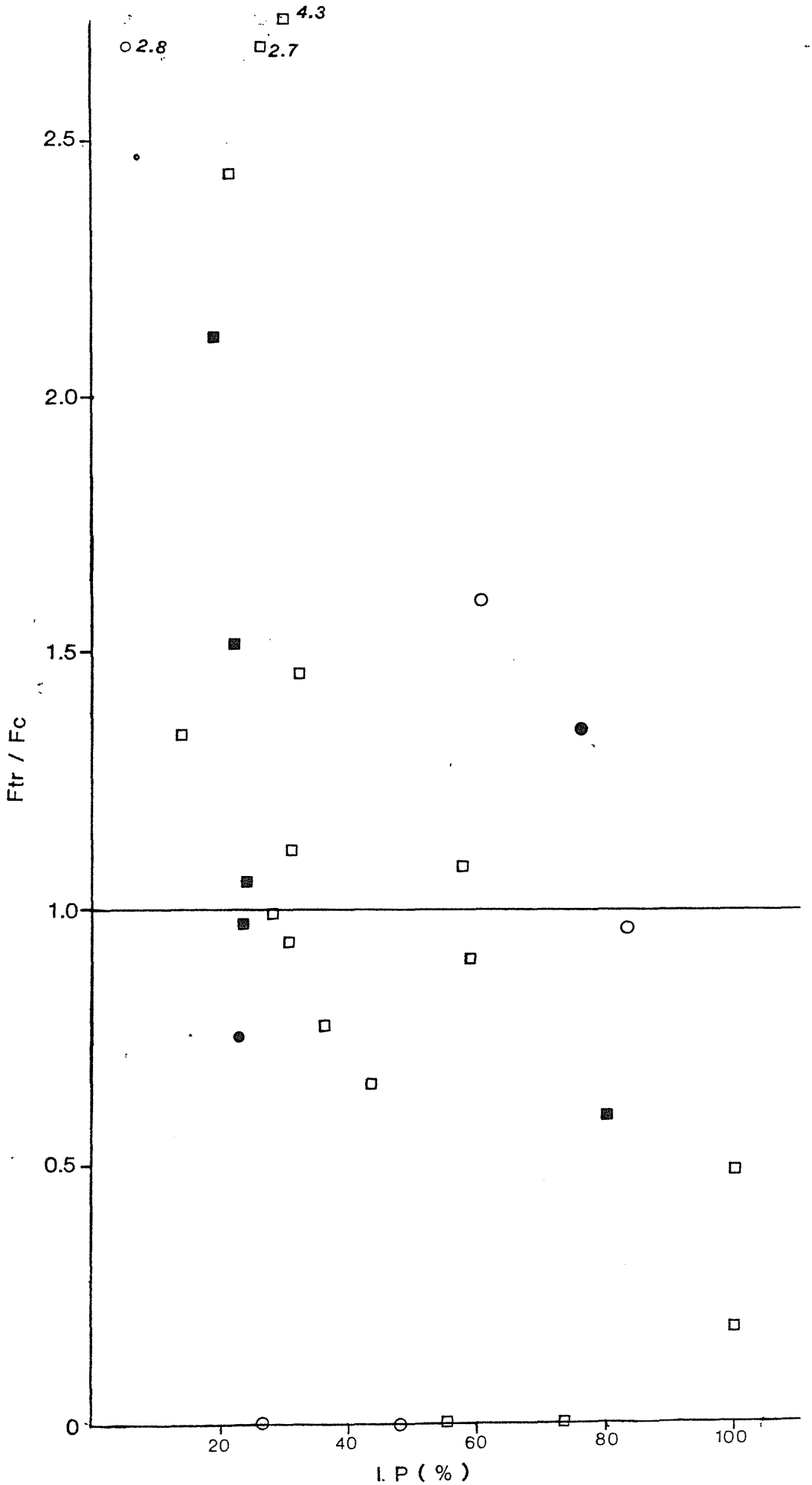

4.3
2.8
2.7
2.5
2.0
1.5
1.0
0.5
0
Ftr / Fc
20
40
60
80
100
I. P (%)

Fig. 34 Changes in frequency of inspiratory laryngeal motoneurone discharge relative to control inspiratory discharge (Ftr/Fc) elicited by lung inflation and deflation at different positions of the expiratory phase (E.P) of the respiratory cycle.

□ During lung inflation (PSR intact)

■ During lung inflation (PSR blocked)

○ During lung deflation (PSR intact)

● During lung deflation (PSR blocked)

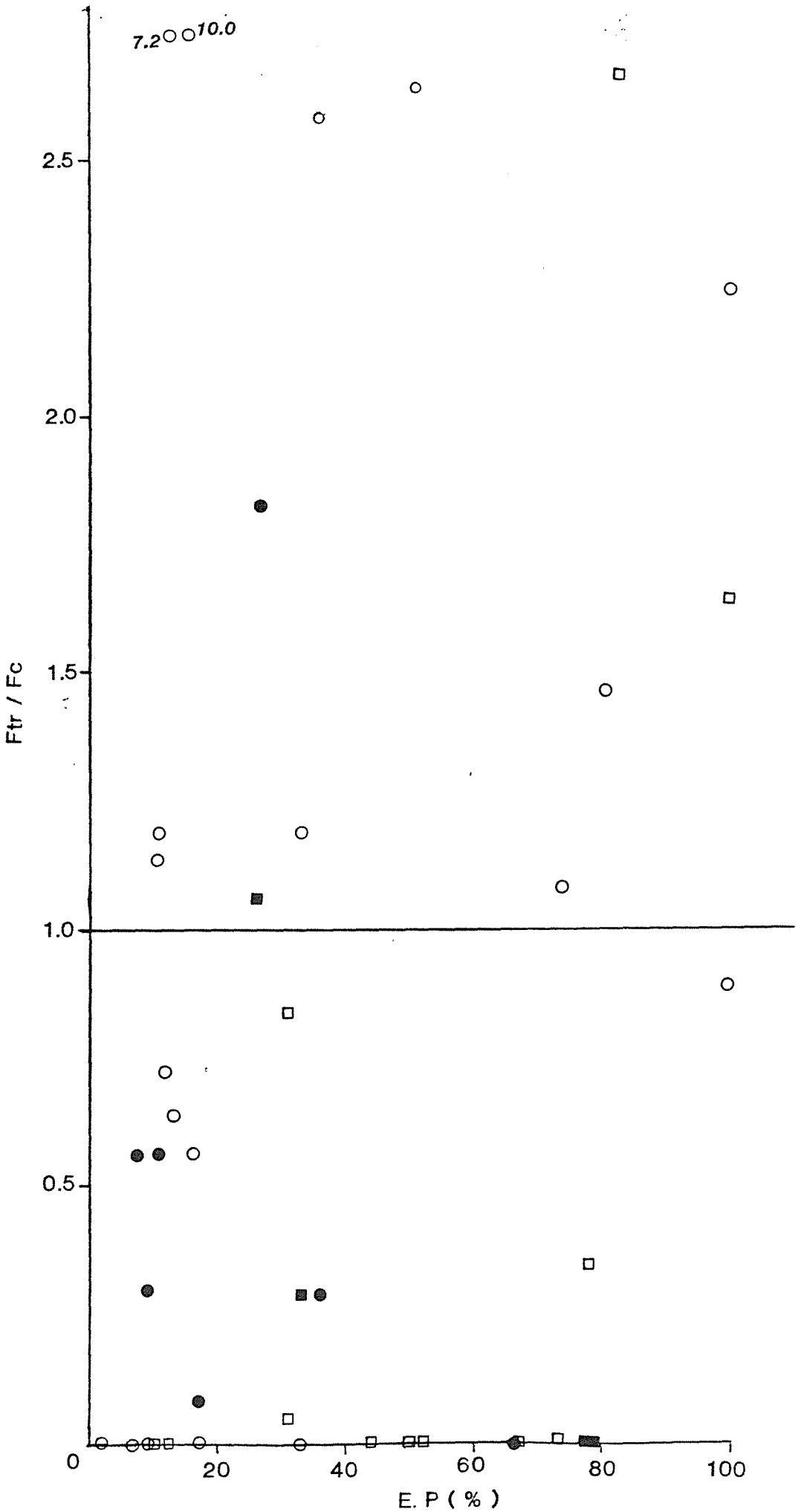

7.2
10.0
2.5
2.0
1.5
1.0
0.5
0
Ftr / Fc
20
40
60
80
100
E. P (%)

Fig. 35 Changes in the frequency of inspiratory laryngeal motoneurone discharge relative to control inspiratory discharge (Ftr / Fc) following the release of lung inflation or deflation.

- ○ Lung deflation when PSR were intact (-ve intact)
- □ Lung inflation when PSR were intact (+ve intact)
- ● Lung deflation when PSR were blocked (-ve block)
- ■ Lung inflation when PSR were blocked (+ve block)

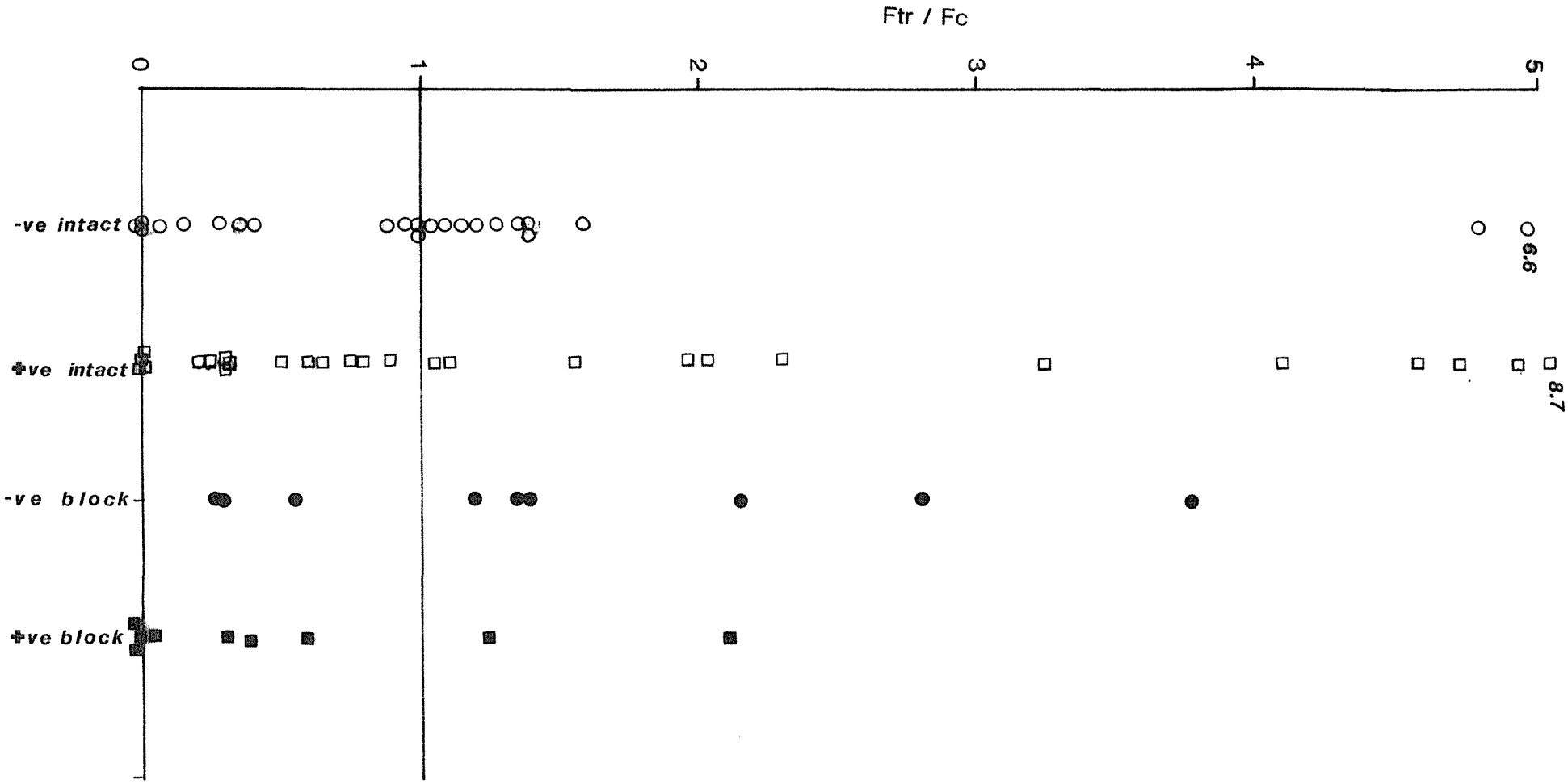

Ftr / Fc
0
1
2
3
4
5
-ve intact
+ve intact
-ve block
+ve block
6.6
8.7

Fig. 36 Changes in frequency of expiratory laryngeal motoneurone discharge relative to control expiratory discharge (Ftr/Fc) elicited by lung inflation and deflation at different positions of the inspiratory phase (I.P) of the respiratory cycle.

□ During lung inflation (PSR intact)

■ During lung inflation (PSR blocked)

○ During lung deflation (PSR intact)

● During lung deflation (PSR blocked)

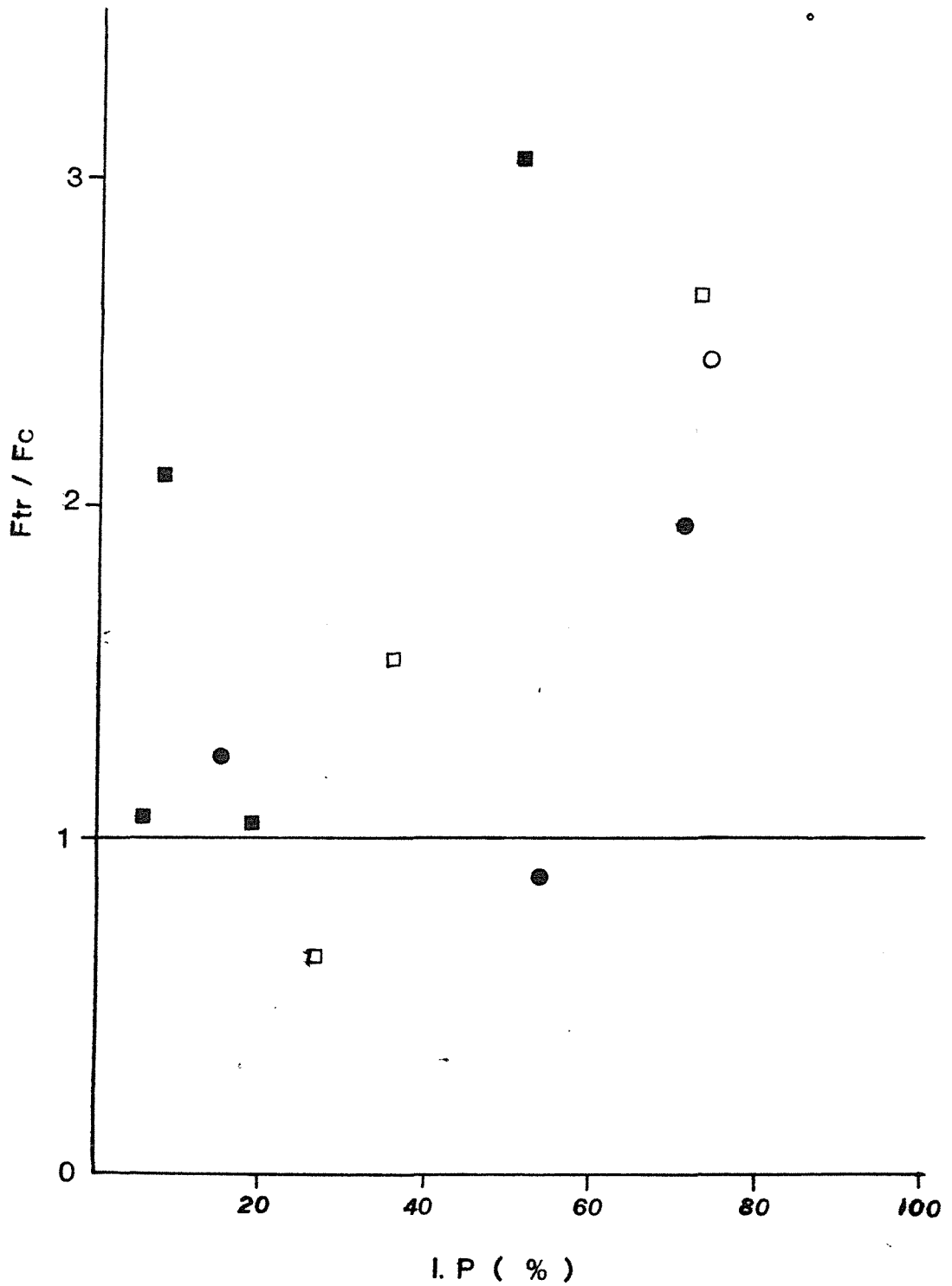

Ftr / Fc
3
2
1
0
20
40
60
80
100
I. P (%)

Fig. 37 Changes in frequency of expiratory laryngeal motoneurone discharge relative to control expiratory discharge (Ftr/Fc) elicited by lung inflation and deflation at different positions of the expiratory phase (E.P) of the respiratory cycle.

□ During lung inflation (PSR intact)

■ During lung inflation (PSR blocked)

○ During lung deflation (PSR intact)

● During lung deflation (PSR blocked)

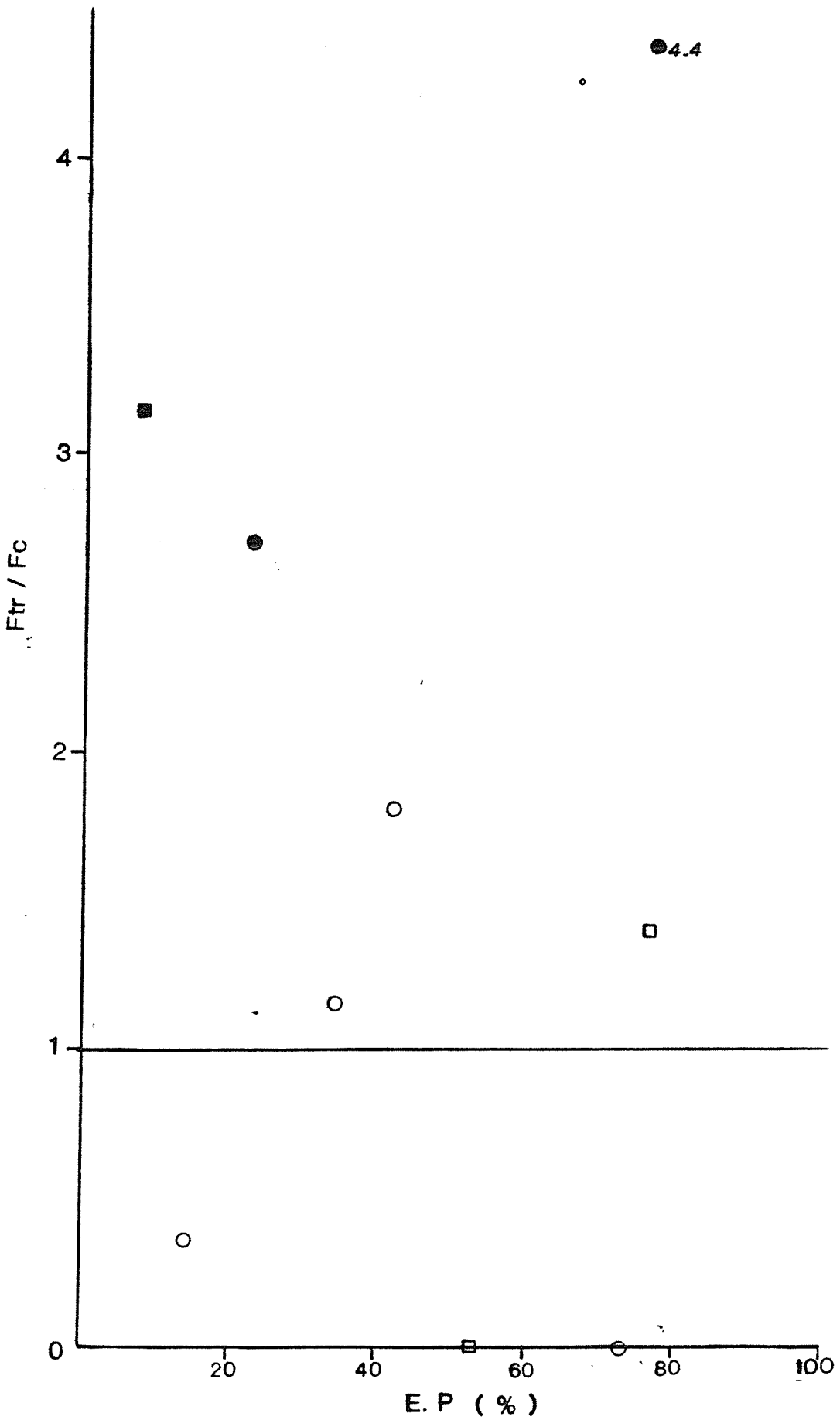

4.4
4
3
2
1
0
Ftr / Fc
20
40
60
80
100
E. P (%)

Fig. 38 Changes in the frequency of expiratory laryngeal motoneurone discharge relative to control expiratory discharge (Ftr / Fc) following the release of lung inflation or deflation.

○ Lung deflation when PSR were intact (−ve intact)

□ Lung inflation when PSR were intact (+ve intact)

● Lung deflation when PSR were blocked (−ve block)

■ Lung inflation when PSR were blocked (+ve block)

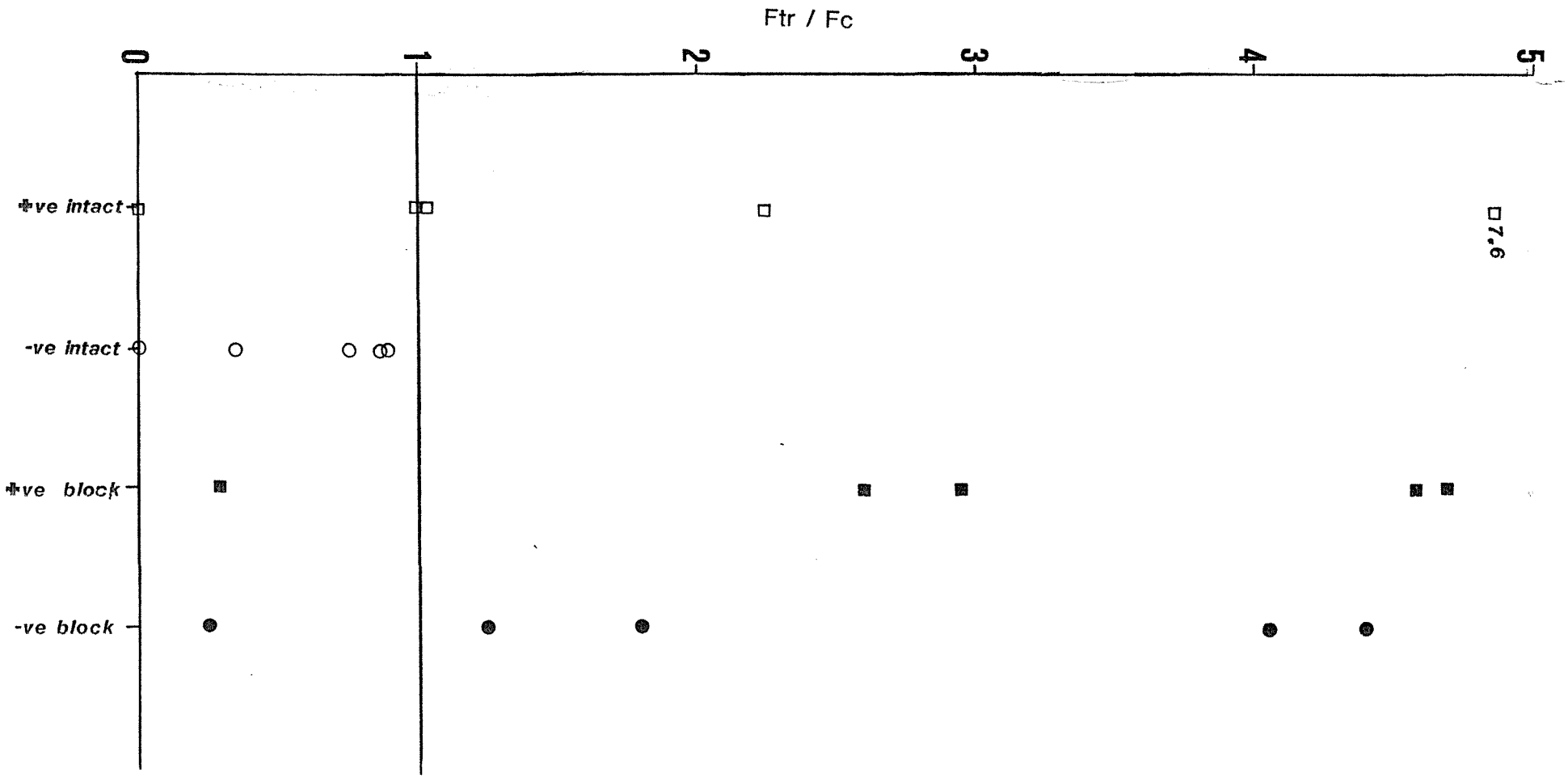

Ftr / Fc
0
1
2
3
4
5
+ve intact
-ve intact
+ve block
-ve block
7.6

FIG. 39 TYPES OF RESPONSE OF RECURRENT LARYNGEAL MOTONEURONES TO LUNG INFLATION

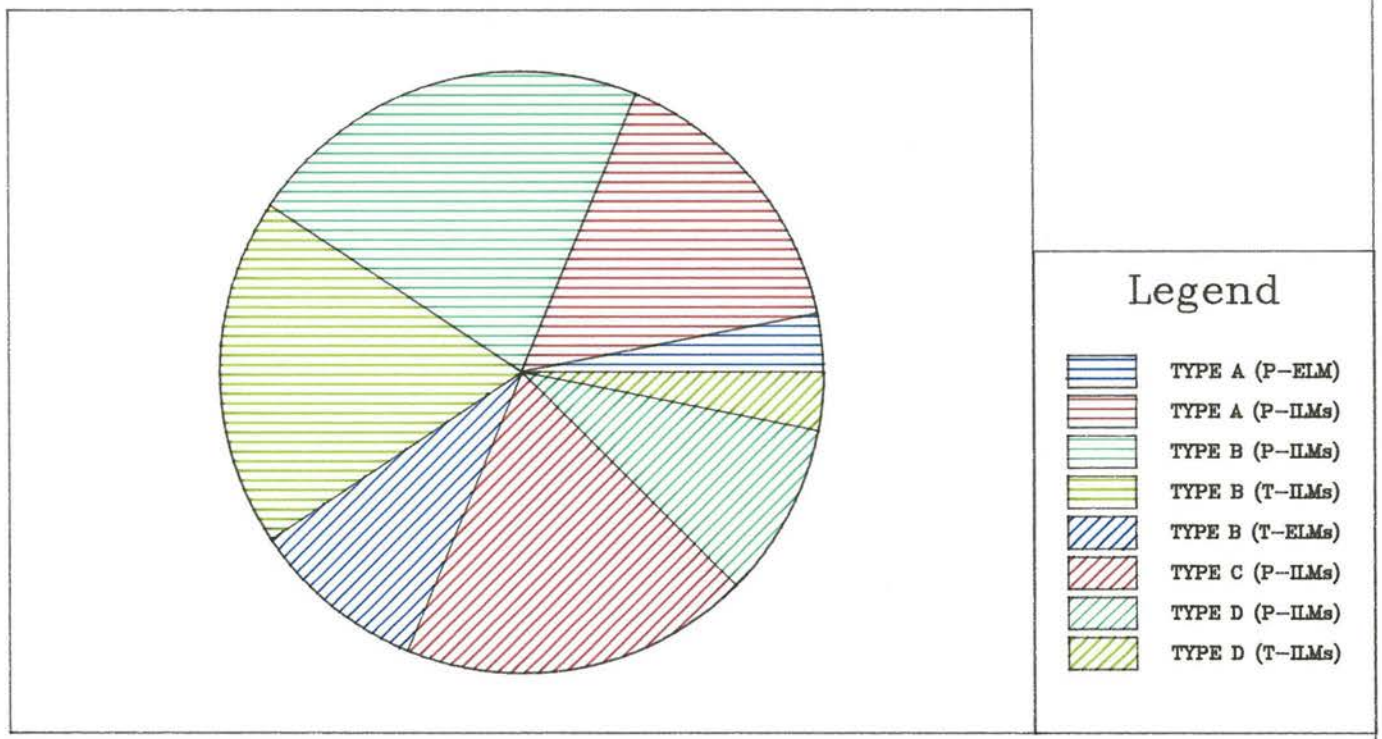


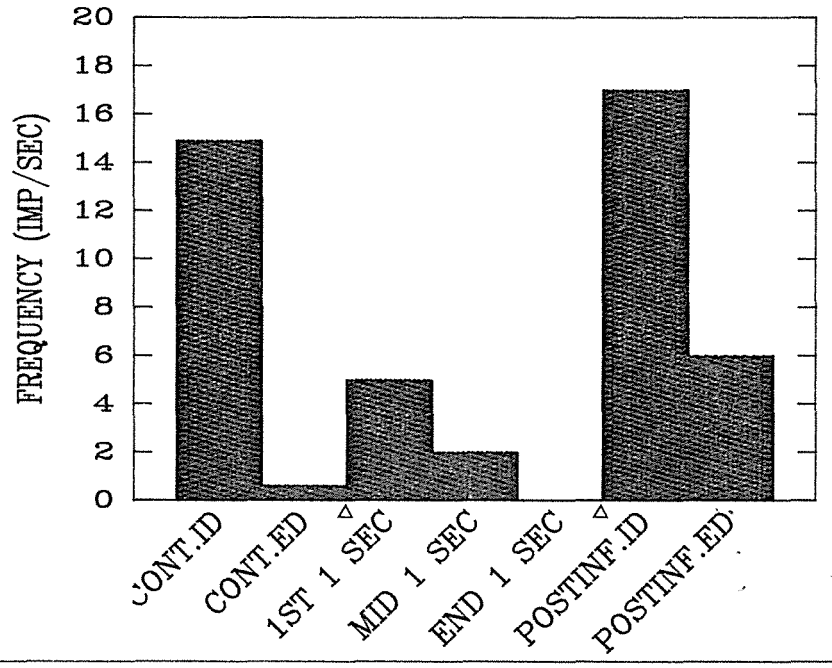
Fig. 40a: Histogram showing changes in discharge frequency during various responses to lung inflation (between the arrows):

i. a type B response of a phasic-inspiratory laryngeal motoneurone

ii. a type B response of a tonic-inspiratory laryngeal motoneurone

Abbreviations: cont. ID and cont. ED: control inspiratory discharge and control expiratory discharge respectively; postinf. ID and postinf. ED: postinflation inspiratory and expiratory discharges respectively.

A "TYPE—B" RESPONSE OF P—ILM TO LUNG INFLATION



A "TYPE—B" RESPONSE OF T—ILM TO LUNG INFLATION

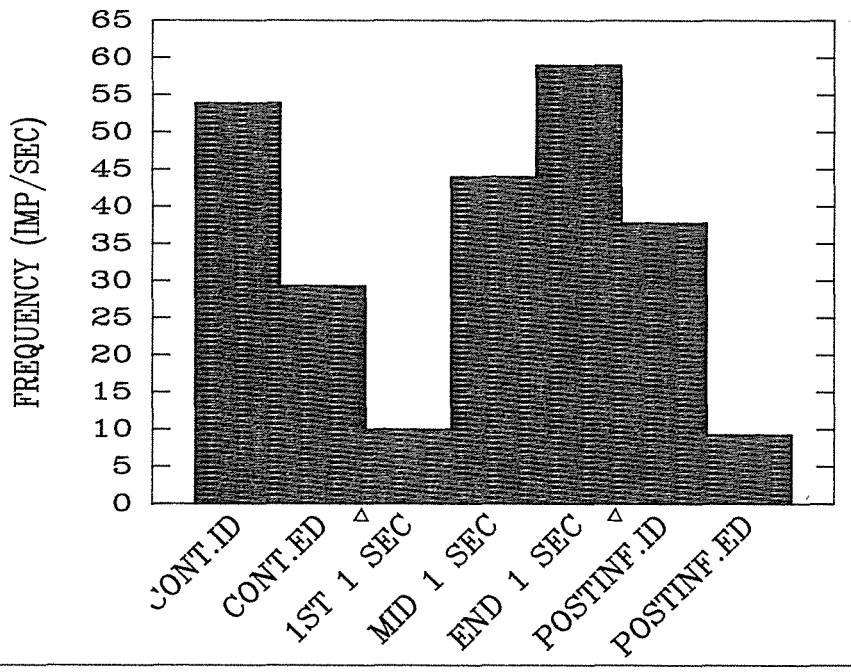


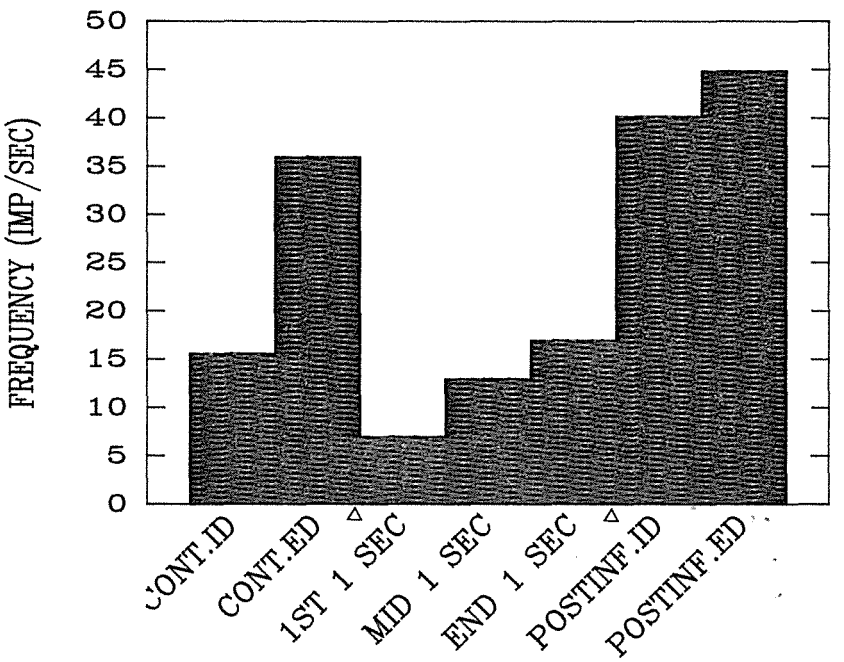
Fig. 40b: Histogram showing changes in discharge frequency during various responses to lung inflation (between the arrows):

i. a type B response of a tonic-expiratory laryngeal motoneurone

ii. a type C response of a phasic-inspiratory laryngeal motoneurone

Abbreviations: cont. ID and cont. ED: control inspiratory discharge and control expiratory discharge respectively; postinf. ID and postinf. ED: postinflation inspiratory and expiratory discharges respectively.

A "TYPE–B" RESPONSE OF T–ELM TO LUNG INFLATION



A "TYPE–C" RESPONSE OF P–ILM TO LUNG INFLATION

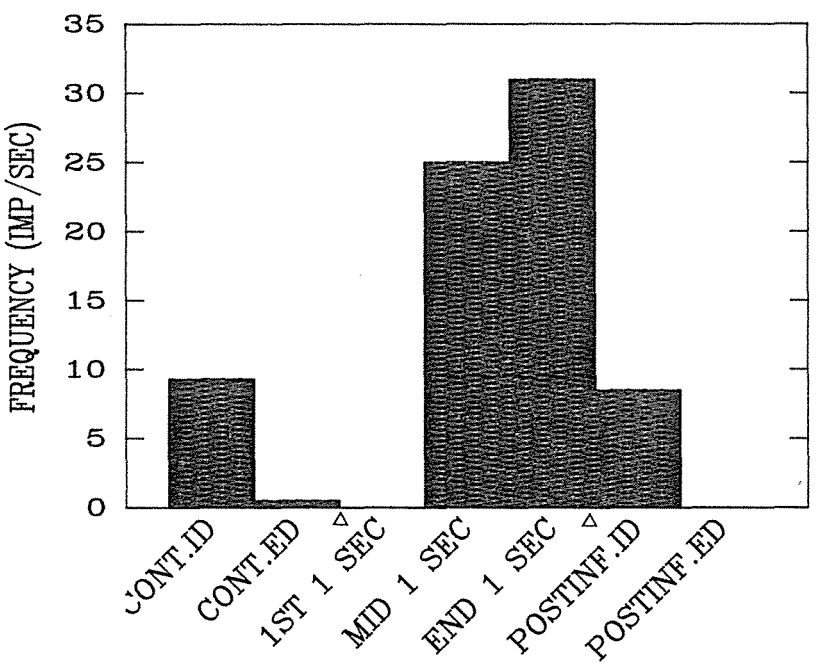


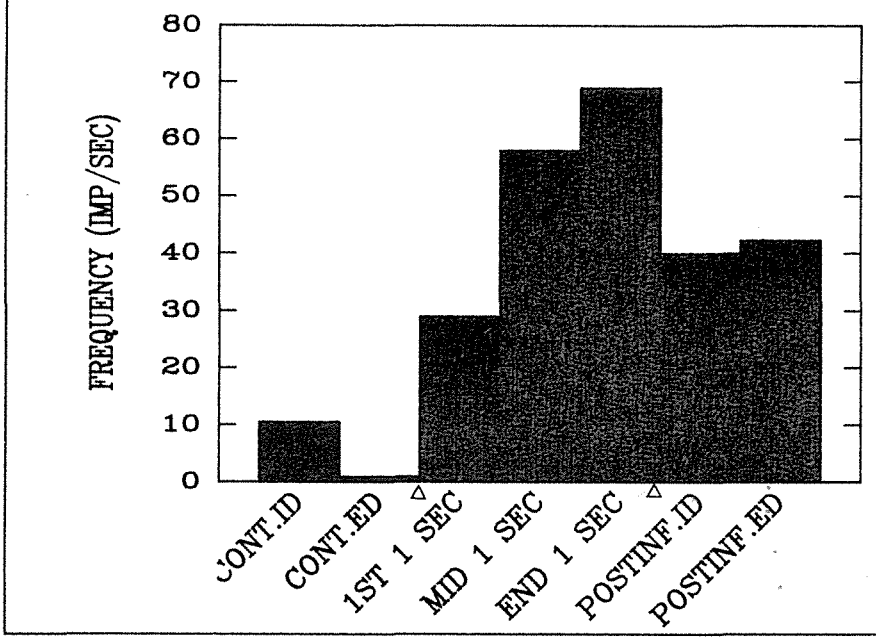
Fig. 40c: Histogram showing changes in discharge frequency during various responses to lung inflation (between the arrows):

i. a type D response of a phasic-inspiratory laryngeal motoneurone

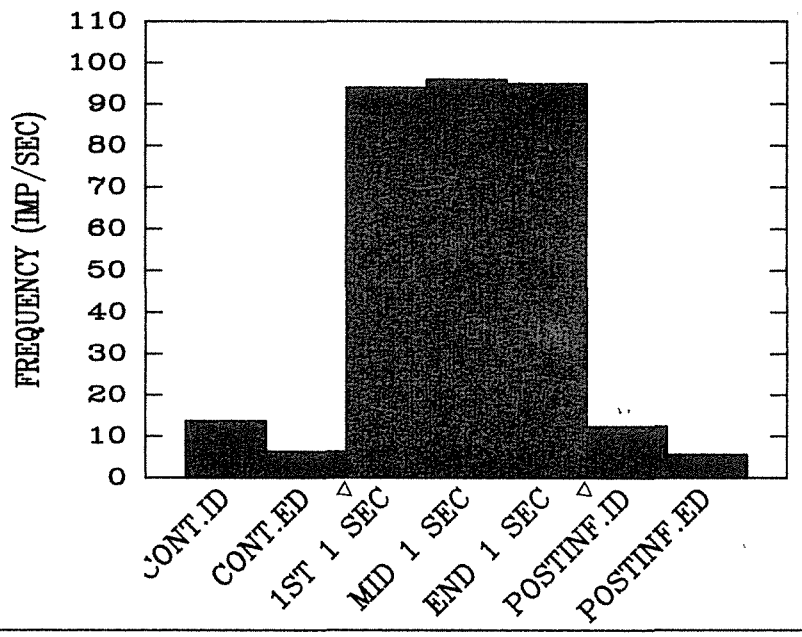
ii. a type D response of a tonic-inspiratory laryngeal motoneurone

Abbreviations: cont. ID and cont. ED: control inspiratory discharge and control expiratory discharge respectively; postinf. ID and postinf. ED: postinflation inspiratory and expiratory discharges respectively.

A "TYPE—D" RESPONSE OF P—ILM TO LUNG INFLATION



A "TYPE—D" RESPONSE OF T—ILM TO LUNG INFLATION



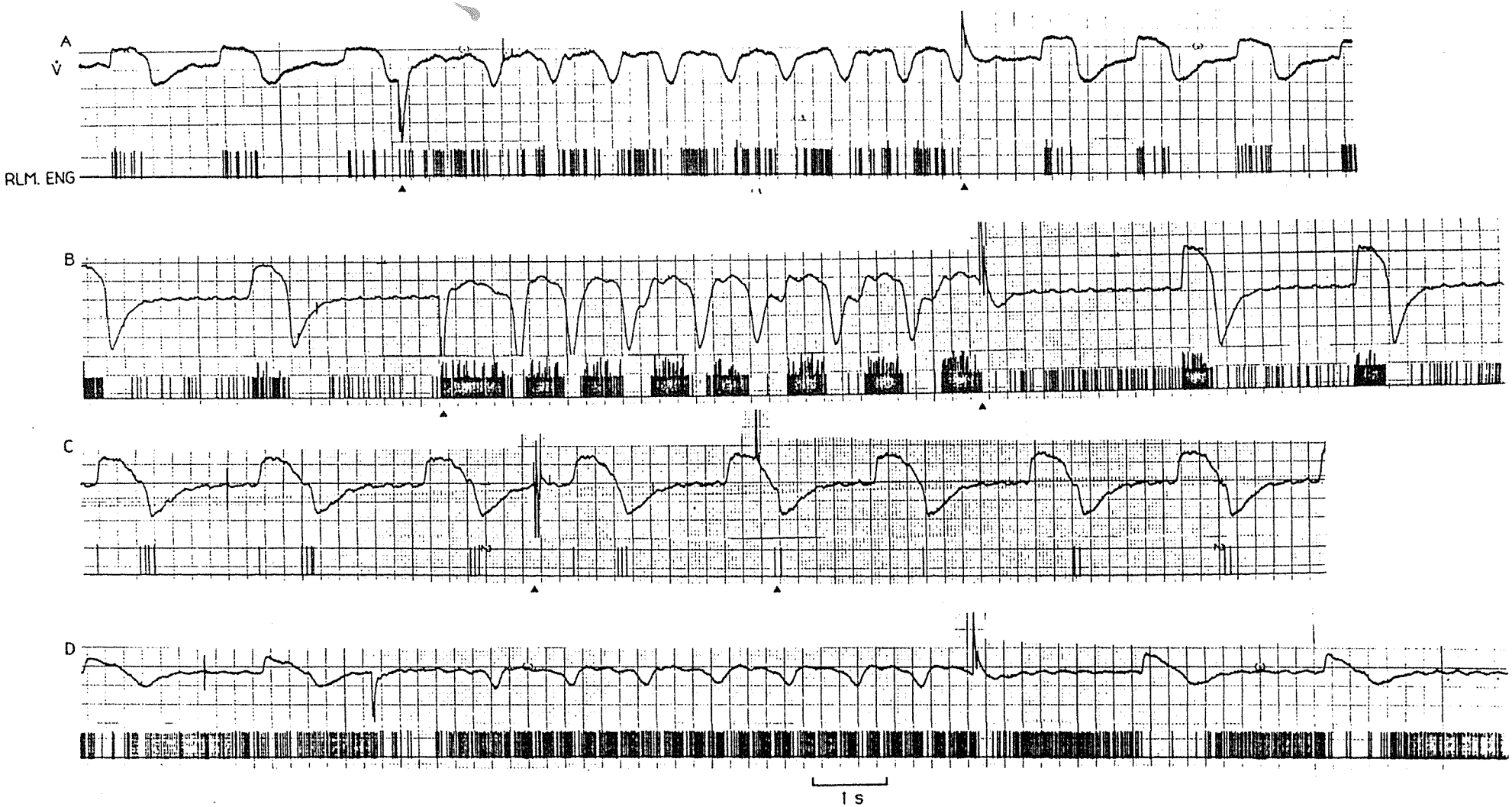


Fig. 41 Response to lung deflation (between the arrows). A: phasic-inspiratory laryngeal motoneurone, B: tonic-inspiratory laryngeal motoneurone, C: phasic-expiratory laryngeal motoneurone, D: tonic-expiratory laryngeal motoneurone. In each record, upper trace: airflow ($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM.ENG). The artifact produced by the opening and closing of the magnetic valves is seen on the airflow trace. Rabbits under pentobarbitone anaesthesia.

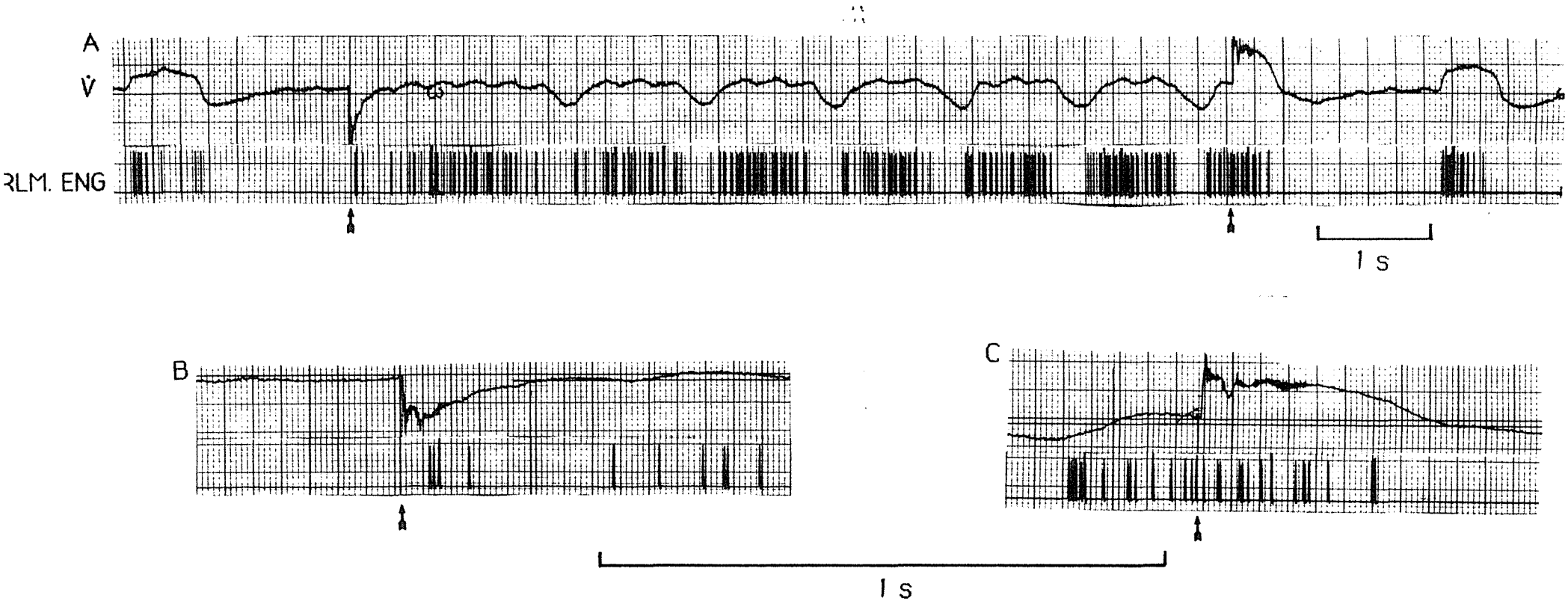
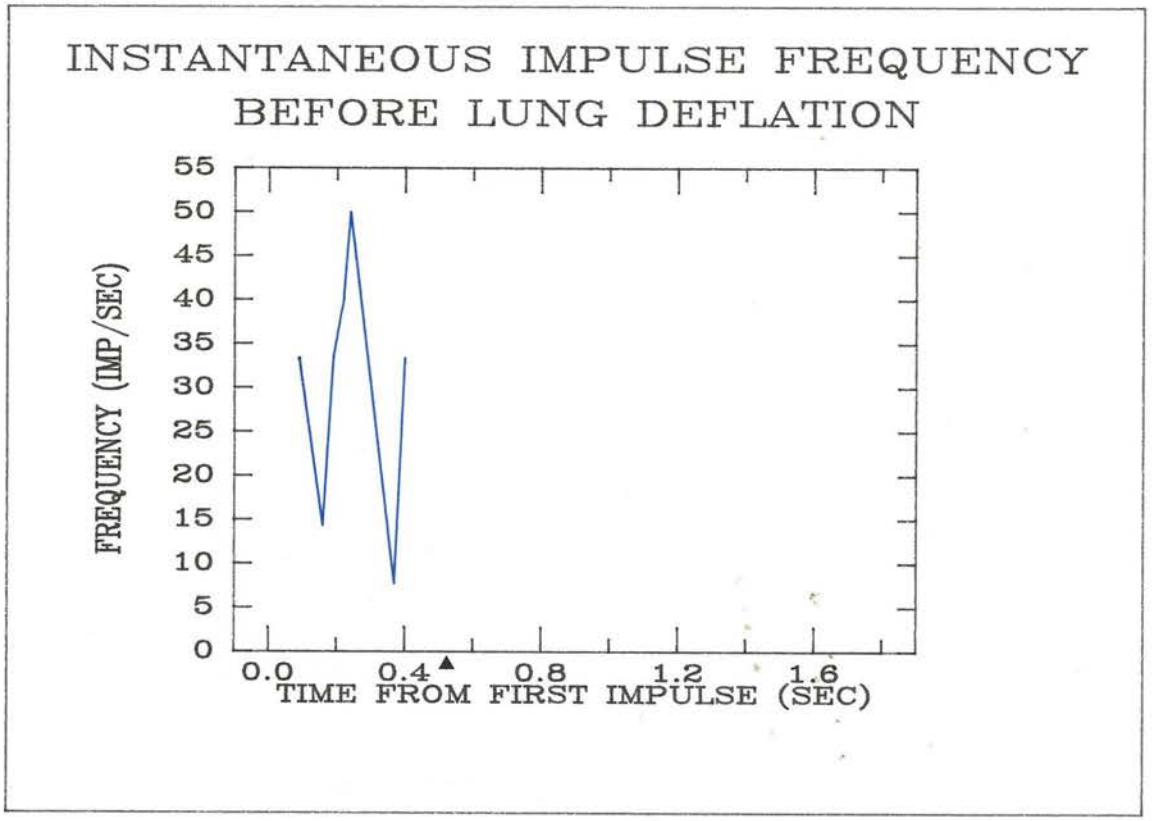


Fig. 42 Typical transient response of a phasic-inspiratory laryngeal motoneurone to lung deflation (between the arrows). Records B and C from the same motoneurone in record A, show transient response immediately following lung deflation and after the release of lung deflation respectively. Rabbit under pentobarbitone anaesthesia. In each record, upper trace: airflow ($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM. ENG).

Fig. 43 Instantaneous frequency plot of the phasic-inspiratory laryngeal motoneurone in Fig. 41 (record A), showing how the impulse frequency changed during lung deflation. Arrow indicates the end of the inspiratory phase of the respiratory cycle.


INSTANTANEOUS IMPULSE FREQUENCY
BEFORE LUNG DEFLATION
FREQUENCY (IMP/SEC)
55
50
45
40
35
30
25
20
15
10
5
0
0.0
0.4
0.8
1.2
1.6
TIME FROM FIRST IMPULSE (SEC)

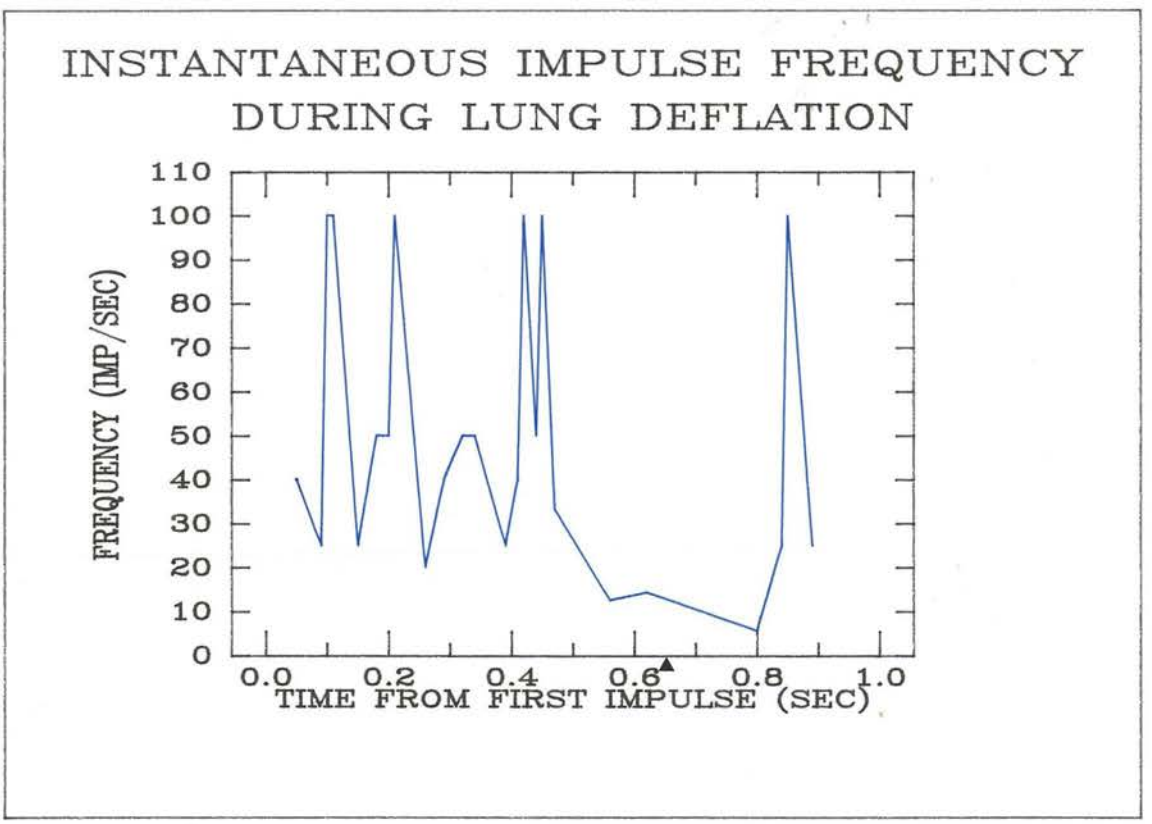
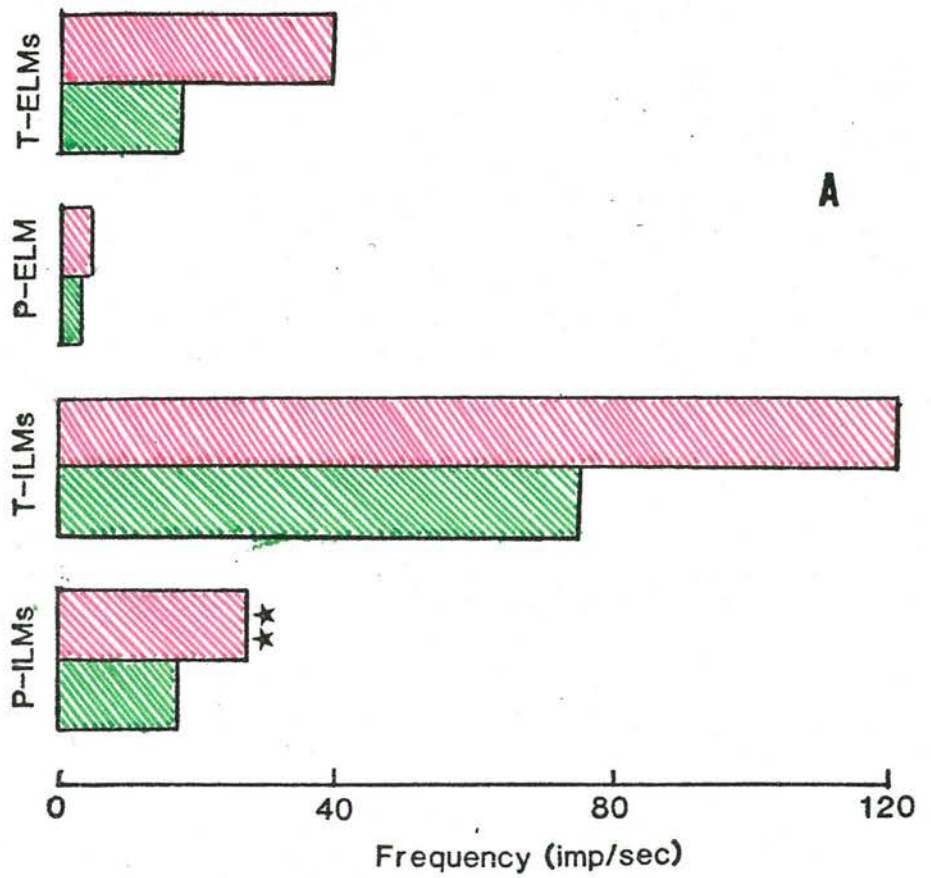

INSTANTANEOUS IMPULSE FREQUENCY
DURING LUNG DEFLATION
FREQUENCY (IMP/SEC)
110
100
90
80
70
60
50
40
30
20
10
0
0.0
0.2
0.4
0.6
0.8
1.0
TIME FROM FIRST IMPULSE (SEC)

Fig. 44 Effect of lung deflation on the mean frequency of recurrent laryngeal motoneurone discharge during the inspiratory (A) and expiratory (B) phases of the respiratory cycle. Significance of difference of response to lung deflation compared with control (before deflation): * $P < 0.05$, ** $P < 0.01$.

Before lung deflation

During lung deflation


T-ELMs
P-ELM
T-ILMs
P-ILMs
A
0
40
80
120
Frequency (imp/sec)

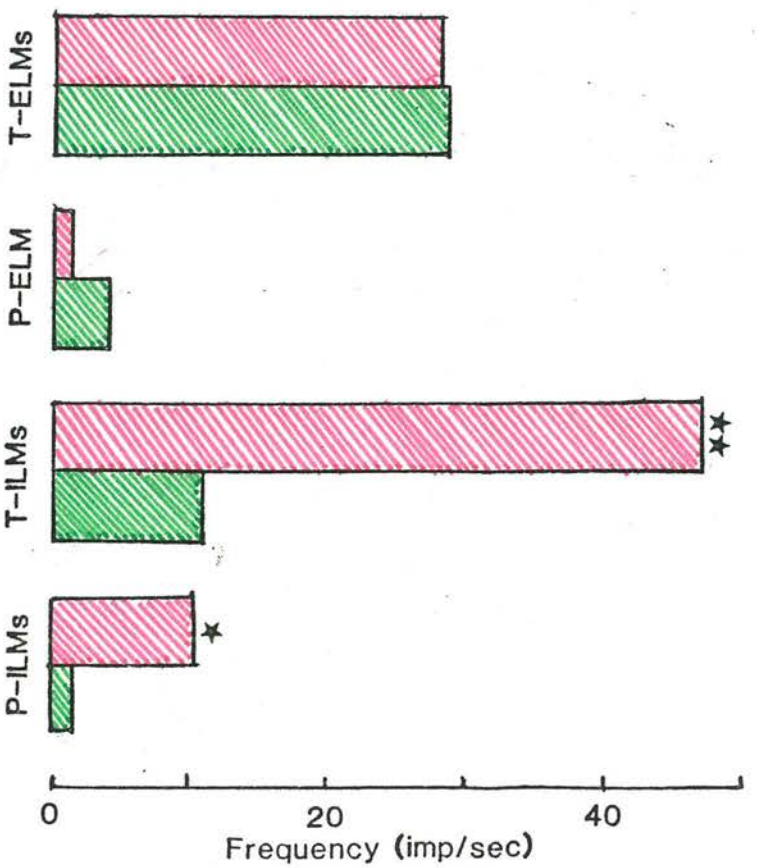
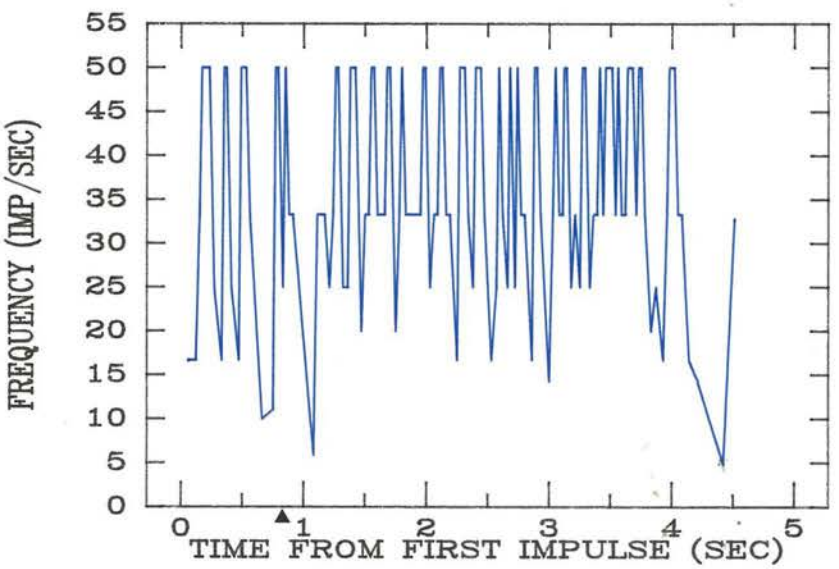
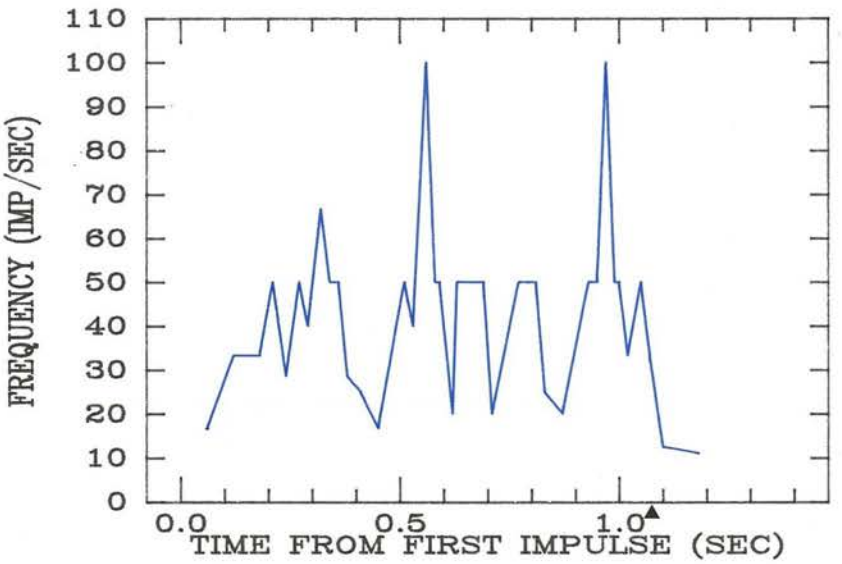

T-ELMs
P-ELM
T-ILMs
P-ILMs
B
0
20
40
Frequency (imp/sec)

Fig. 45 Instantaneous frequency plot of the tonic-expiratory laryngeal motoneurone in Fig. 41 (record D), showing how the impulse frequency changed during lung deflation. Arrow indicates the end of the inspiratory phase of the respiratory cycle.

INSTANTANEOUS IMPULSE FREQUENCY IN A CONTROL BREATH



INSTANTANEOUS IMPULSE FREQUENCY DURING LUNG DEFLATION



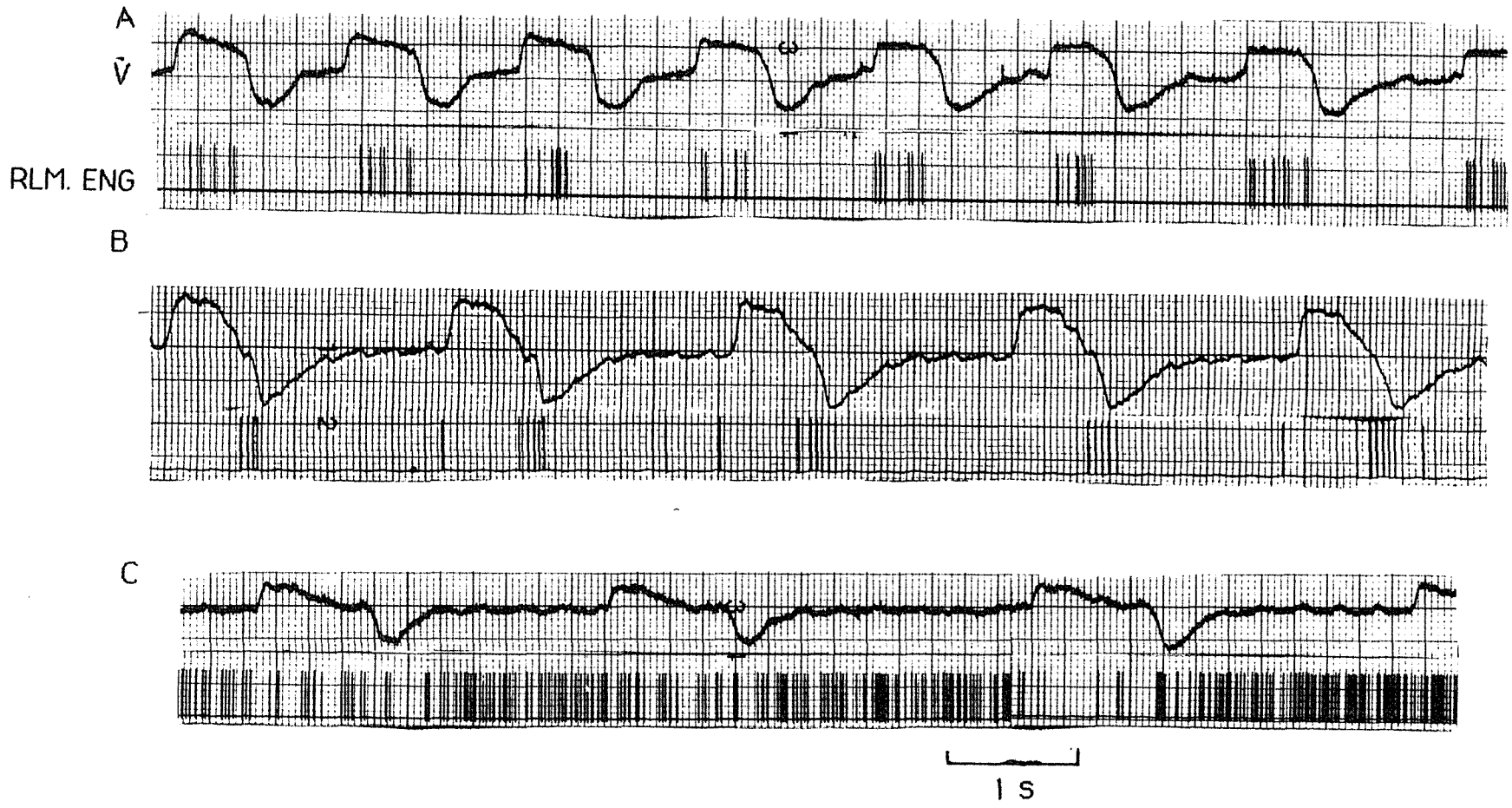


Fig. 46a Spontaneous discharge of three recurrent laryngeal motoneurones before pulmonary stretch receptor block. A. Phasic-inspiratory laryngeal motoneurone, B. Phasic-expiratory laryngeal motoneurone, C. Tonic-expiratory laryngeal motoneurone. Rabbits under pentobarbitone anaesthesia. In each record, upper trace: airflow ($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM.ENG).

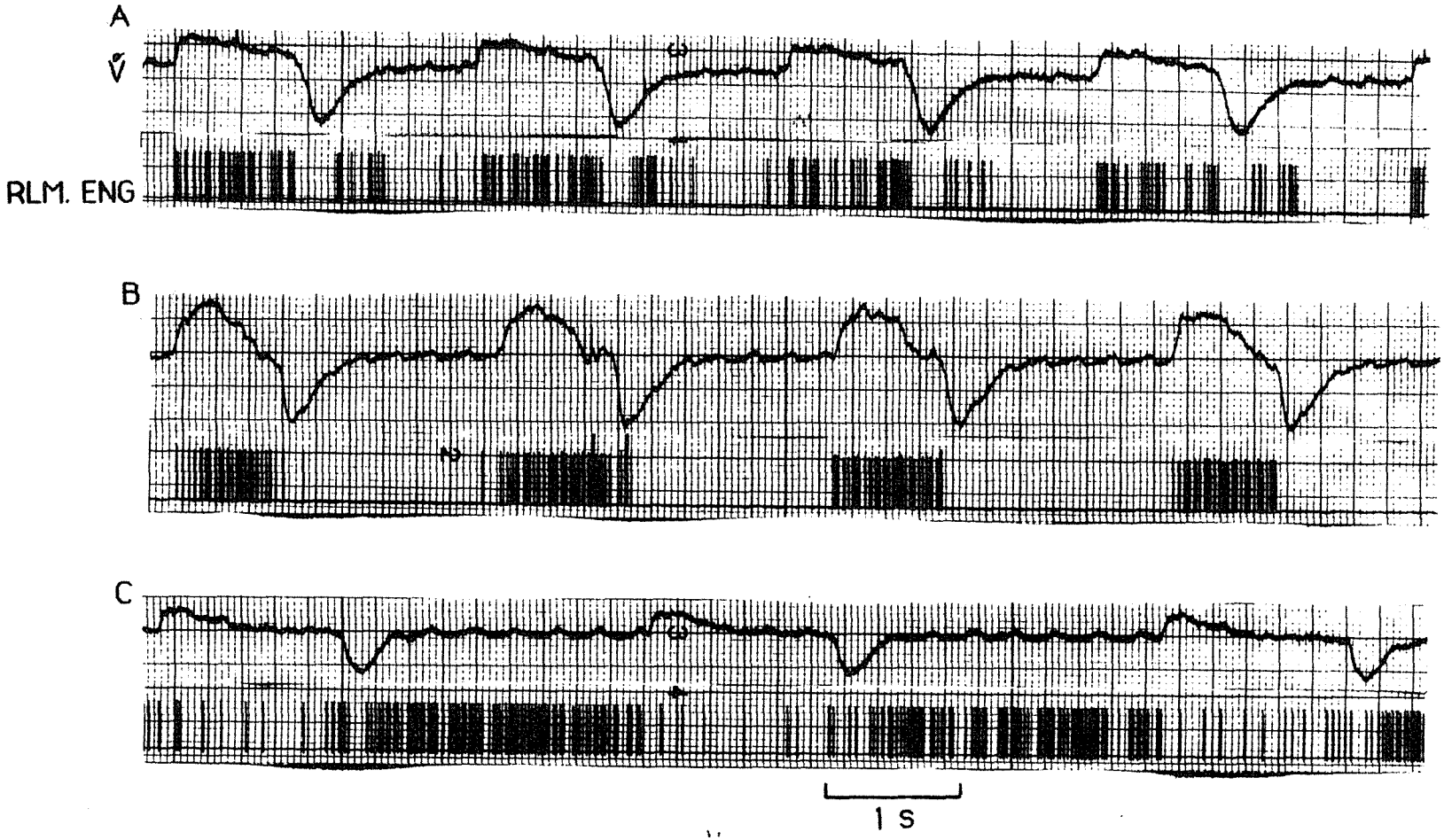
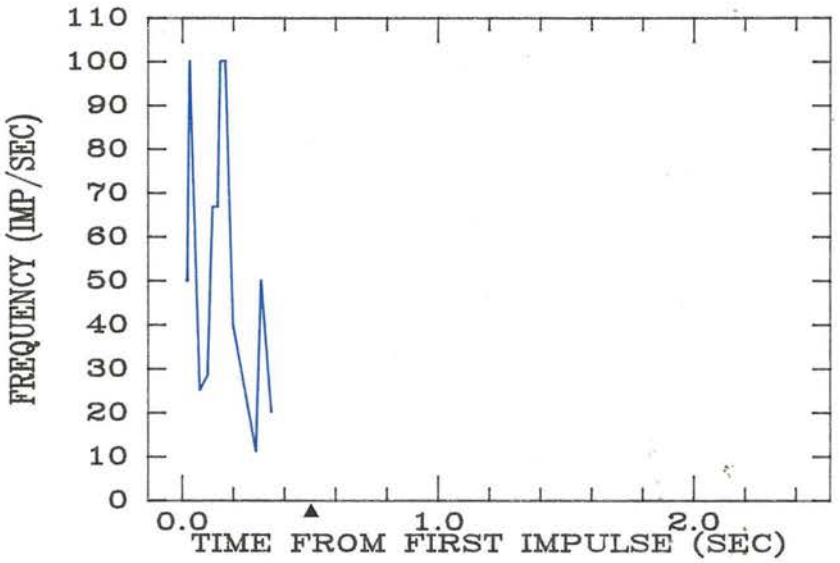


Fig. 46b Effect of pulmonary stretch receptor block with sulphur dioxide on the spontaneous discharge of the three motoneurones in Fig. 46a. A: A phasic-inspiratory laryngeal motoneurone. B: A phasic-expiratory laryngeal motoneurone. C: A tonic-expiratory laryngeal motoneurone. In each record, upper trace: airflow($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM. ENG). Rabbits under pentobarbitone anaesthesia.

Fig. 47 Instantaneous frequency plot of the phasic-inspiratory laryngeal motoneurone in Figs. 46a,b (records A), showing how the impulse frequency changed when pulmonary stretch receptors were blocked. Arrow indicates the end of the inspiratory phase of the respiratory cycle.

INSTANTANEOUS IMPULSE FREQUENCY
IN A CONTROL BREATH



INSTANTANEOUS IMPULSE FREQUENCY
WHEN STRETCH RECEPTORS WERE BLOCKED

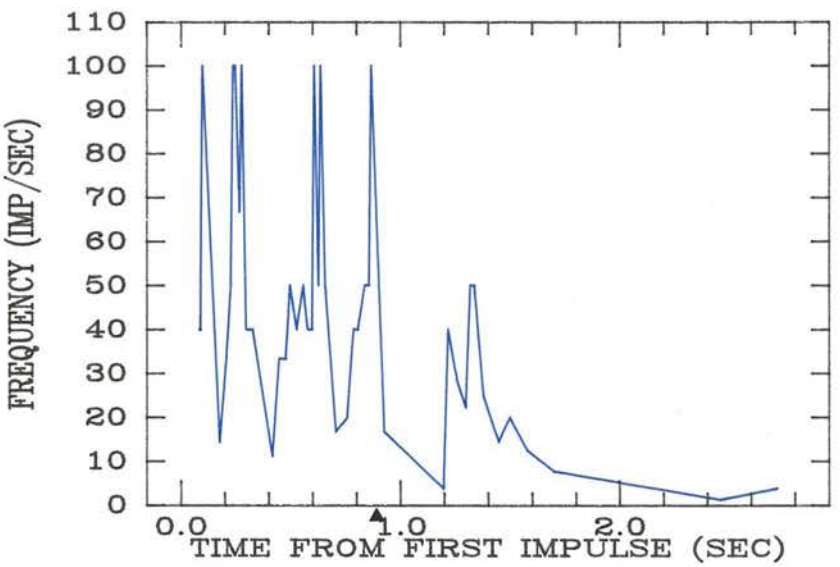


Fig. 48 Effect of pulmonary stretch receptor block by exposure to 200 ppm sulphur dioxide on the frequency of P-ELM discharge in different quartiles of the inspiratory (I.P) and expiratory (E.P) phases of the respiratory cycle during eupnoeic breathing (N = 1 fibre).

 (PSR intact)

 (PSR blocked)

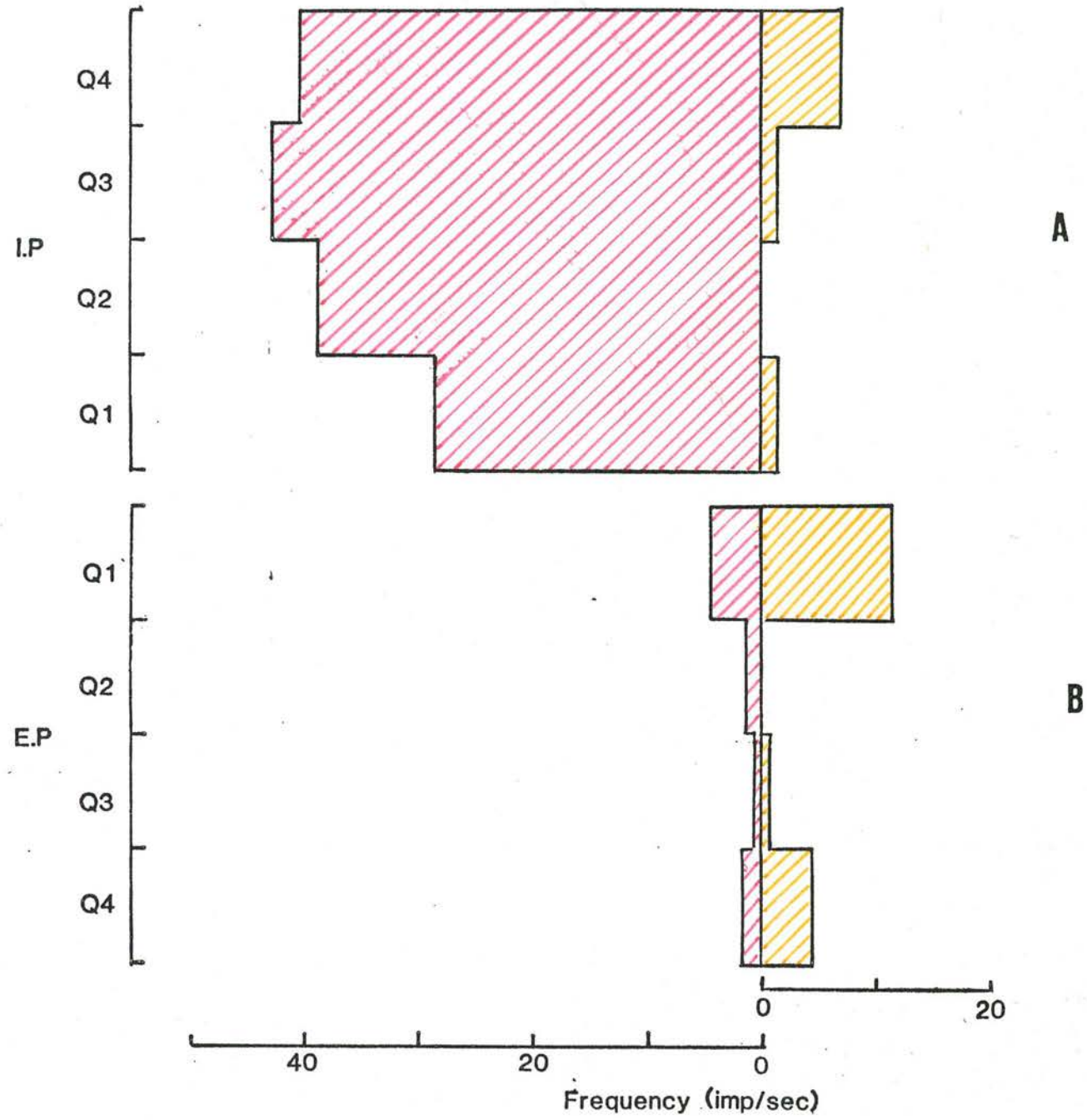
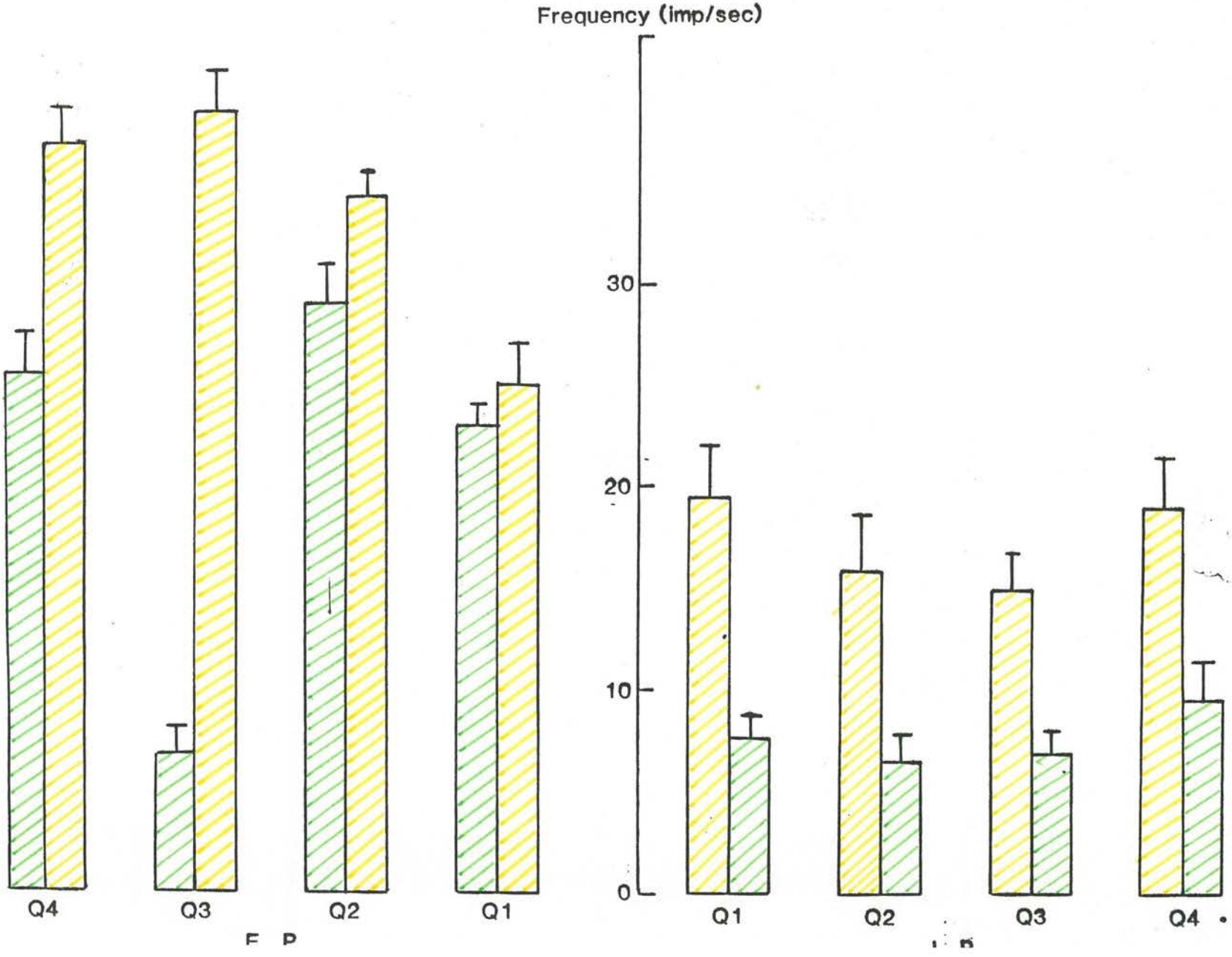

Q4
Q3
I.P
Q2
Q1
A
Q1
Q2
E.P
Q3
Q4
B
0
20
40
20
0
Frequency (imp/sec)

Fig. 49 Effect of pulmonary stretch receptor block by 20 minutes exposure to 200 ppm sulphur dioxide on the mean frequency of tonic-expiratory laryngeal motoneurone discharge in different quartiles of the inspiratory (I.P) and expiratory (E.P) phases of the respiratory cycle. Vertical bars represent standard errors.

Stretch receptors intact

Stretch receptors blocked


Frequency (imp/sec)
30
20
10
0
Q4
Q3
Q2
Q1
Q1
Q2
Q3
Q4

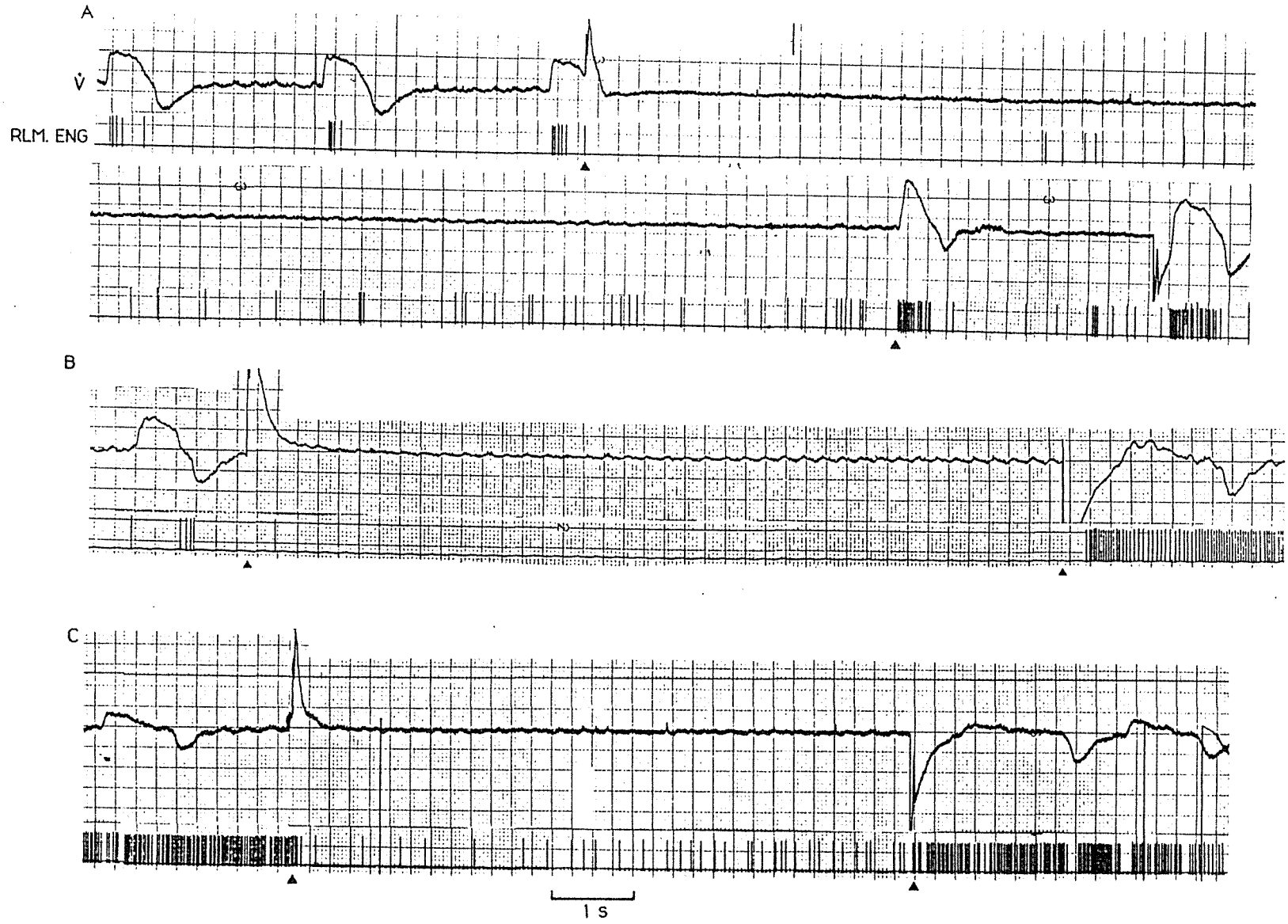


Fig. 50a Response to lung inflation before stretch receptor block (arrows indicate onset and release of lung inflation). A: Phasic-inspiratory laryngeal motoneurone (upper and lower panels are from the same record), B: Phasic-expiratory laryngeal motoneurone, C: Tonic-expiratory laryngeal motoneurone. In each record, upper trace: airflow ($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM. ENG). Rabbits under pentobarbitone anaesthesia. The artifact produced by the opening and closing of the magnetic valves is clearly seen on the airflow record.

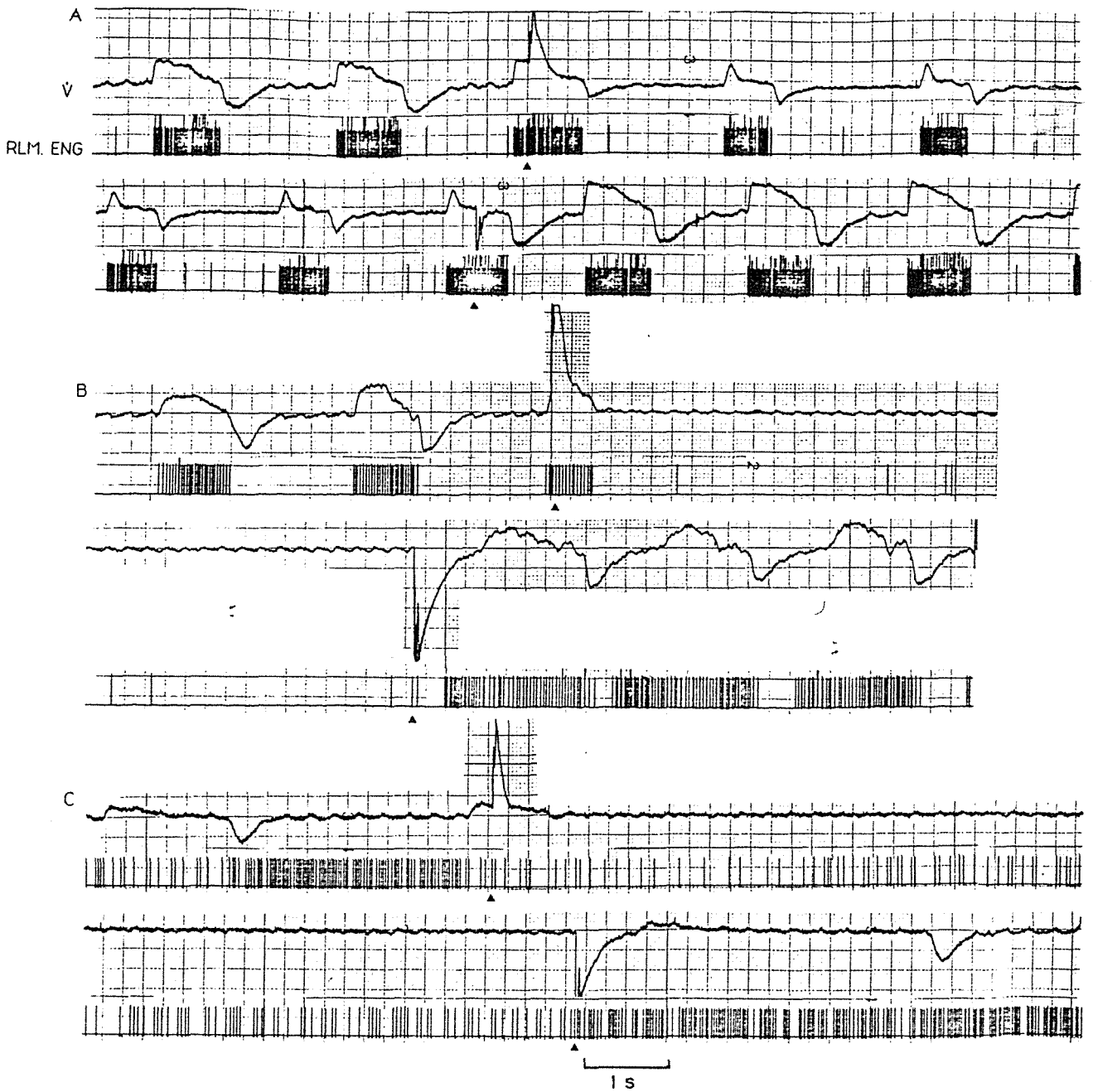


Fig. 50b Response of the three motoneurones in Fig. 50a to lung inflation (between the arrows) during stretch receptor block. A: Phasic-inspiratory laryngeal motoneurone, B: Phasic-expiratory laryngeal motoneurone, C: Tonic-expiratory laryngeal motoneurone. In each record, upper and lower panels are continuous record; upper trace: airflow ($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM.ENG). The artifact produced by the opening and closing of the magnetic valves is seen on the airflow record. Rabbits under pentobarbitone anaesthesia.

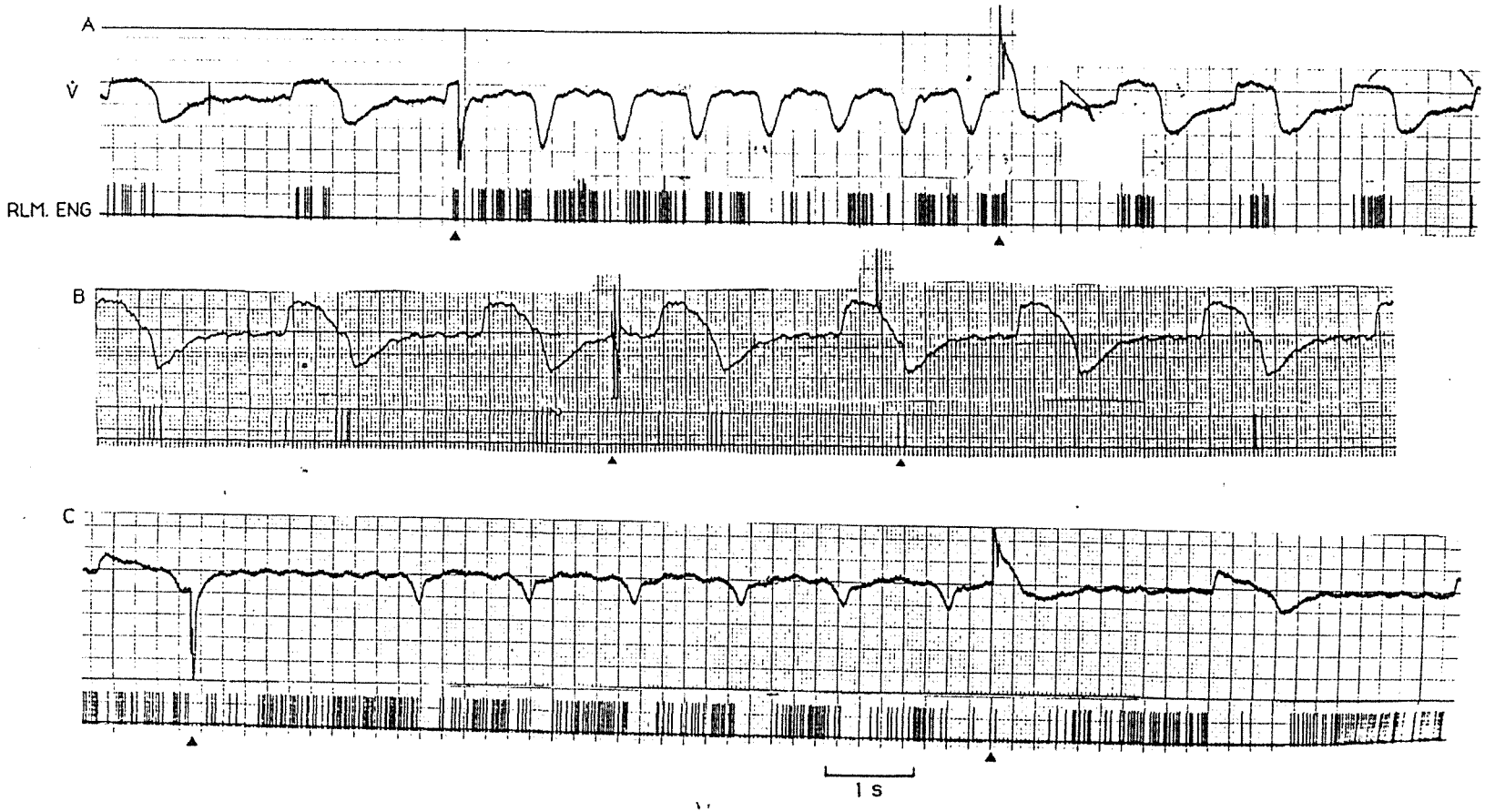


Fig. 51a Response to lung deflation (between the arrows) before stretch receptor block. A: Phasic-inspiratory laryngeal motoneurone. B: Phasic-expiratory laryngeal motoneurone. C: Tonic-expiratory laryngeal motoneurone. In each record, upper trace: airflow ($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM. ENG). Rabbits under pentobarbitone anaesthesia. The artifact produced by the opening and closing of the magnetic valves is clearly seen on the airflow trace.

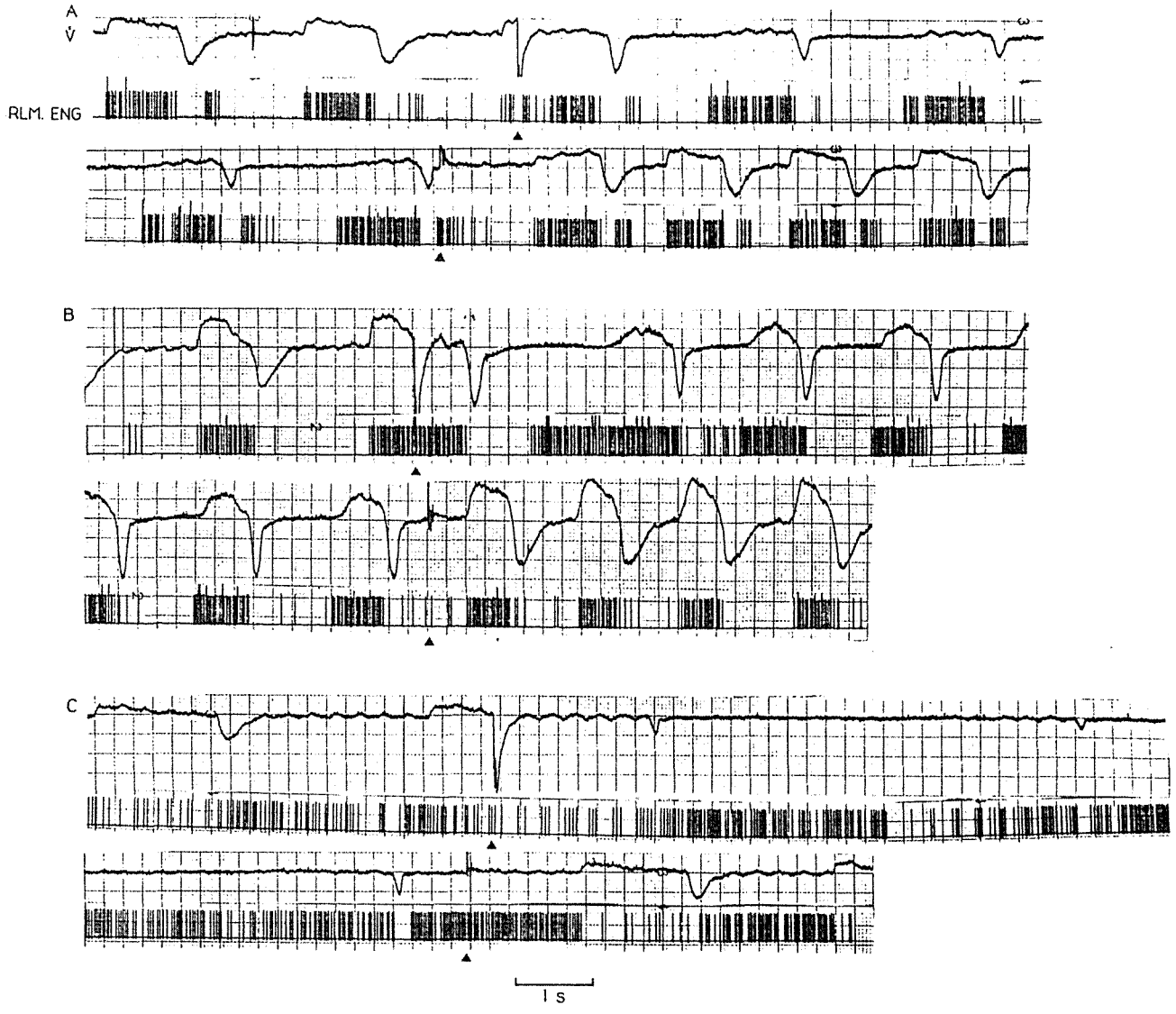
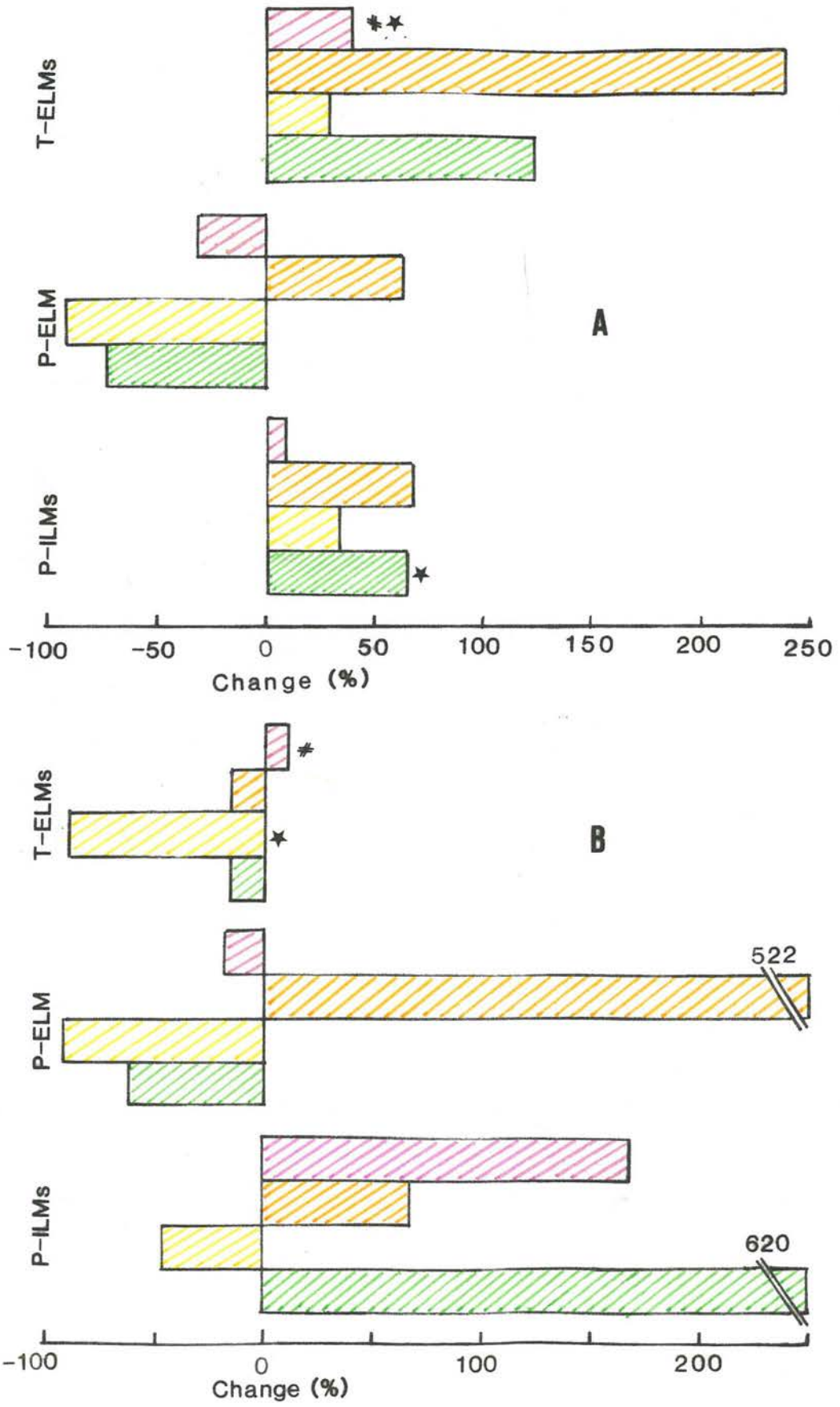


Fig. 51b Response of the three motoneurones in Fig. 51a to lung deflation (between the arrows) during stretch receptor block. A: Phasic-inspiratory laryngeal motoneurone, B: Phasic-expiratory laryngeal motoneurone, C: Tonic-expiratory laryngeal motoneurone. In each record, upper and lower panels are from the same record; upper trace: airflow ($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM. ENG). Rabbits under pentobarbitone anaesthesia. The artifact produced by the opening and closing of the magnetic valves is clearly seen on the airflow record.

Fig. 52 Mean percentage changes in frequency and duration of recurrent laryngeal motoneurone discharge in inspiration and expiration caused by lung deflation before and during stretch receptor block. Significance of difference of response to lung deflation : * $P < 0.05$. Significance of difference of response to lung deflation in blocked condition compared iwth control : # $P < 0.05$.
A. Inspiratory discharge
B. Expiratory discharge


T-ELMs
P-ELM
P-ILMs
A
B
-100
-50
0
50
100
150
200
250
Change (%)
522
620

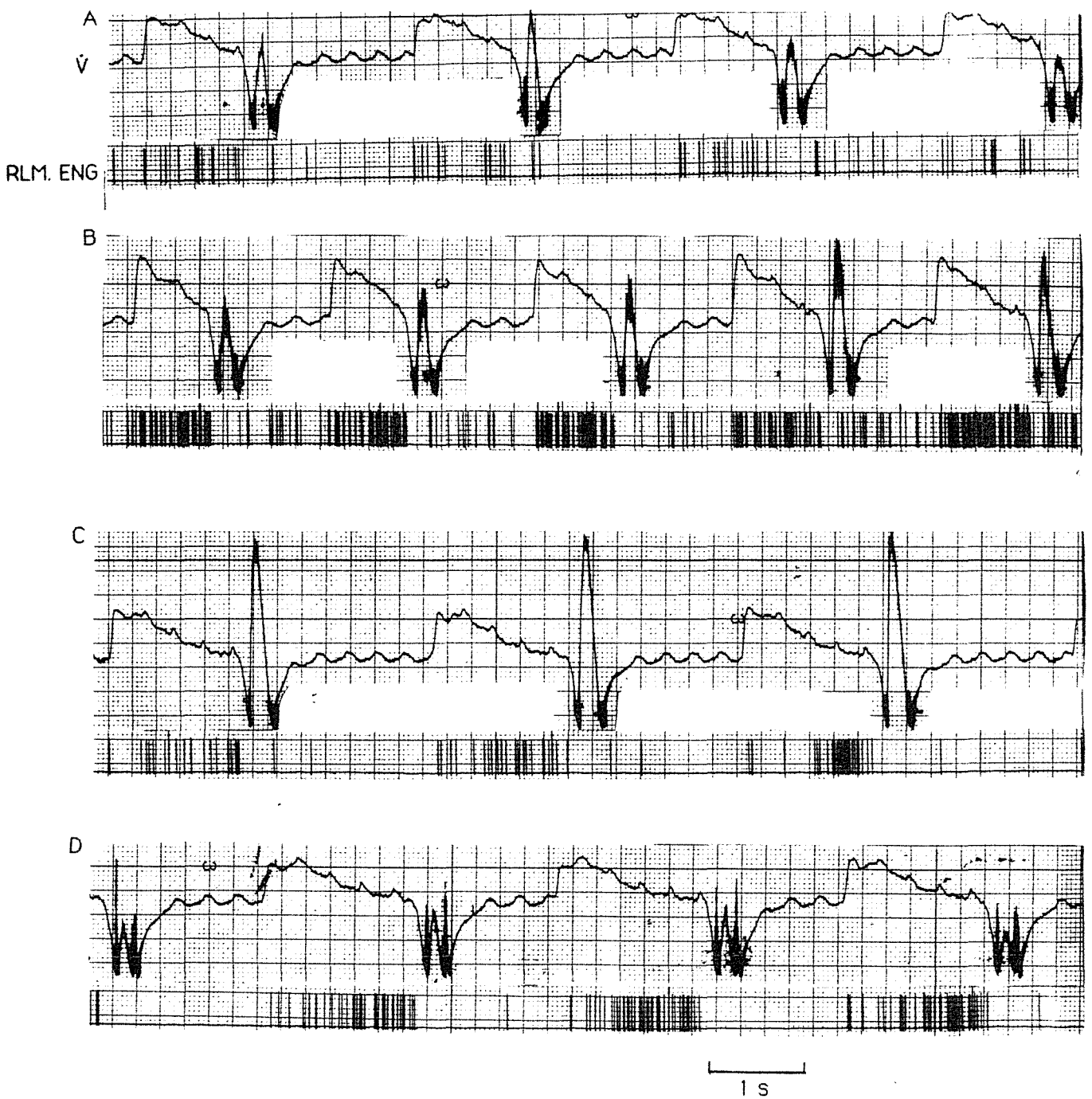
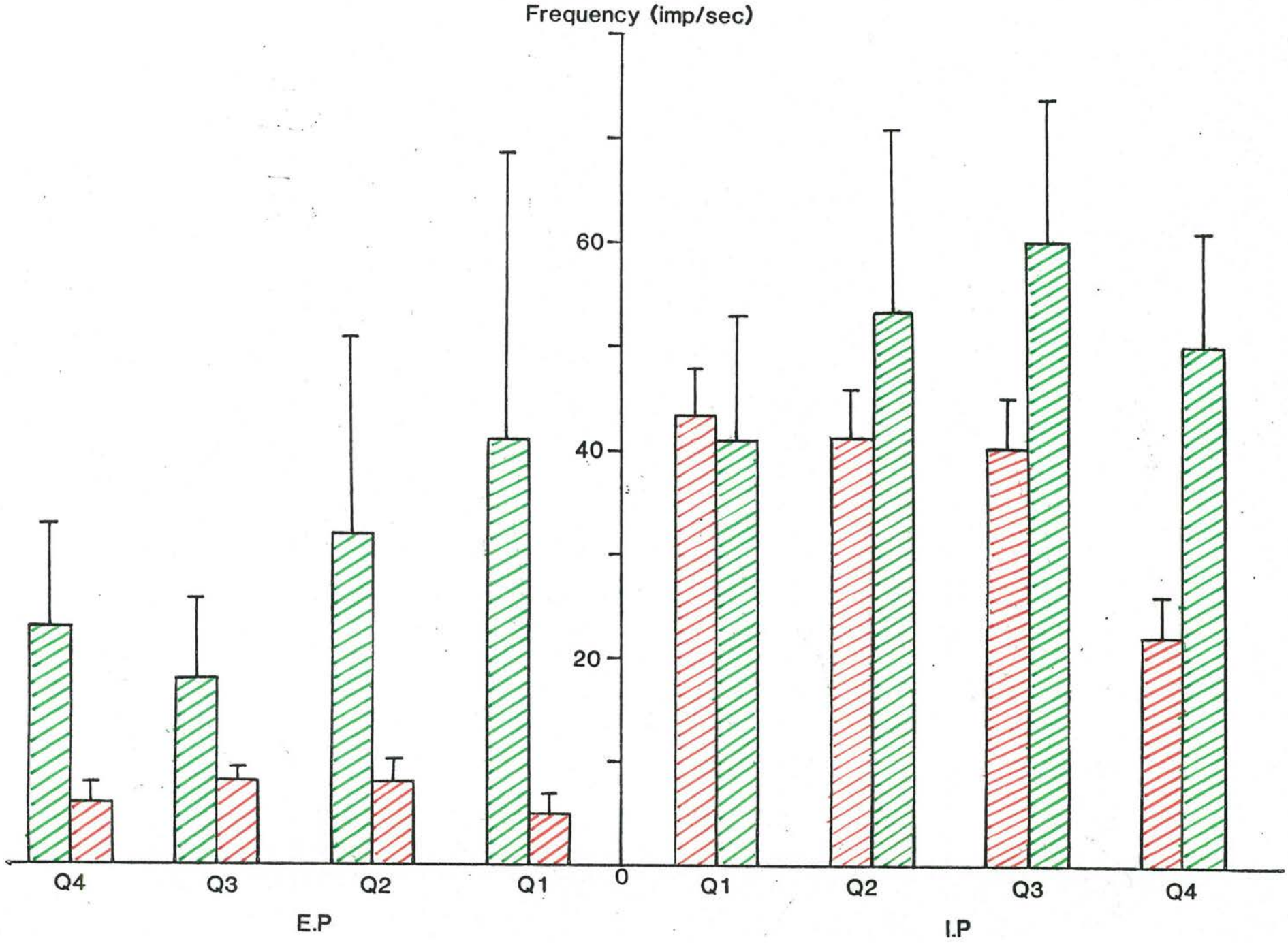


Fig. 53 Effect of breathing through an added dead space on the discharge patterns of a phasic-inspiratory laryngeal motoneurone before and during stretch receptor (PSR) block. A: During control breathing (PSR intact). B: During hypercapnia (PSR intact). C: During control breathing (PSR block). D: During hypercapnia (PSR block). In each record, upper trace: airflow ($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM.ENG). Rabbit under pentobarbitone anaesthesia. Artifacts are clearly seen on airflow traces.

Fig. 54 Effect of breathing through an added dead space on the mean frequency of P-ULM discharge in different quartiles of the inspiratory (I.P) and expiratory (E.P) phases of the respiratory cycle during stretch receptor block. Vertical bars represent standard errors.

During a control breath

During hypercapnia


Frequency (imp/sec)
60
40
20
0
Q4
Q3
Q2
Q1
E.P
Q1
Q2
Q3
Q4
I.P

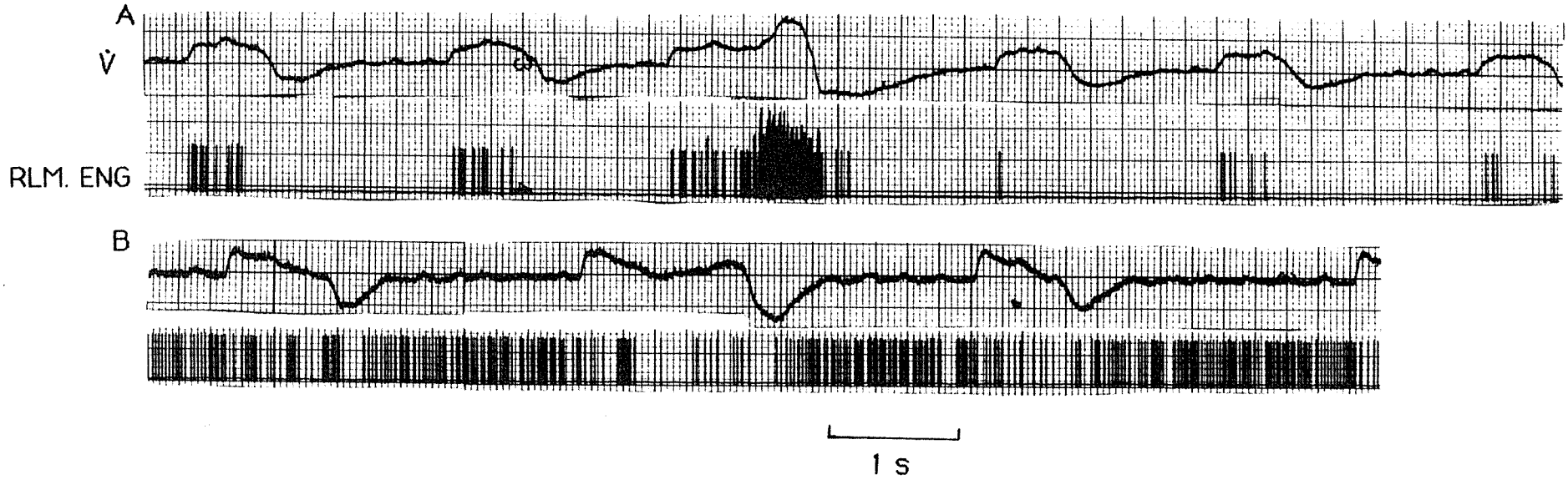


Fig. 55 Discharge patterns of recurrent laryngeal motoneurones during spontaneous augmented breaths. A: A phasic-inspiratory laryngeal motoneurone, B: A tonic-expiratory laryngeal motoneurone. In each record, upper trace: airflow ($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM.ENG). Rabbits under pentobarbitone anaesthesia.

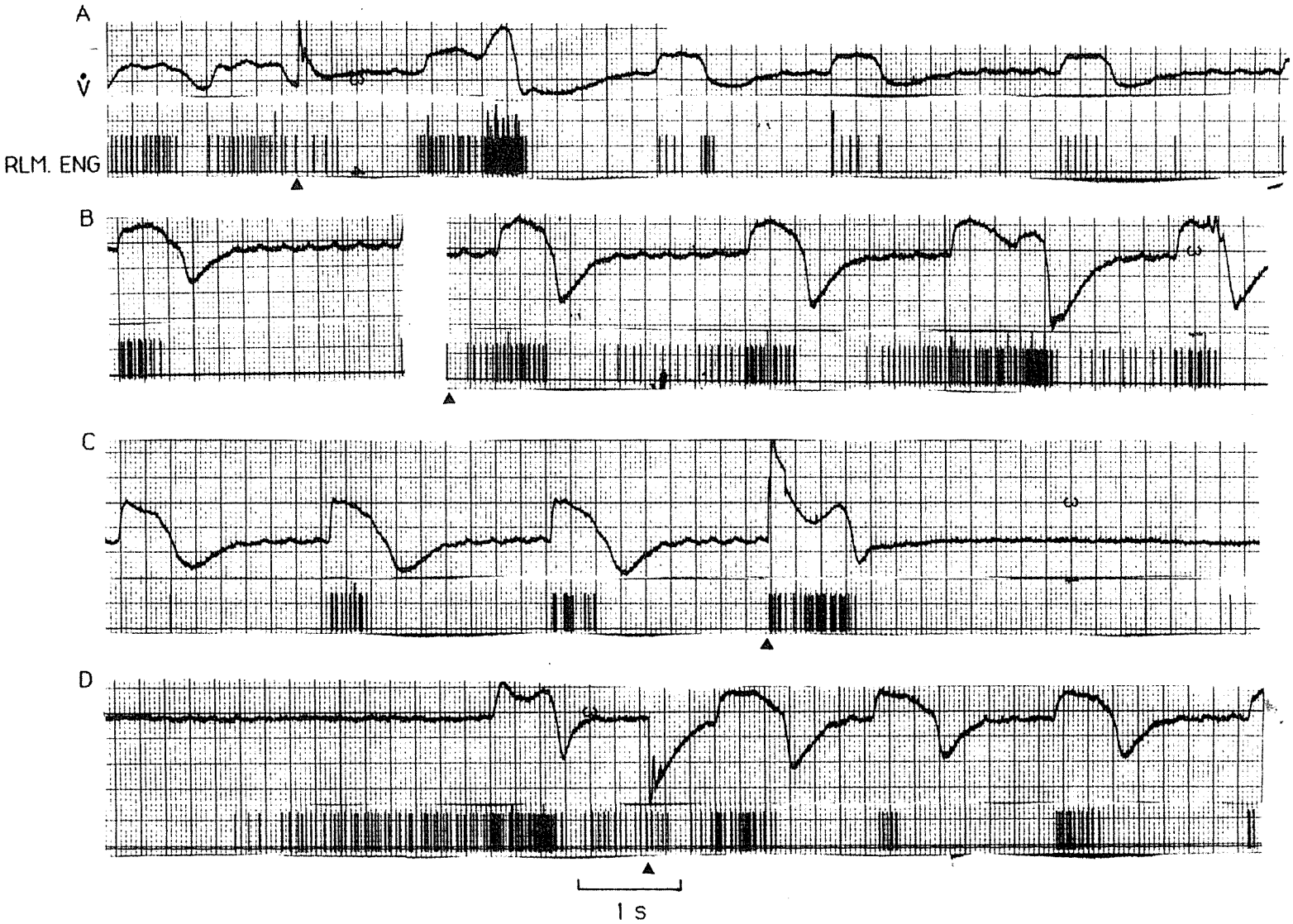
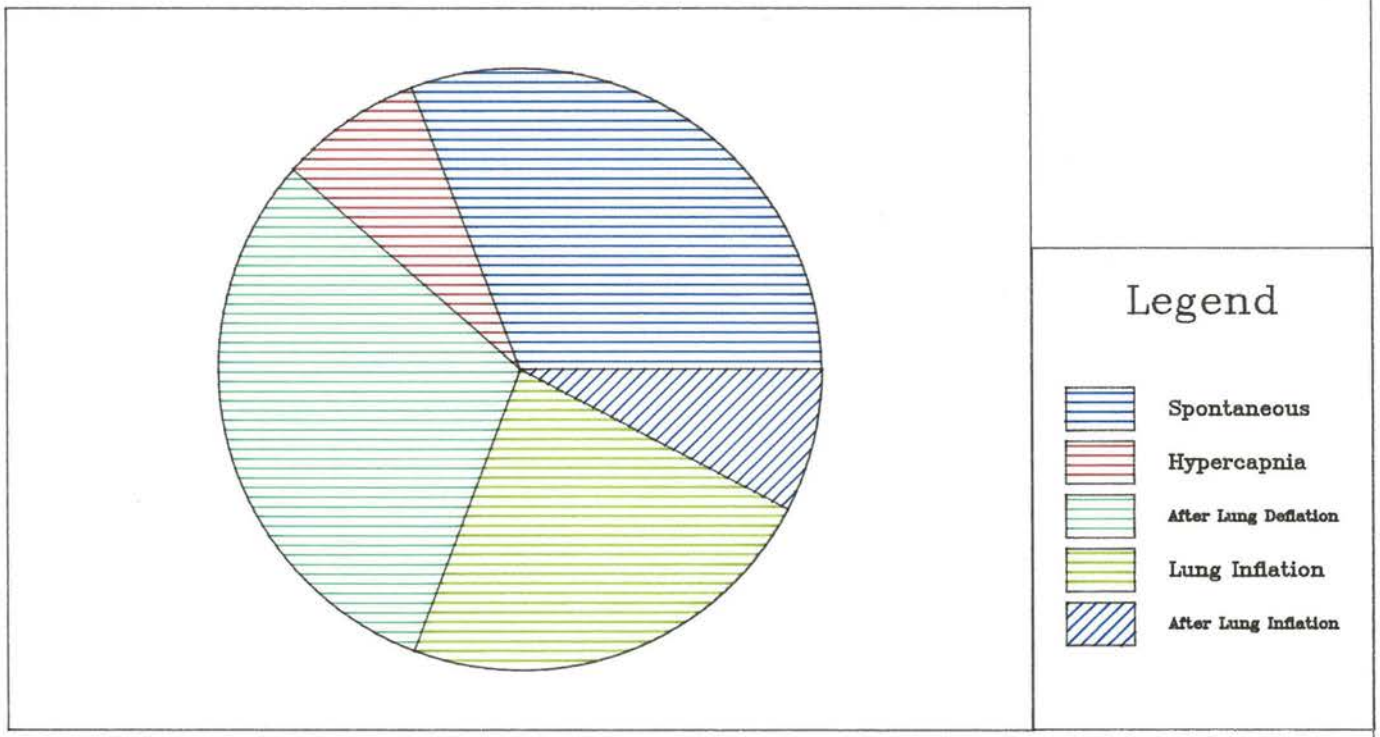


Fig. 56 Record showing the discharge patterns of phasic-inspiratory laryngeal motoneurones during augmented breaths induced by various stimuli. A: After lung deflation (Arrow indicates release of lung deflation), B: During hypercapnia (Arrow indicates the addition of dead space). C: Immediately following lung inflation (indicated by arrow). D: During lung inflation (Arrow indicates release of lung inflation). Rabbits under pentobarbitone anaesthesia. In each record, upper trace: airflow ($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM.ENG).

FIG. 57 VARIOUS STIMULI CAUSING AUGMENTED BREATHS



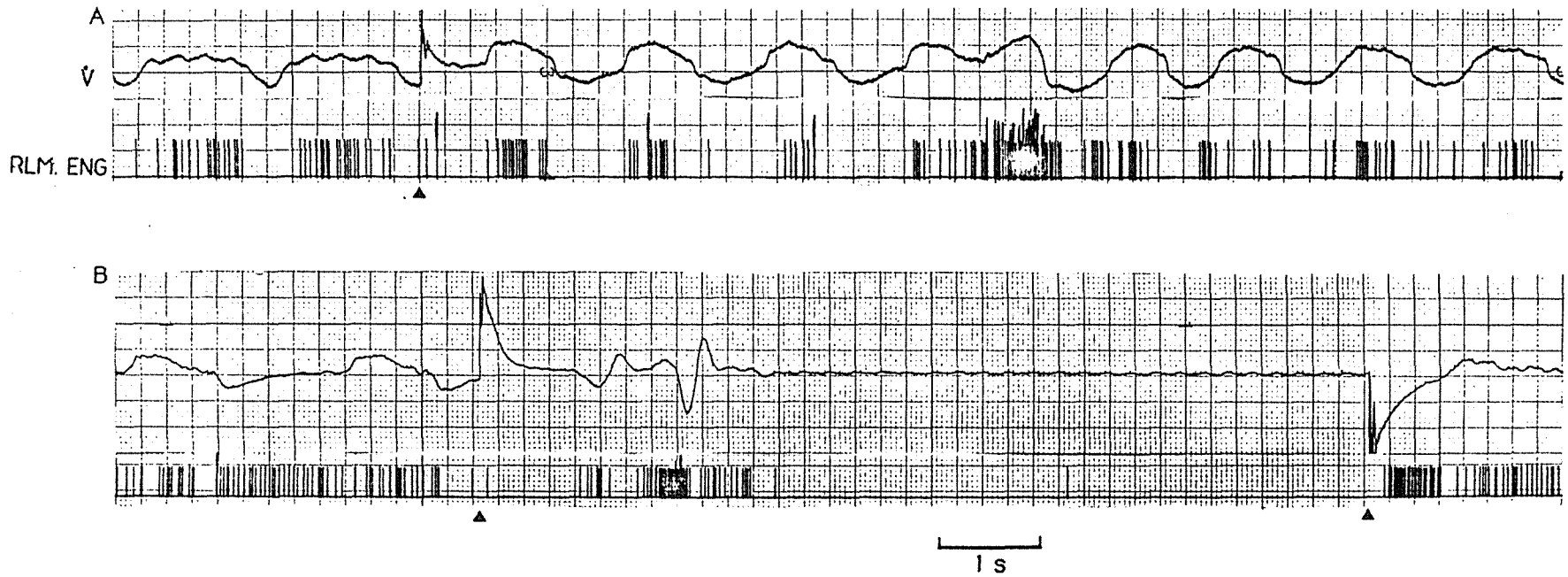


Fig. 58 Discharge patterns of phasic-inspiratory laryngeal motoneurones during augmented breaths when stretch receptors were blocked by sulphur dioxide. A. Augmented breath occurring after lung deflation. B: Augmented breath occurring during lung inflation. Rabbits under pentobarbitone anaesthesia. In each record, upper trace: airflow ($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM. ENG).

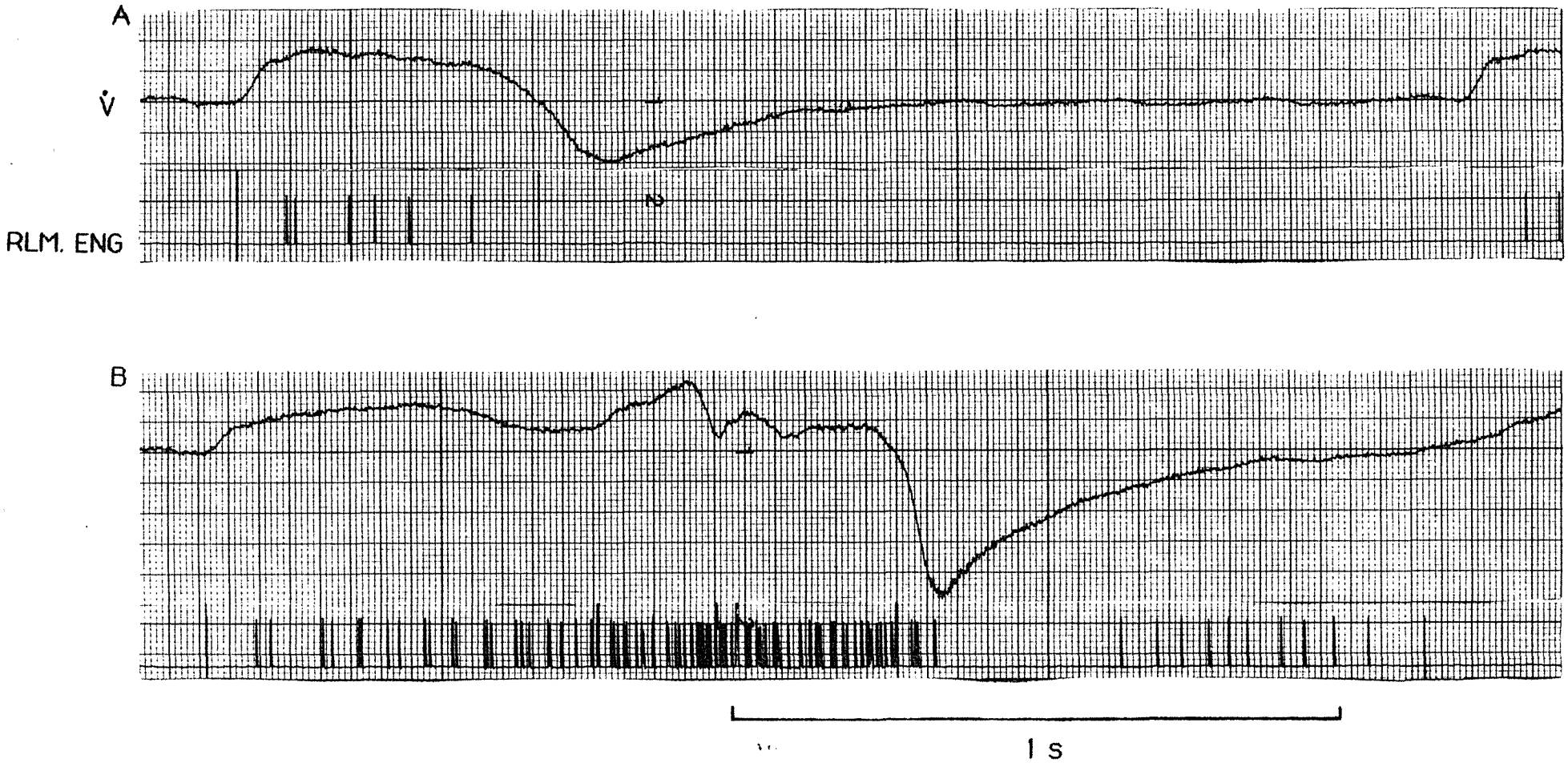
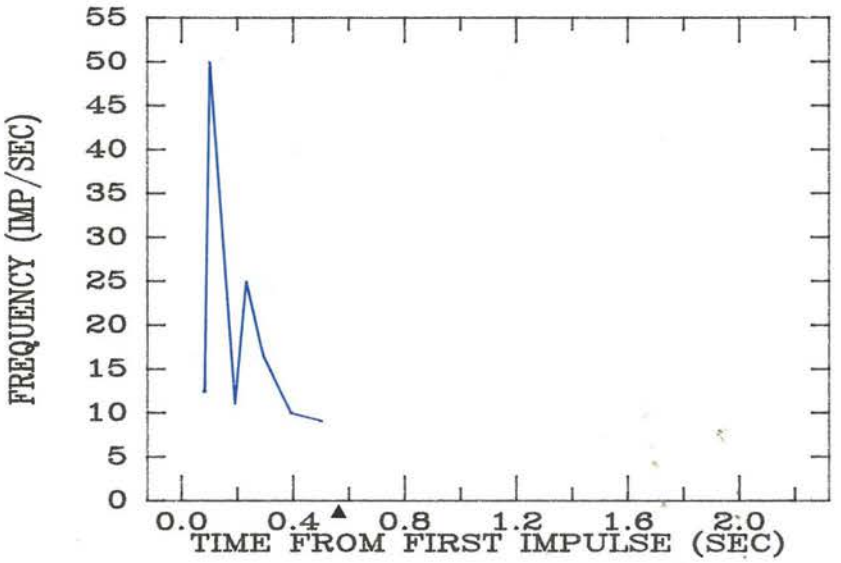


Fig. 59 Record showing sudden acceleration in impulse frequency of a phasic-inspiratory laryngeal motoneurone in the second half of inspiration during an augmented breath. A: Control breath, B: Augmented breath. In each record, upper trace: airflow ($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM. ENG). Rabbit under pentobarbitone anaesthesia.

Fig. 60 Instantaneous frequency plot of the phasic-inspiratory laryngeal motoneurone in Fig. 59, showing how the impulse frequency changed during an augmented breath. Arrow indicates the end of the inspiratory phase of the respiratory cycle.

INSTANTANEOUS IMPULSE FREQUENCY IN A CONTROL BREATH



INSTANTANEOUS IMPULSE FREQUENCY IN A SPONTANEOUS AUGMENTED BREATH

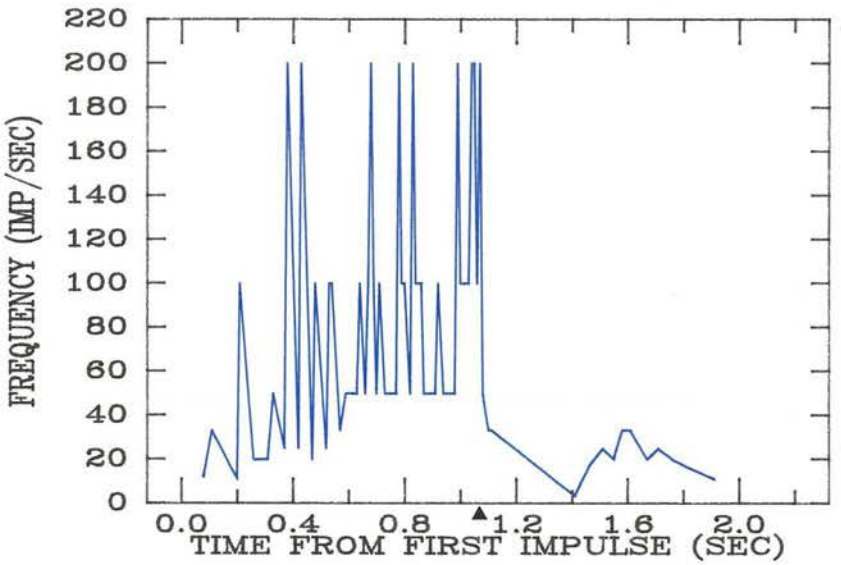


Fig. 61 Changes in mean frequency of phasic-inspiratory laryngeal motoneurone discharge in different quartiles of the inspiratory (I.P) and expiratory (E.P) phases of the respiratory cycle during augmented breaths (9 breaths). Vertical bars represent standard errors.

Control breath

Augmented breath

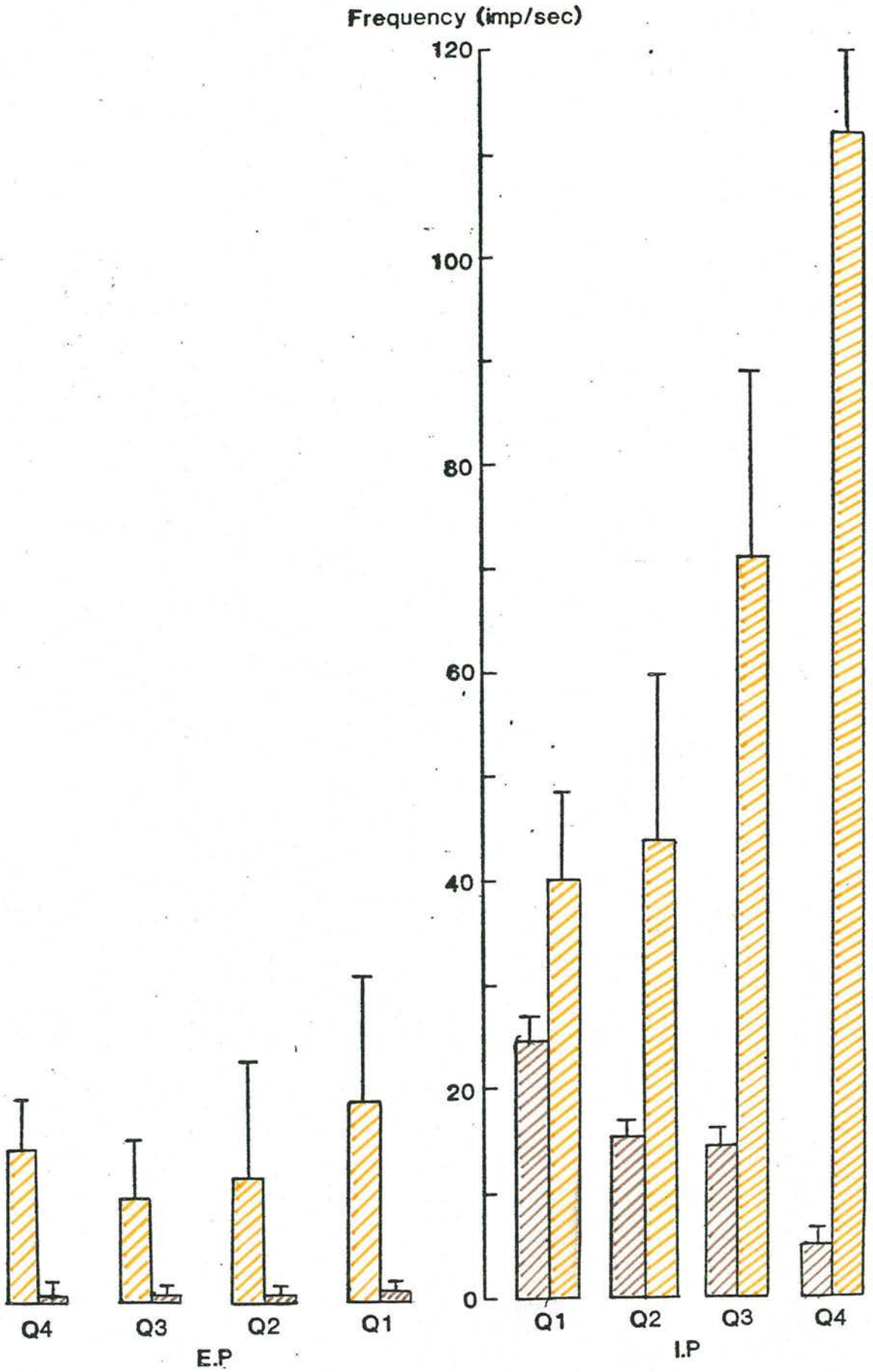
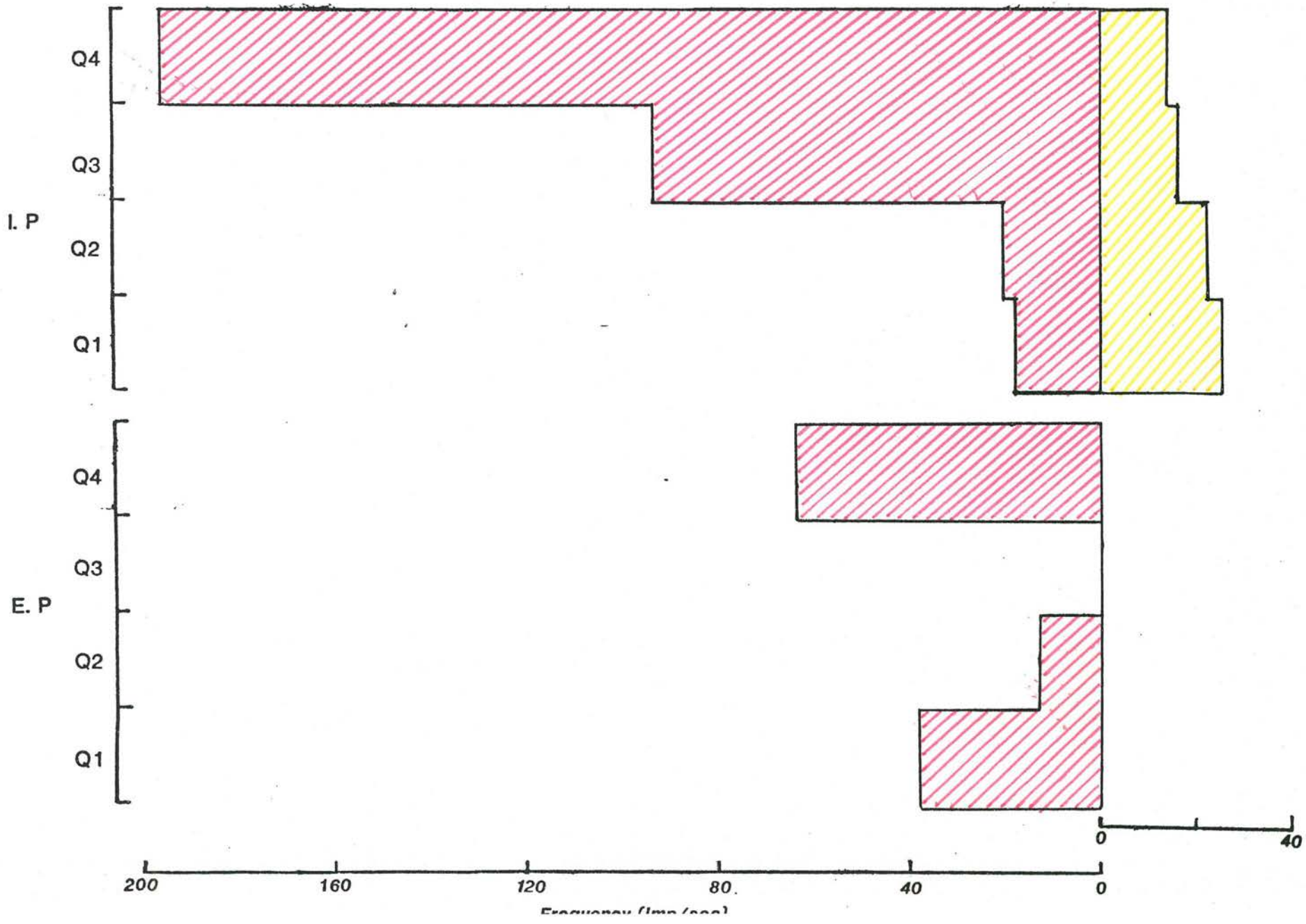

Frequency (imp/sec)
120
100
80
60
40
20
0
Q4
Q3
Q2
Q1
E.P
Q1
Q2
Q3
Q4
I.P

Fig. 62 Frequency of P-LM discharge in different quartiles of the inspiratory (I.P) and expiratory (E.P) phases of the respiratory cycle during an augmented breath after 20 minutes exposure to 200 ppm sulphur dioxide. (N= 1 breath).

During eupnoeic breathing (PSR blocked)

During augmented breath (PSR blocked)


I. P
Q4
Q3
Q2
Q1
E. P
Q4
Q3
Q2
Q1
0
40
200
160
120
80
40
0

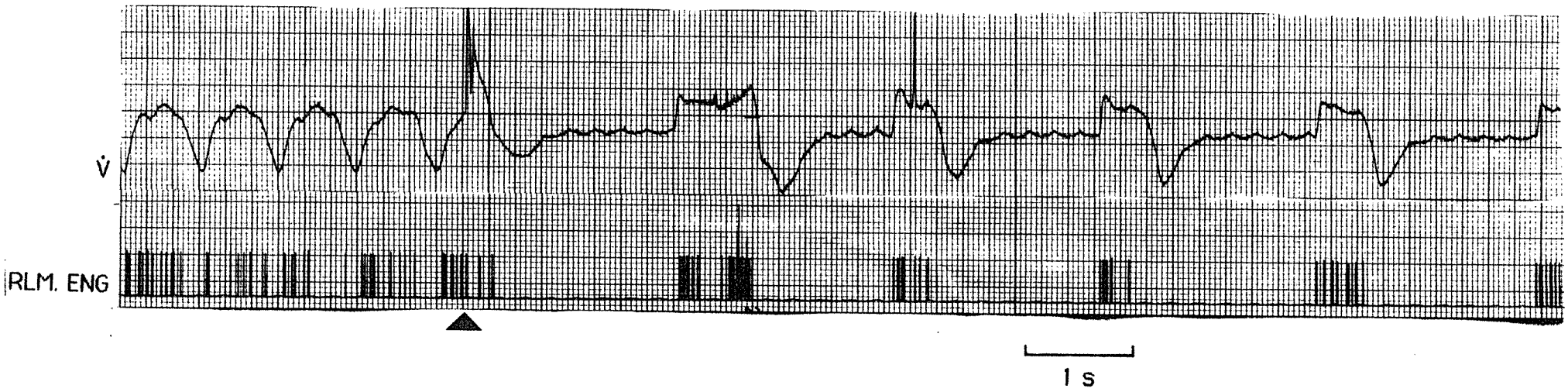


Fig. 63 Record of an augmented breath showing a lessening of phasic-inspiratory laryngeal motoneurone activity before the augmented half of the breath began. Rabbit under pentobarbitone anaesthesia. Upper trace: airflow ($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM. ENG). Arrow indicate the end of lung deflation.

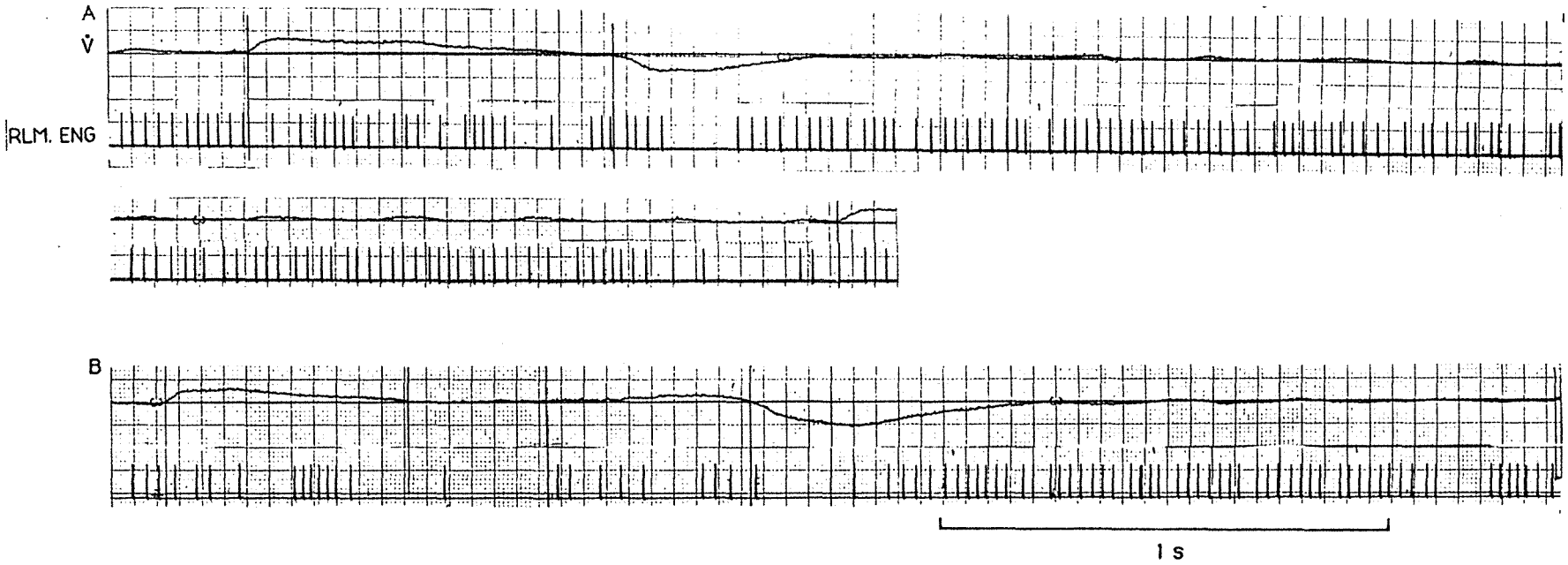
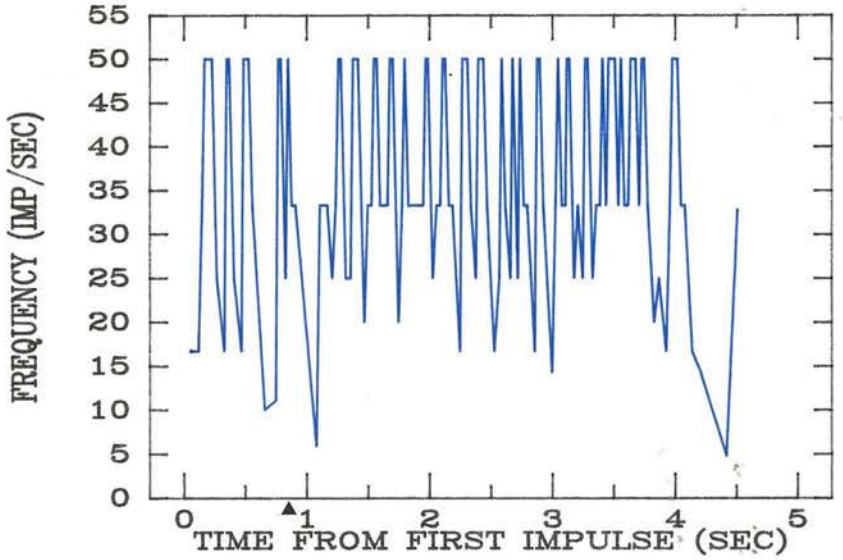


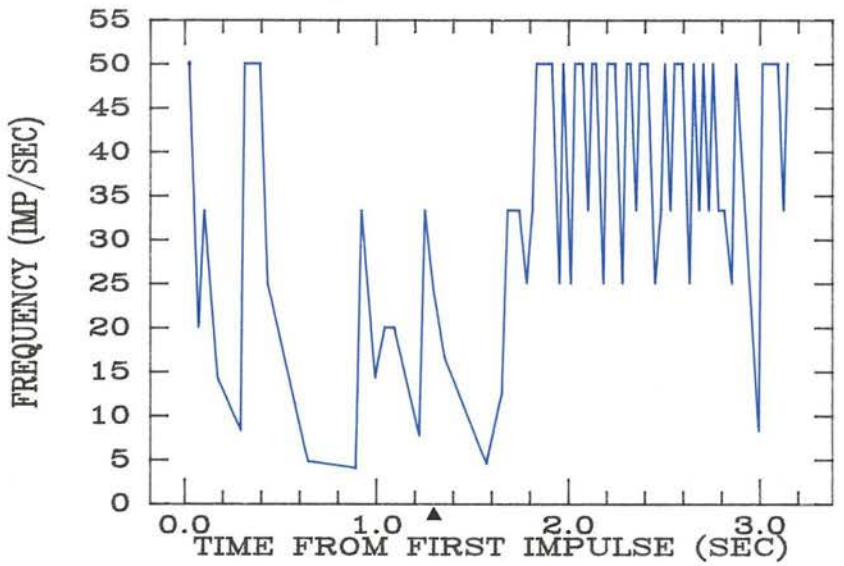
Fig. 64 Discharge patterns of a tonic-expiratory laryngeal motoneurone during a spontaneous augmented breath. A: Control breath (upper and lower panels are continuous record), B: Augmented breath. In each record, upper trace: airflow ($\dot{V}$) signal, inspiration upwards; lower trace: recurrent laryngeal motoneurone electroneurogram (RLM. ENG). Rabbit under pentobarbitone anaesthesia.

Fig. 65 Instantaneous frequency plot of the tonic-expiratory laryngeal motoneurone in Fig. 64, showing how the impulse frequency changed during an augmented breath. Arrow indicates the end of the inspiratory phase of the respiratory cycle.

INSTANTANEOUS IMPULSE FREQUENCY IN A CONTROL BREATH



INSTANTANEOUS IMPULSE FREQUENCY IN A SPONTANEOUS AUGMENTED BREATH



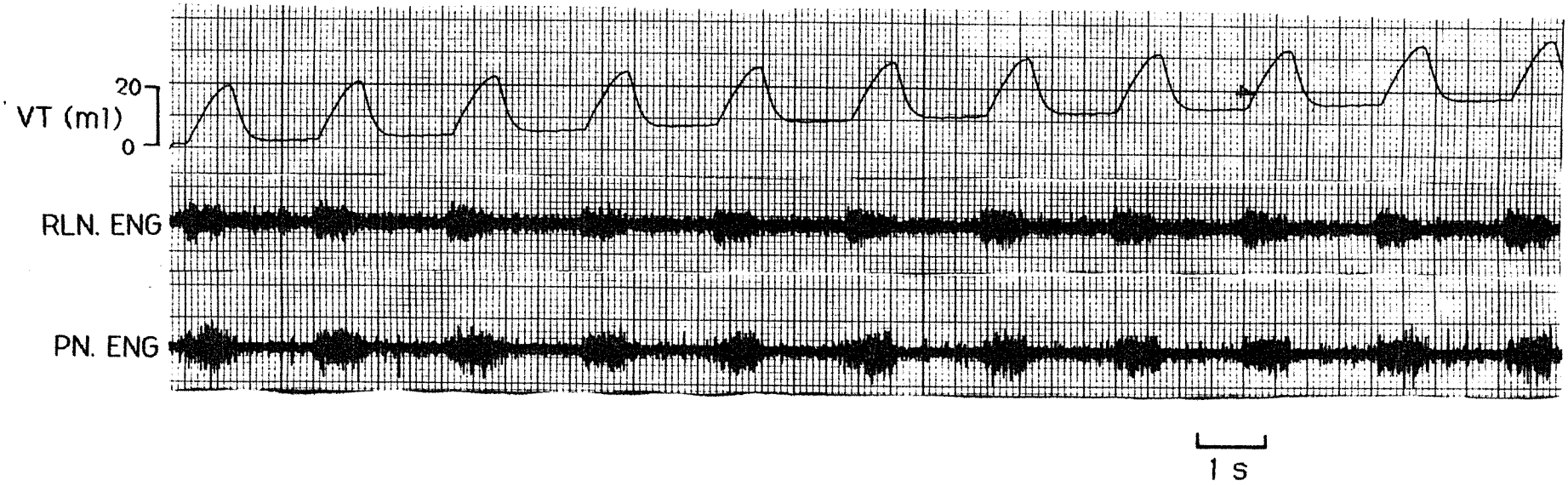


Fig. 66 Spontaneous activity of the recurrent laryngeal and phrenic nerves in relation to tidal volume. Rabbit under pentobarbitone anaesthesia. Upper trace: tidal volume (V_T, inflation upwards). Middle trace: recurrent laryngeal electroneurogram (RLN.ENG). Lower trace: phrenic electroneurogram (PN.ENG).

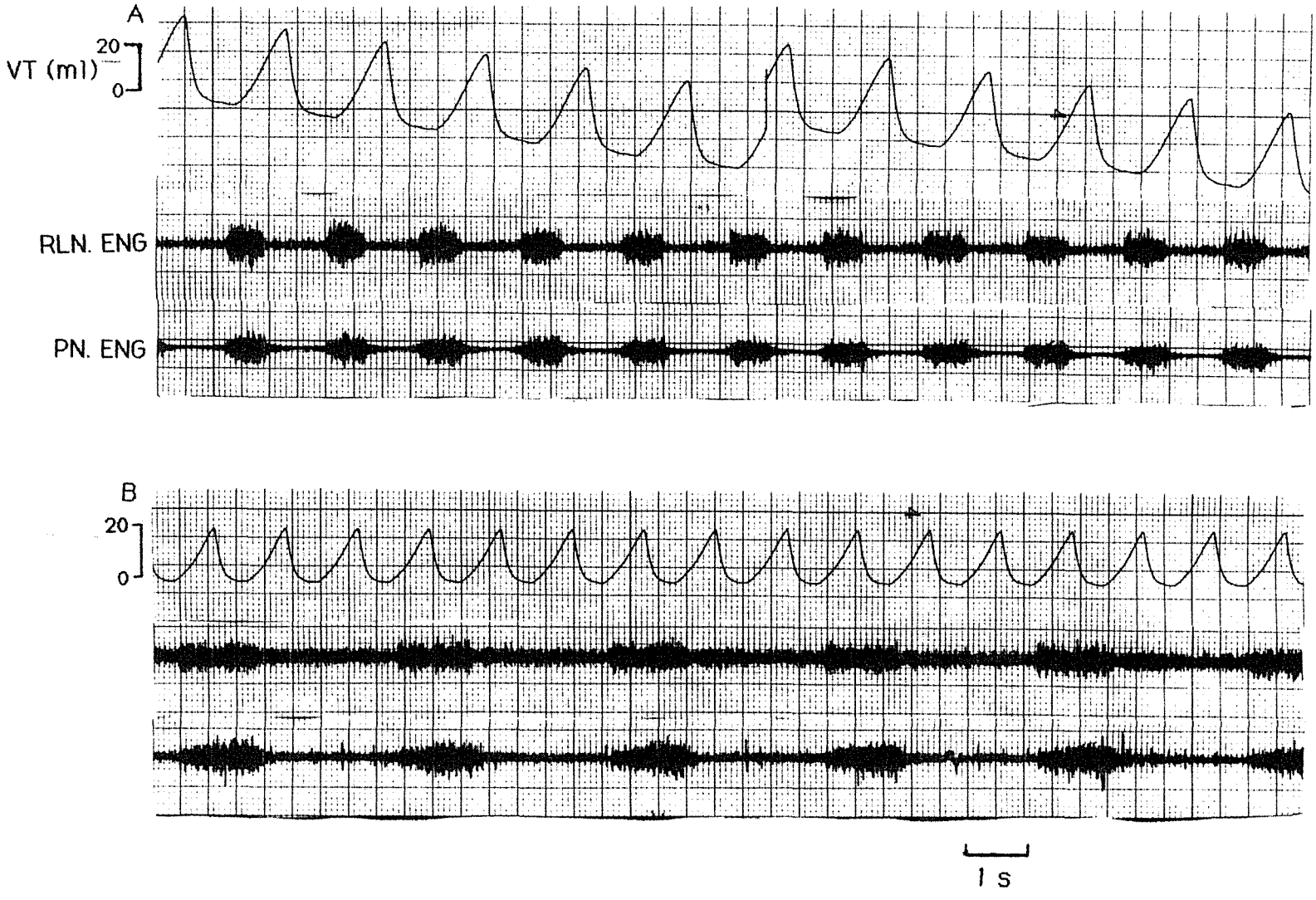


Fig. 67a Anaesthetized rabbits, paralysed and artificially ventilated. Records showing the activity of recurrent laryngeal (RLN) and phrenic nerves (PN) in relation to the inflation and deflation phases of the ventilator. A: 1:1 synchronization of pump and PN/RLN cycles. B: 3:1 synchronization of pump and PN/RLN cycles. In each record, upper trace: tidal volume (V_T), inflation upwards (with some integrator drift and resetting); middle trace: recurrent laryngeal electroneurogram (RLN. ENG); lower trace: phrenic electroneurogram (PN.ENG).

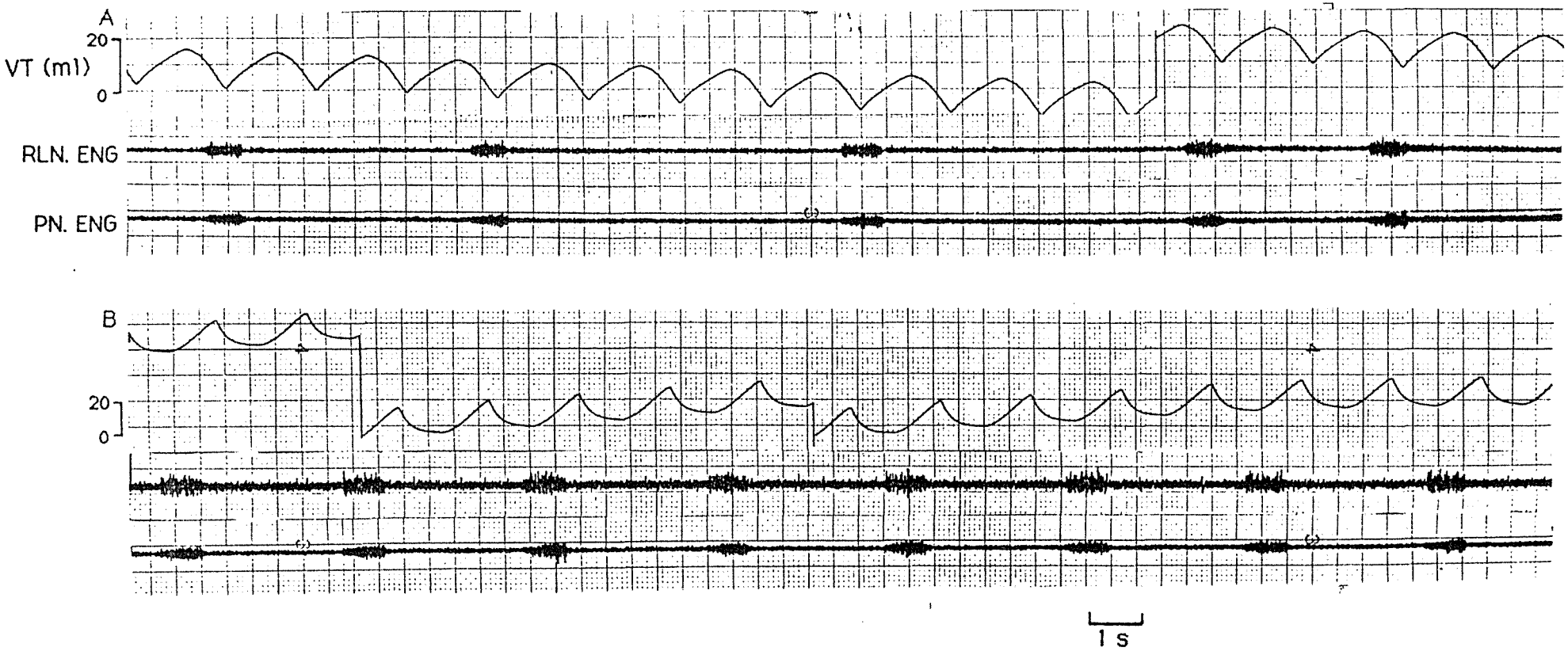
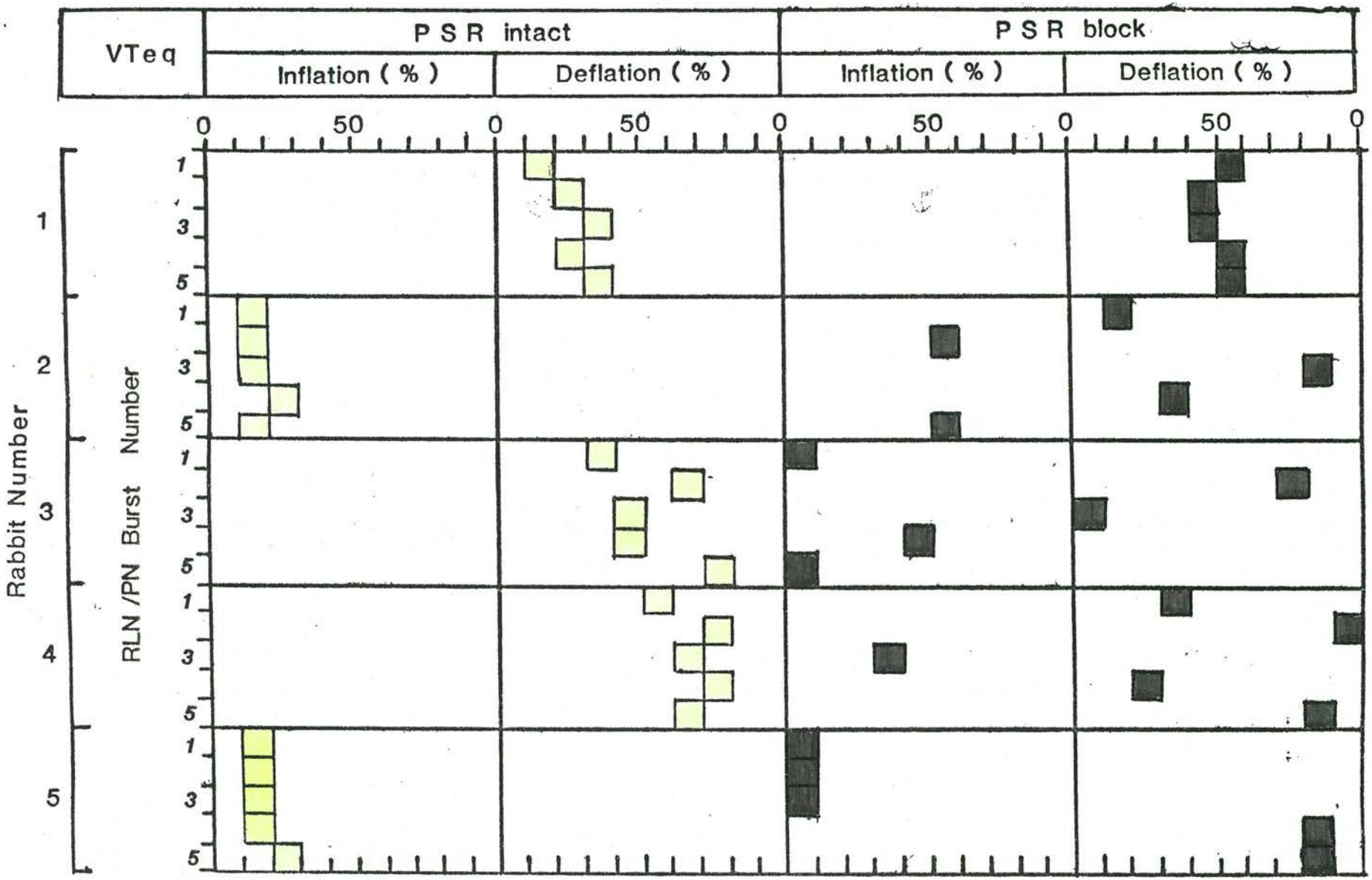


Fig. 67b Anaesthetized rabbits, paralysed and artificially ventilated. Record showing the activity of recurrent laryngeal (RLN) and phrenic nerves (PN) in relation to the inflation and deflation phases of the ventilator. A: 3:1 synchronization of pump and PN/RLN cycles (inflation downwards). B: 2:1 synchronization of pump and PN/RLN cycles (inflation upwards). In each record, upper trace: tidal volume (V_T), with some integrator drift and resetting; middle trace: recurrent laryngeal electroneurogram (RLN.ENG); lower trace: phrenic electroneurogram (PN.ENG).

Fig. 68 The time of onset of the recurrent laryngeal (RLN) and phrenic (PN) bursts in relation to the inflation and deflation phases of the ventilator at spontaneous resting tidal volume (V_{Teq}). Each of the two phases was divided into ten equal intervals and the onsets of RLN and PN bursts were identified in them. This analysis was applied to each of the five experiments in the controls and in total stretch receptor block. Five successive RLN and PN bursts were included for each of the two conditions in each of the five rabbits.


VTeq
P S R intact
P S R block
Inflation (%)
Deflation (%)
Inflation (%)
Deflation (%)
0
50
Rabbit Number
1
2
3
4
5
RLN /PN Burst Number
1
3
5

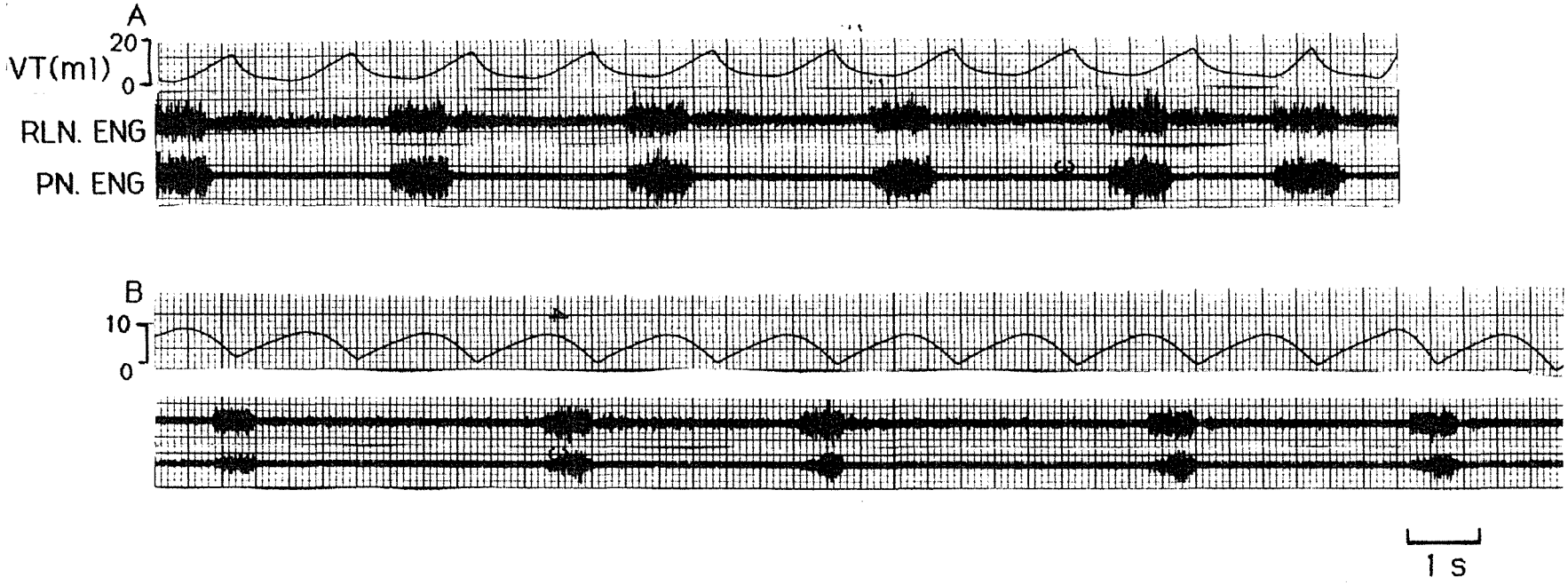
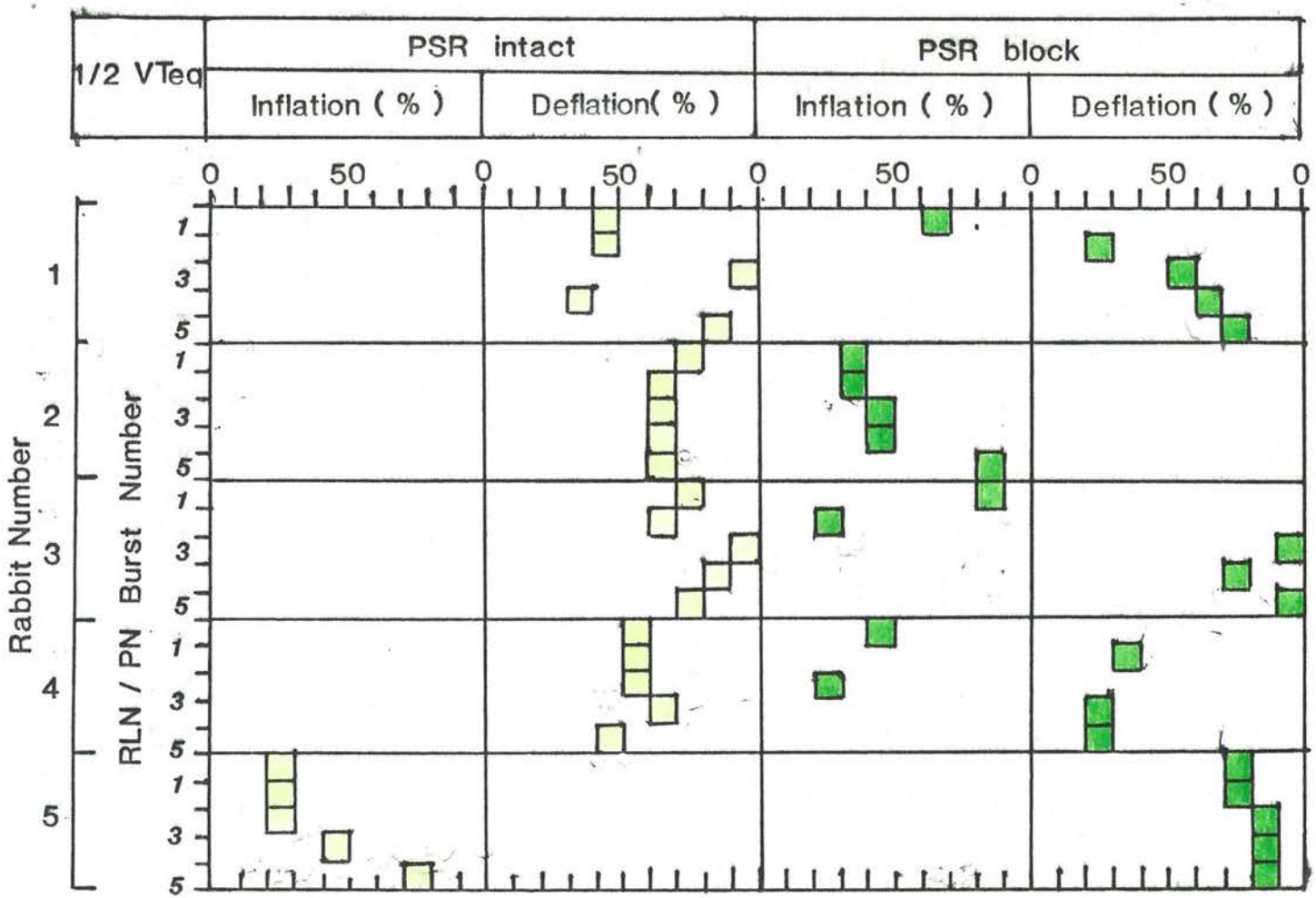


Fig. 69 Anaesthetized rabbits, paralysed and artificially ventilated. Effect of decreasing tidal volume to half of eupnoeic level on the phase relationship between RLN/PN discharge and pump cycle. A: onset of RLN and PN bursts during lung deflation (inflation upwards). B: onset of RLN and PN discharge during lung inflation (inflation downwards). In each record, upper trace: tidal volume (V_T), with some integrator drift and resetting; middle trace: recurrent laryngeal electroneurogram (RLN.ENG); lower trace: phrenic electroneurogram (PN.ENG).

Fig. 70 The time of onset of the recurrent laryngeal (RLN) and phrenic (PN) bursts in relation to the inflation and deflation phases of the ventilator when the tidal volume was at half eupnoeic level (1/2 V_{Teq}). Each of the two phases was divided into ten equal intervals and the onsets of RLN and PN bursts were identified in them. This analysis was applied to each of the five experiments in the controls and in total stretch receptor block. Five successive RLN and PN bursts were included for each of the two conditions in each of the five rabbits.


1/2 VTeq
PSR intact
PSR block
Inflation (%)
Deflation(%)
Inflation (%)
Deflation (%)
0
50
Rabbit Number
RLN / PN Burst Number
1
2
3
4
5
1
3
5

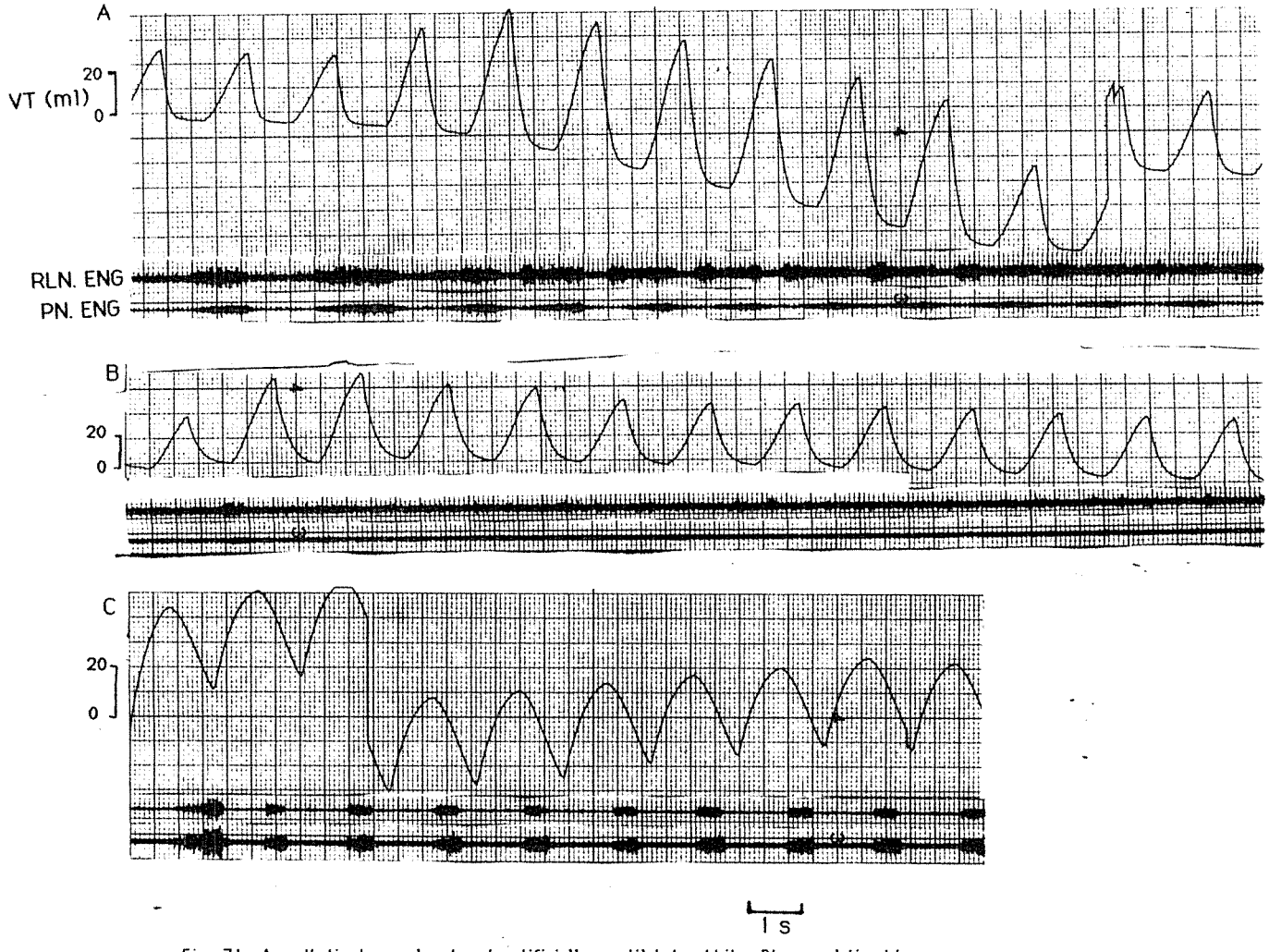
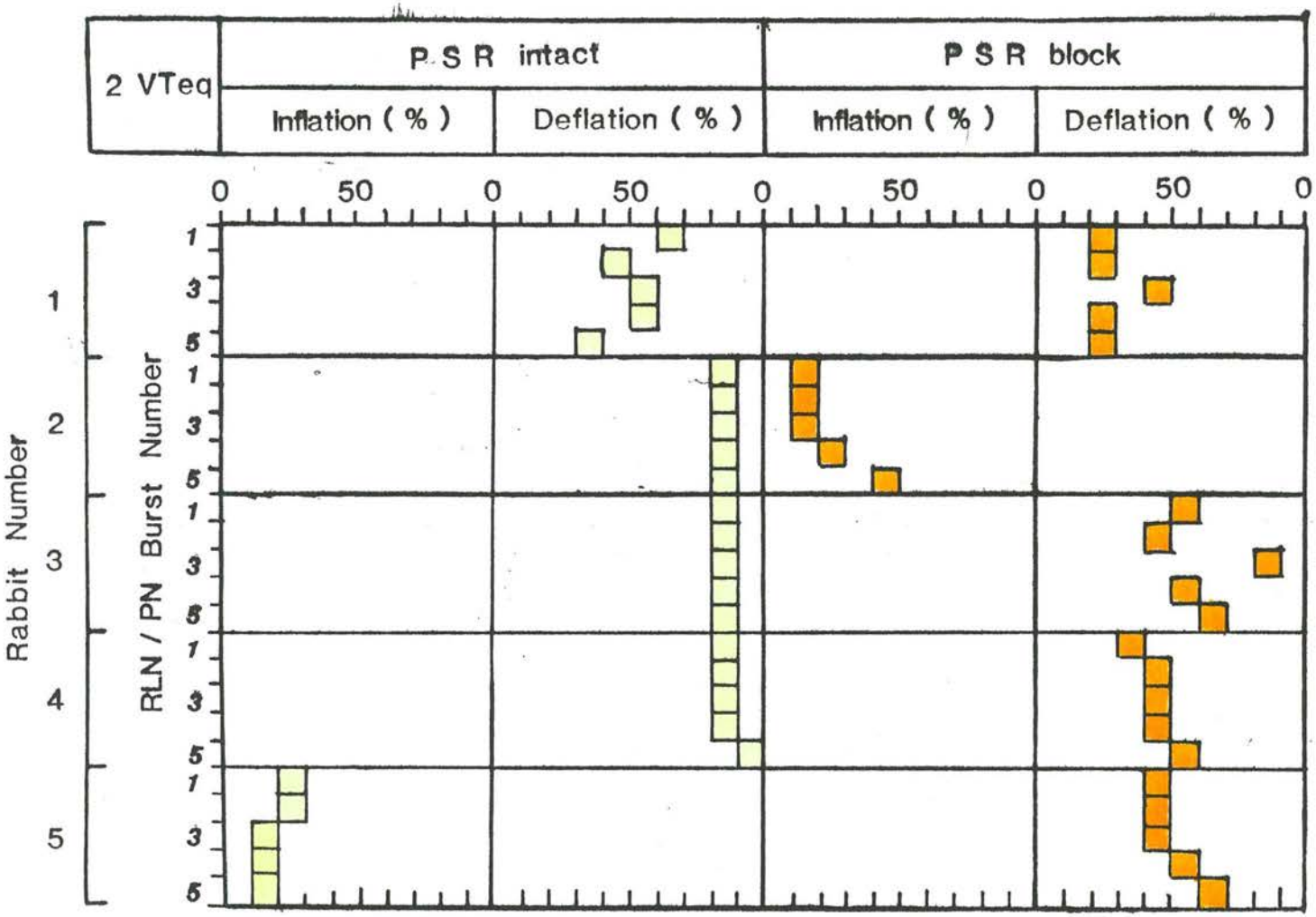


Fig. 71 Anaesthetized, paralysed and artificially ventilated rabbits. Phase relationship between RLN/PN discharge and pump cycle when tidal volume was 100% greater than eupnoeic level. A: onset of RLN and PN activity occurring during lung deflation (inflation upwards). B: depression and eventual disappearance of RLN and PN inspiratory bursts (inflation upwards). C: onset of RLN and PN activity during lung inflation (inflation downwards). In each record, upper trace: tidal volume (V_T), with some integrator drift and resetting; middle trace: recurrent laryngeal electroneurogram (RLN.ENG); lower trace: phrenic electroneurogram (PN.ENG). All three records were taken from different

Fig. 72 The time of onset of the recurrent laryngeal (RLN) and phrenic (PN) bursts in relation to the inflation and deflation phases of the ventilator when the tidal volume was 100% greater than eupnoeic level ($2V_{Teq}$). Each of the two phases was divided into ten equal intervals and the onsets of RLN and PN bursts were identified in them. This analysis was applied to each of the five experiments in the controls and in total stretch receptor block. Five successive RLN and PN bursts were included for each of the two conditions in each of the five rabbits.


2 VTeq
P S R intact
P S R block
Inflation (%)
Deflation (%)
Inflation (%)
Deflation (%)
0
50
Rabbit Number
RLN / PN Burst Number
1
2
3
4
5
1
3
5

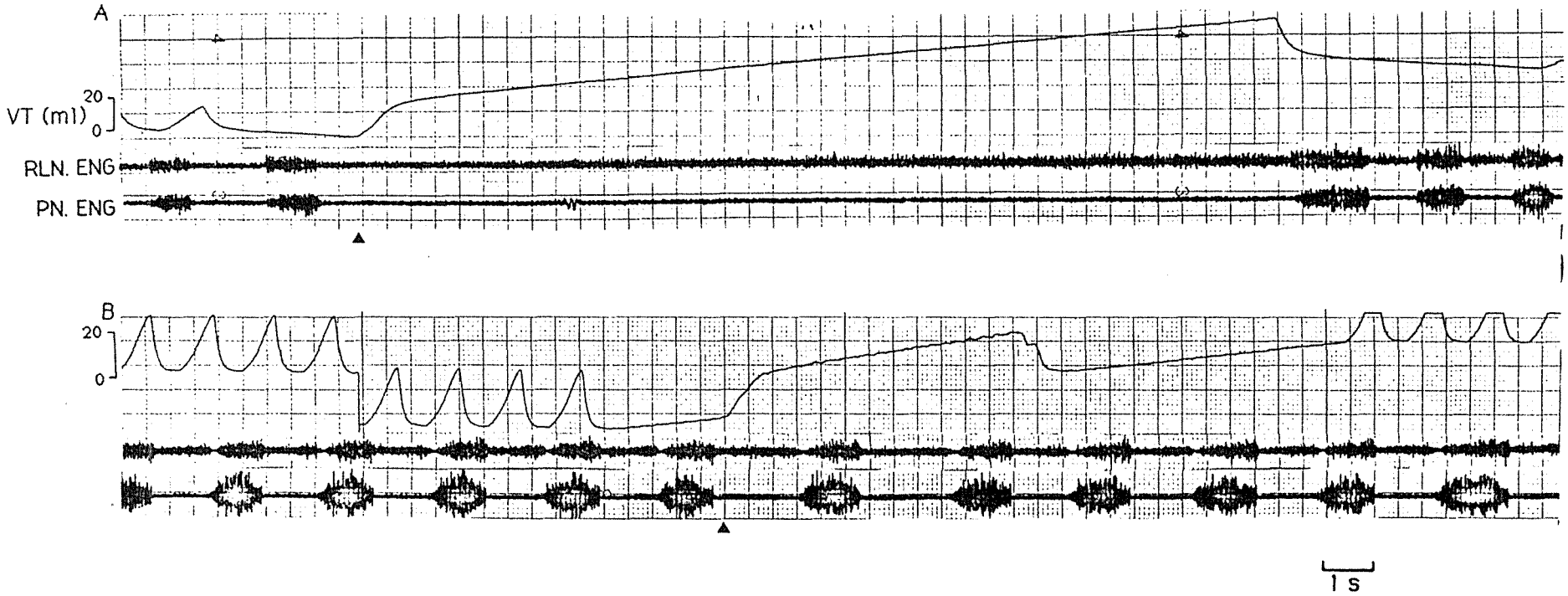


Fig. 73. Anaesthetized rabbits, paralysed and artificially ventilated. Effect of the Hering-Breuer inflation test with 10 cm H_2O positive pressure on the recurrent laryngeal and phrenic activity before and during stretch receptor block (records A and B respectively). In each record, upper trace: tidal volume (V_T); inflation upwards; middle trace: recurrent laryngeal electroneurogram (RLN.ENG); lower trace: phrenic electroneurogram (PN.ENG). The shift in baseline in the tidal volume trace was due to resetting the airflow integrator.

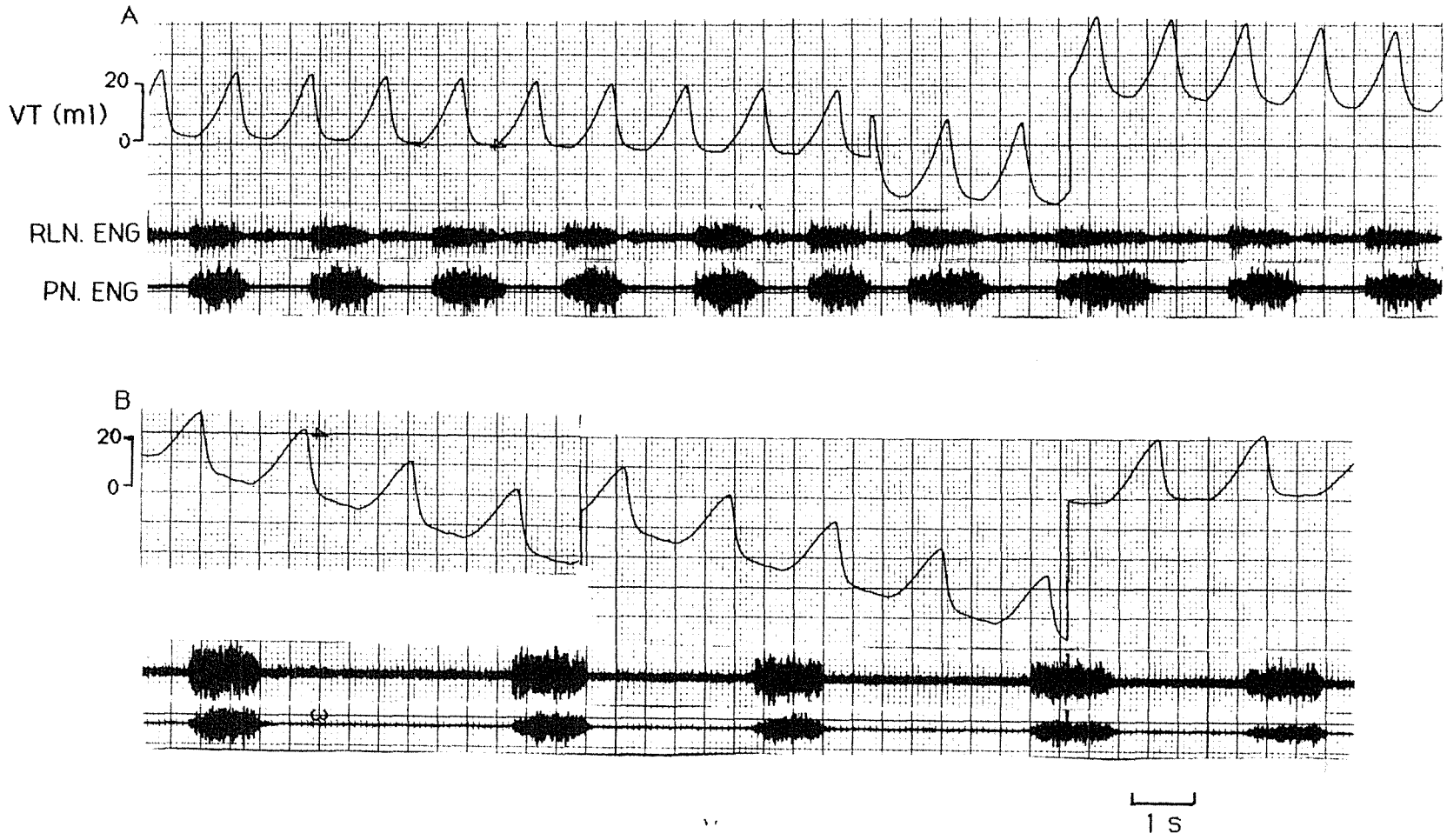


Fig. 74 Anaesthetized rabbits, paralysed and artificially ventilated. Effect of pulmonary stretch receptor block on the discharge patterns of recurrent laryngeal and phrenic nerves in relation to the inflation and deflation phases of the ventilator. A: depicts the change in pump to neural respiratory cycle ratio from 2:1 to 1:1 and back to 2:1 again. The administration of sulphur dioxide recruited previously silent expiratory fibres in the RLN. B: depicts the change in pump to neural respiratory cycle ratio from 3:1 to 2:1 and back to 3:1 again. In each record, upper trace: tidal volume (V_T); inflation upwards (with some integrator drift and resetting); middle trace: recurrent laryngeal electroneurogram (RLN.ENG); lower trace: phrenic electroneurogram (PN.ENG).

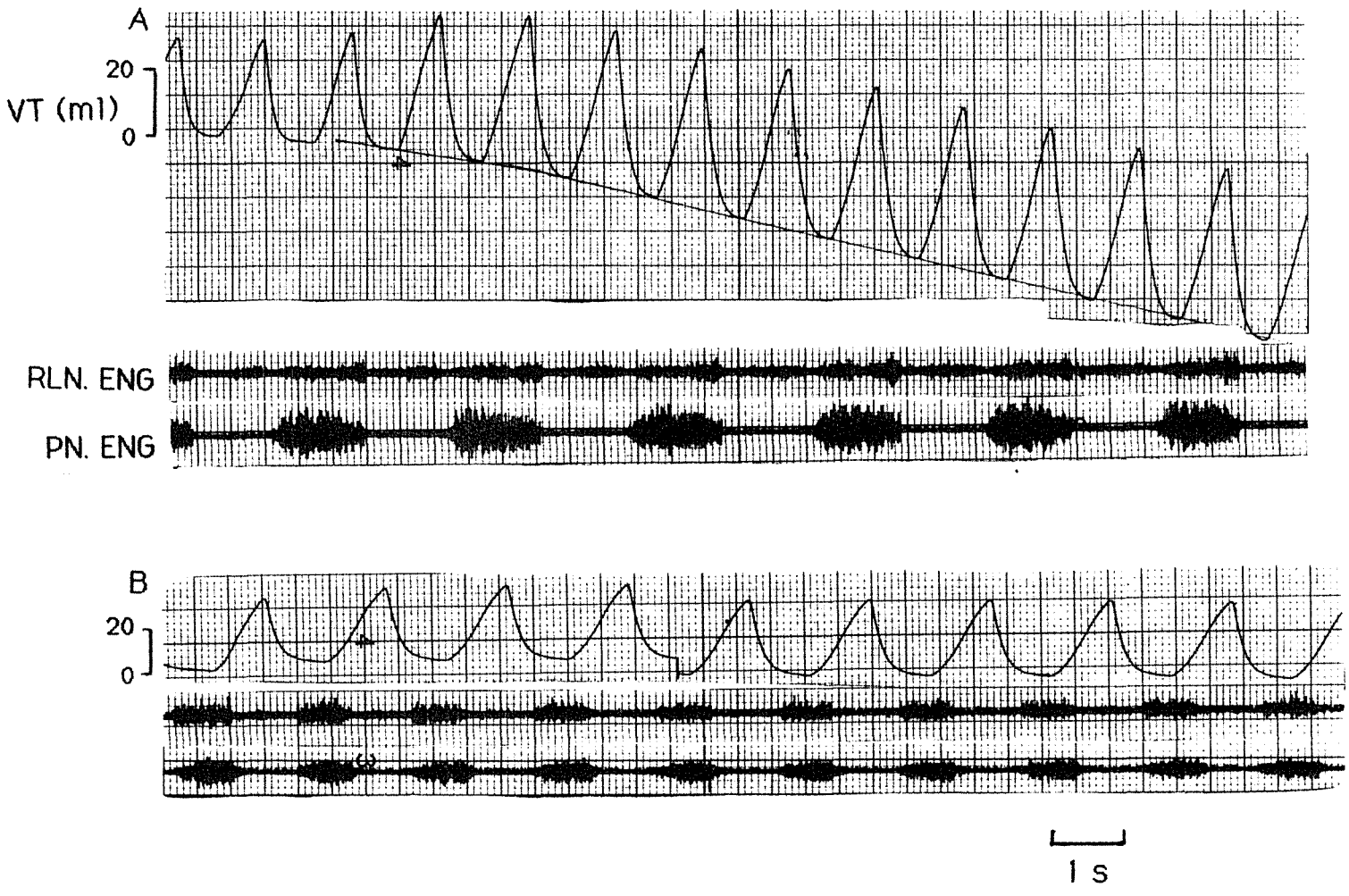


Fig. 75 Anaesthetized, paralysed and artificially ventilated rabbits. Two typical records of the changes in the activity of recurrent laryngeal and phrenic nerves when stretch receptors were blocked and the tidal volume (V_T) was 100% greater than eupnoeic level. In A, a distinct expiratory discharge was also elicited. In each record, upper trace: tidal volume, inflation upwards (with some integrator drift); middle trace: recurrent laryngeal electroneurogram (RLN.ENG); lower trace: phrenic electroneurogram (PN.ENG).

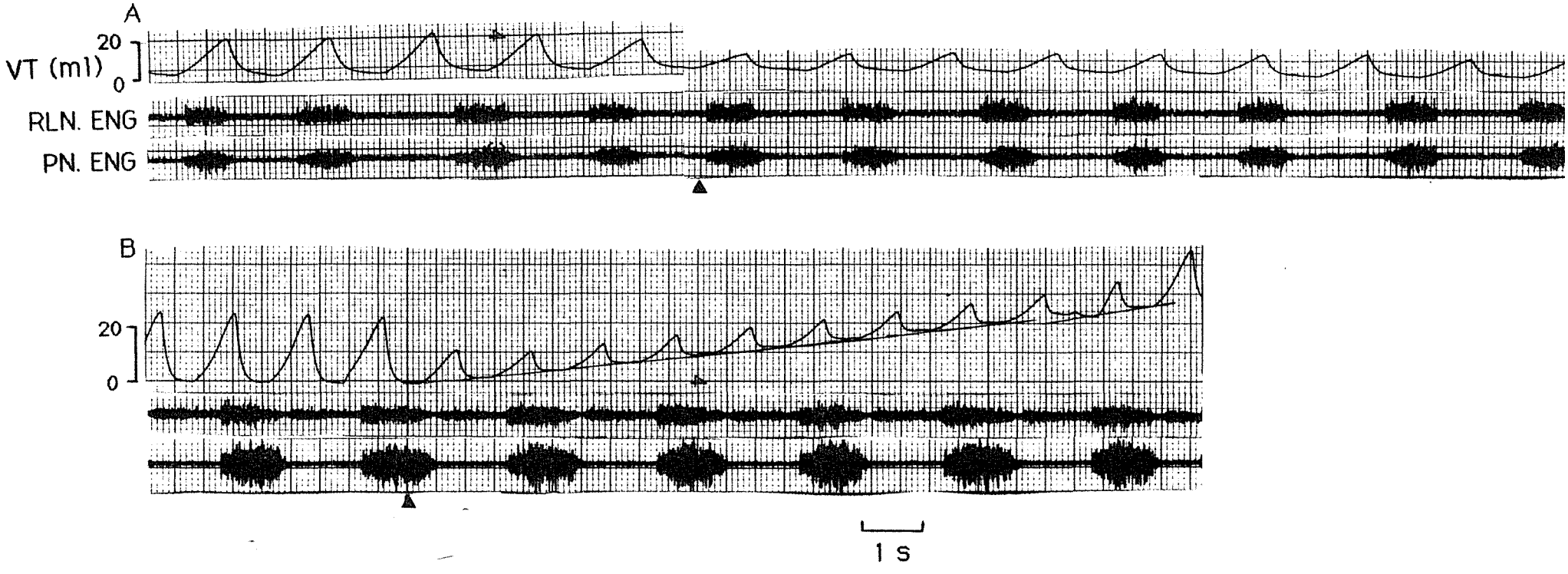
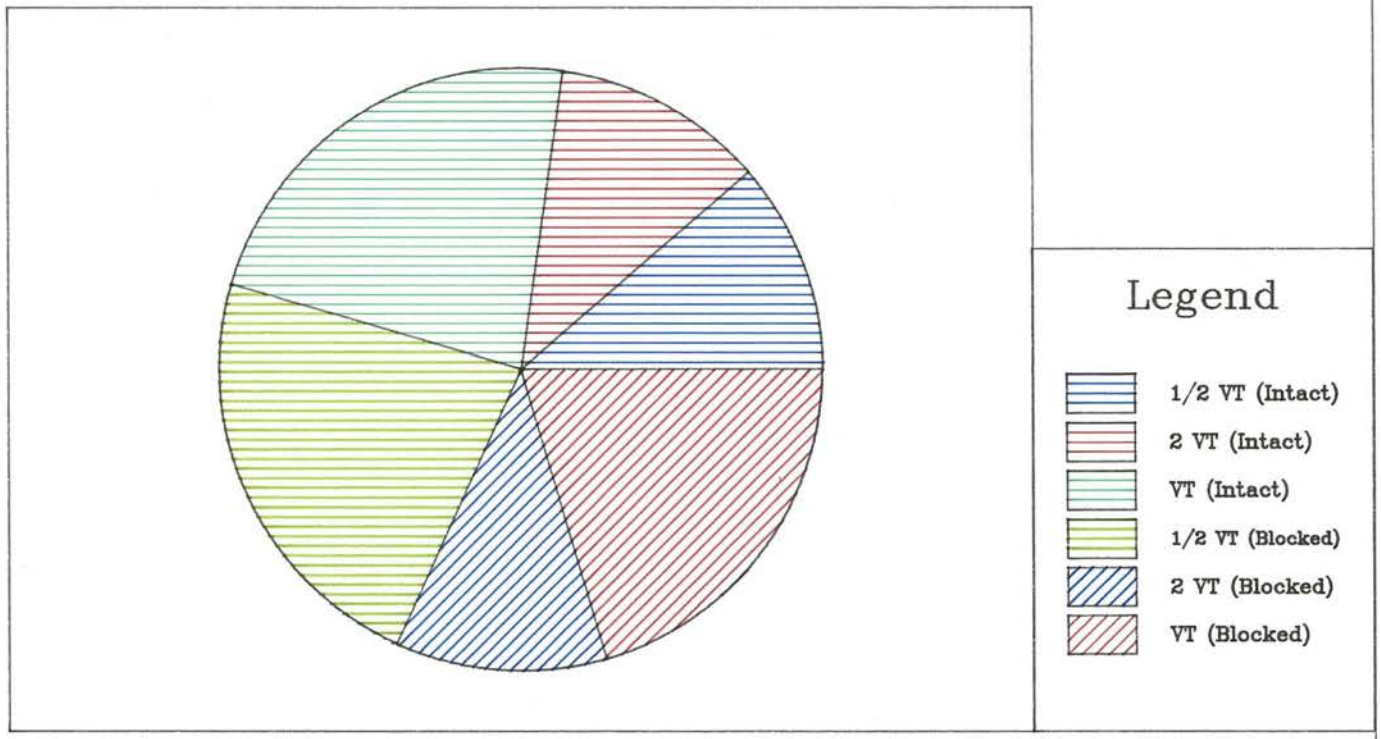


Fig. 76 Anaesthetized rabbits, paralysed and artificially ventilated. Effect of decreasing tidal volume (indicated by arrow) to half the spontaneous eupnoeic value on the phase relationship between recurrent laryngeal (RLN) and phrenic nerves (PN) and pump cycle during stretch receptor block. A: RLN and PN activty occurred with no phase relationship to pump cycle. B: RLN and PN bursts occurred during the inflation phase of the pump cycle. In each record, upper trace: tidal volume (V_T), inflation upwards (with some integrator drift); middle trace: recurrent laryngeal electroneurogram (RLN.ENG); lower trace: phrenic electroneurogram (PN.ENG).

FIG. 77 ONSET OF RLN AND PN BURSTS DURING PUMP INFLATION STROKE AT DIFFERENT VENTILATING TIDAL VOLUMES



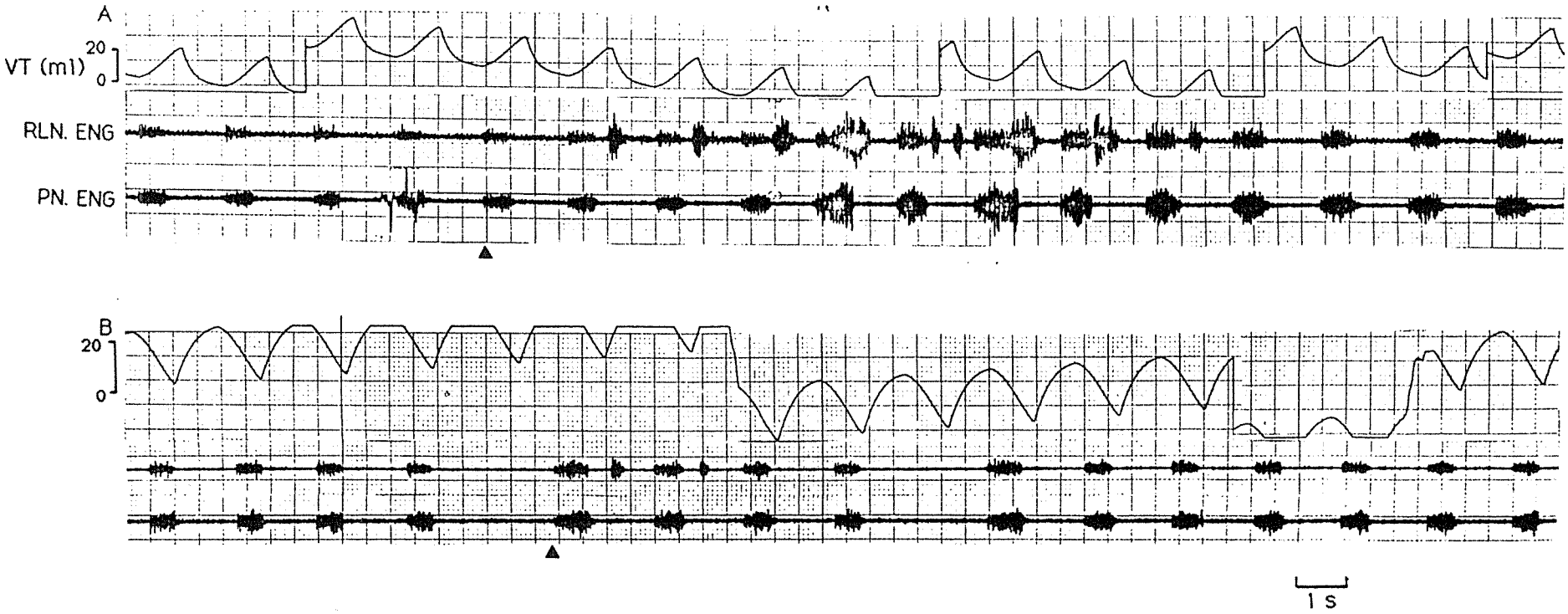


Fig. 78 Records (A and B) showing the discharge patterns of recurrent laryngeal and phrenic nerves during cough following sulphur dioxide administration (indicated by arrow). In each record, upper trace: tidal volume (V_T), inflation upwards in A, inflation downwards in B (with some integrator drift and resetting); middle trace: recurrent laryngeal electroneurogram (RLN.ENG); lower trace: phrenic electroneurogram (PN.ENG).

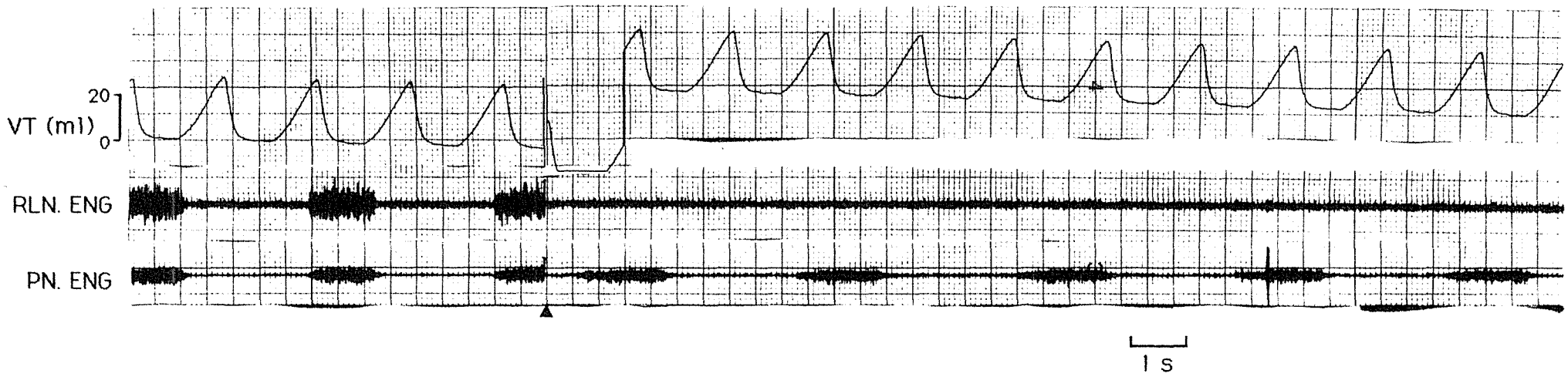
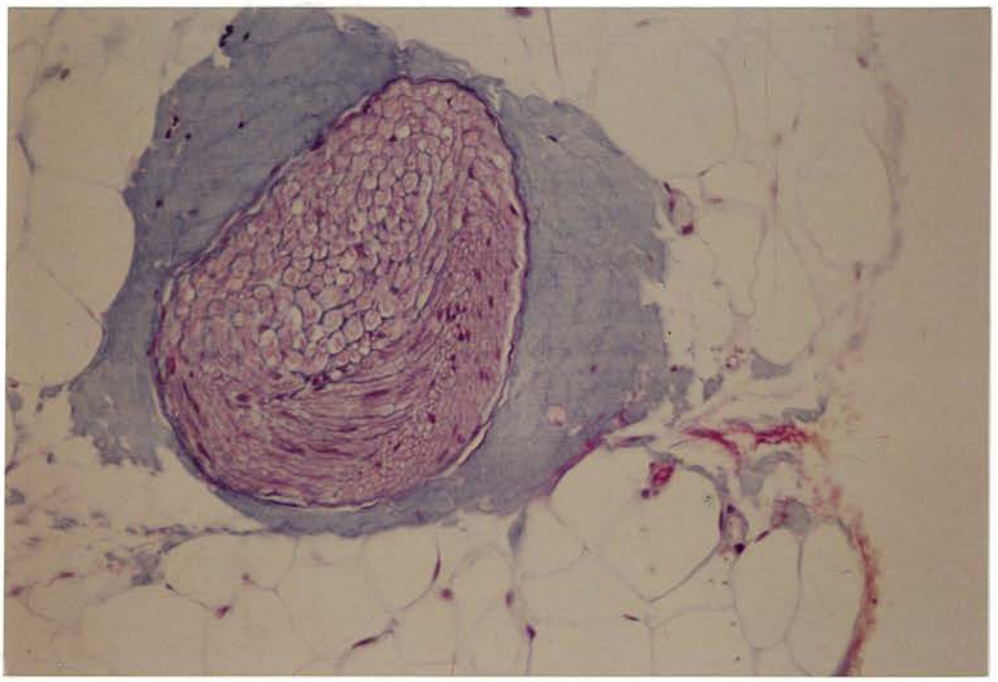


Fig. 79 Record showing the disappearance of recurrent laryngeal nerve activity consequent on bilateral cervical vagotomy (indicated by arrow). Upper trace: tidal volume (V_T), inflation upwards (with some integrator drift and resetting); middle trace: recurrent laryngeal electroneurogram (RLN.ENG); lower trace: phrenic electroneurogram (PN.ENG).

Plate 1 *A transverse section of the right recurrent laryngeal nerve at the level of the clavicle. The sections were stained with Masson's blue trichrome.*

a. *Showing the distribution of large and small diameter, myelinated fibres in the fascicle (x250).*

b. *A segment of the fascicle at a higher magnification showing the large and small diameter, myelinated fibres occupying relatively discrete areas of the nerve (x620).*



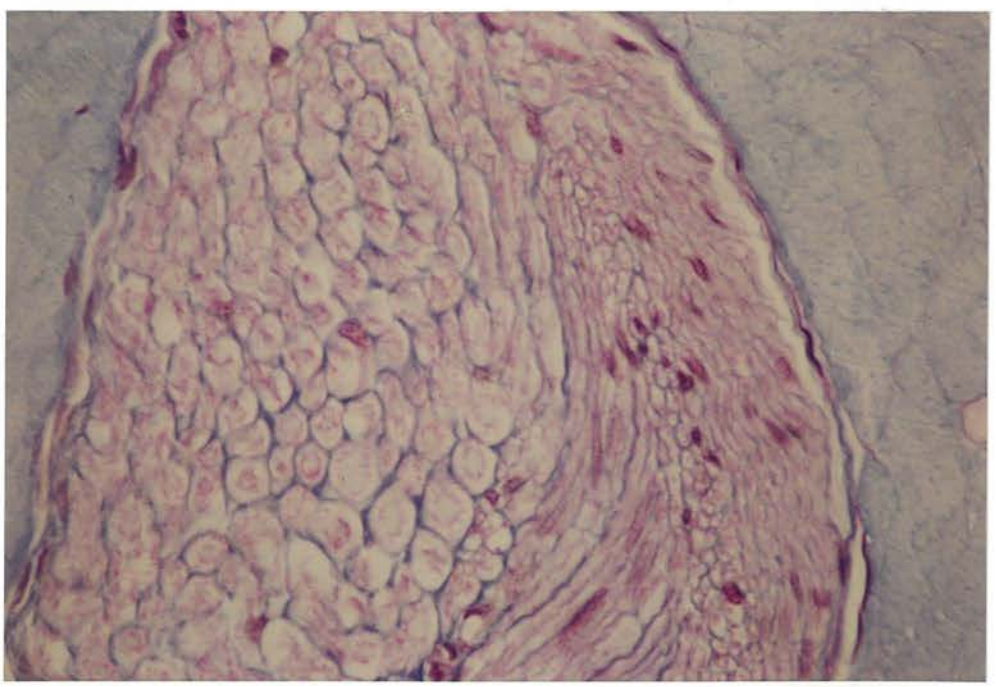
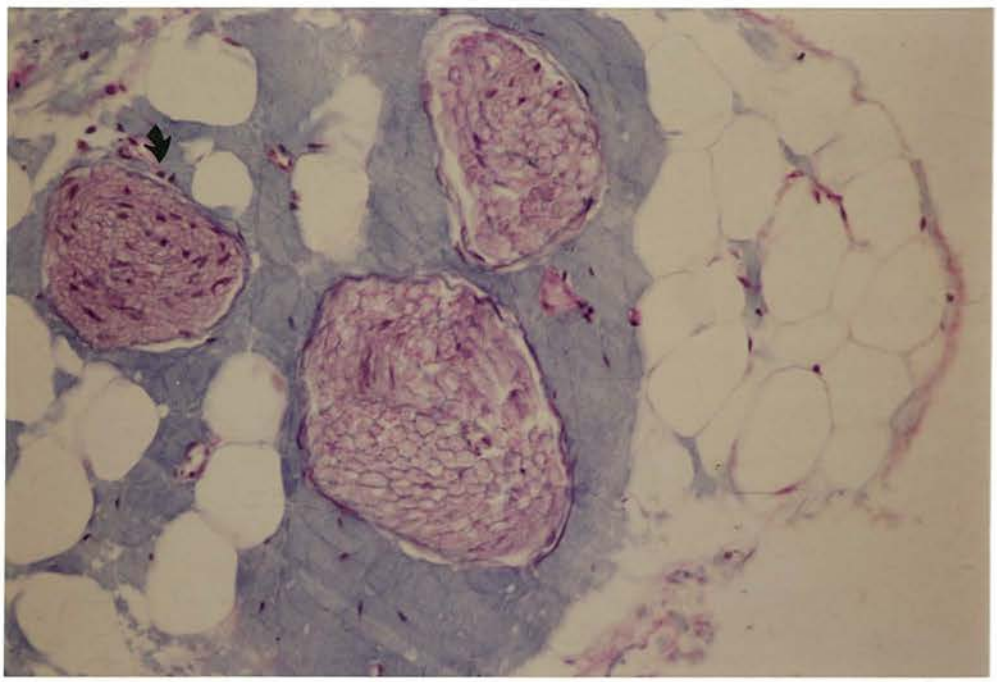


Plate 2 A transverse section of the left recurrent laryngeal nerve at the level of the clavicle. The sections were stained with Masson's blue trichrome.

a. Showing fascicular characteristics of the nerve at this level (x250). Compare with photomicrograph of right recurrent laryngeal nerve (Plate 1a).

b. One of the three fascicles (indicated by an arrow in (a)) at a higher magnification, showing the distribution of large and small diameter, myelinated fibres (x620).



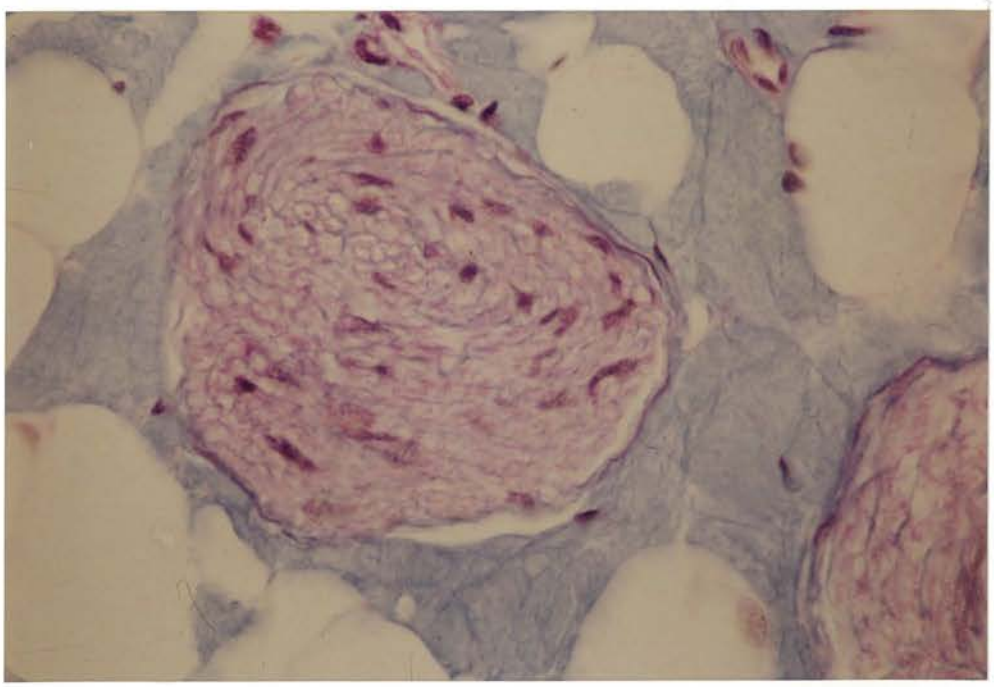
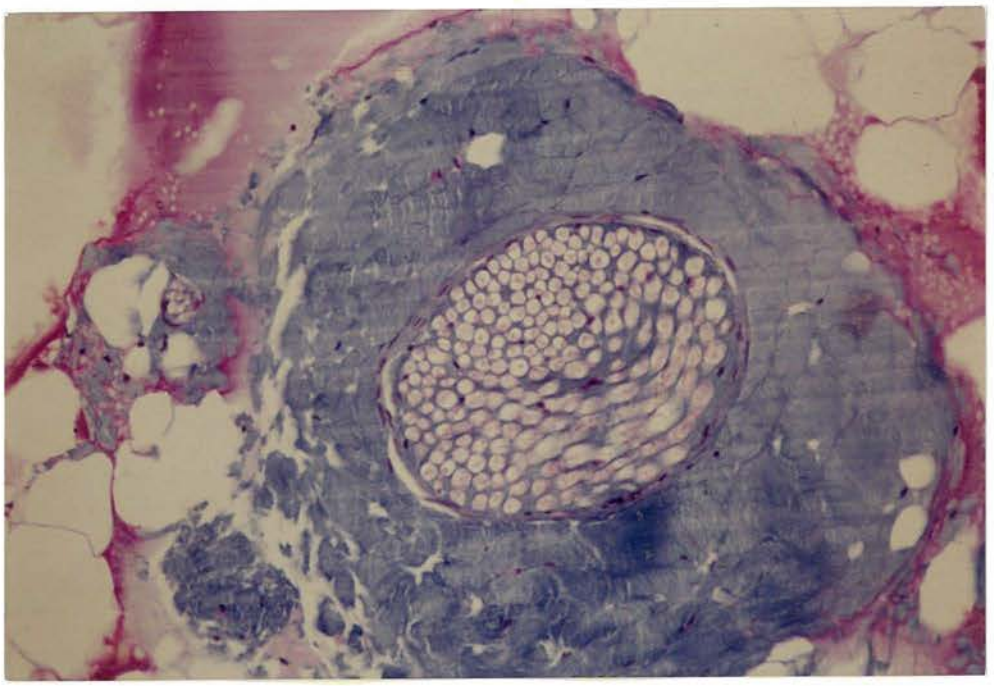


Plate 3 A transverse section of the right recurrent laryngeal nerve near its entry into the larynx. The sections were stained with Masson's blue trichrome.

a. Showing the large and the small diameter, myelinated fibres occupying sharply defined areas (x250).

b. Showing the fascicle comprising predominantly large diameter, myelinated fibres (x620).



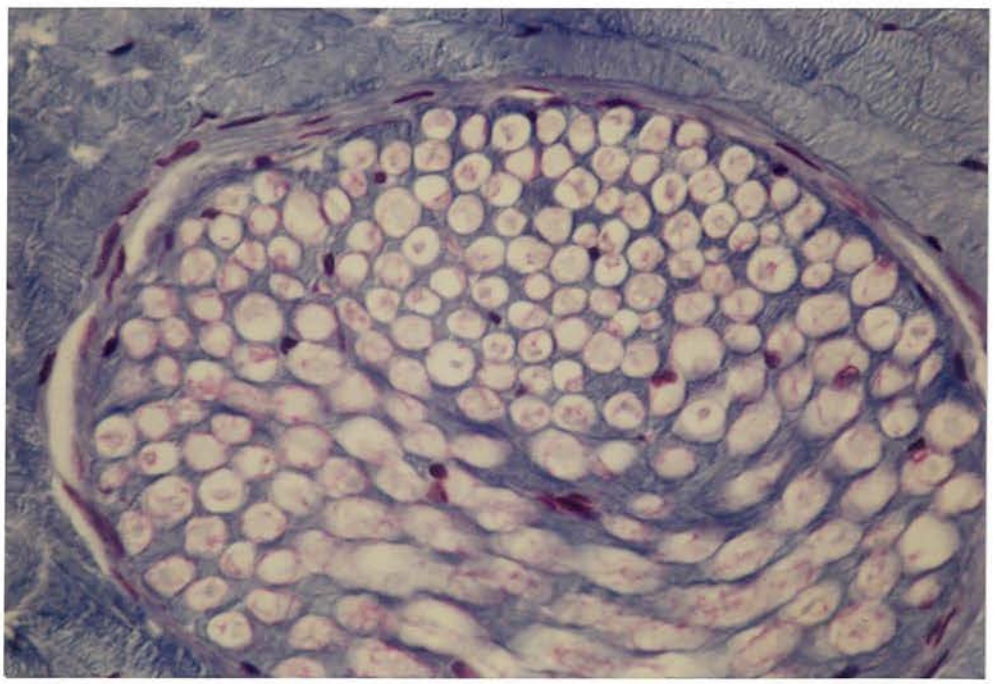
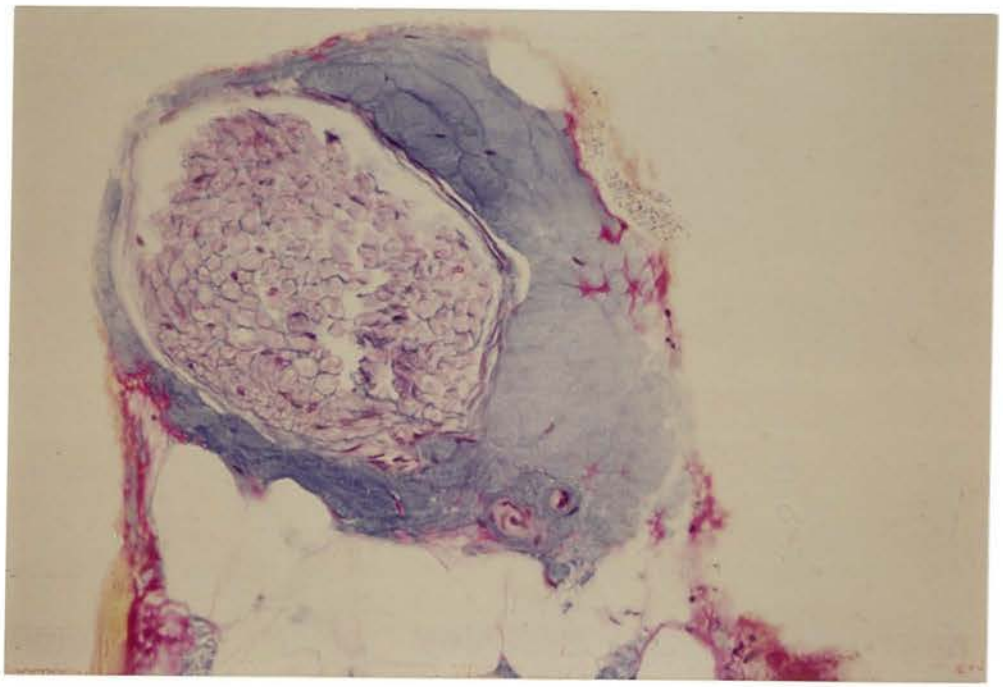
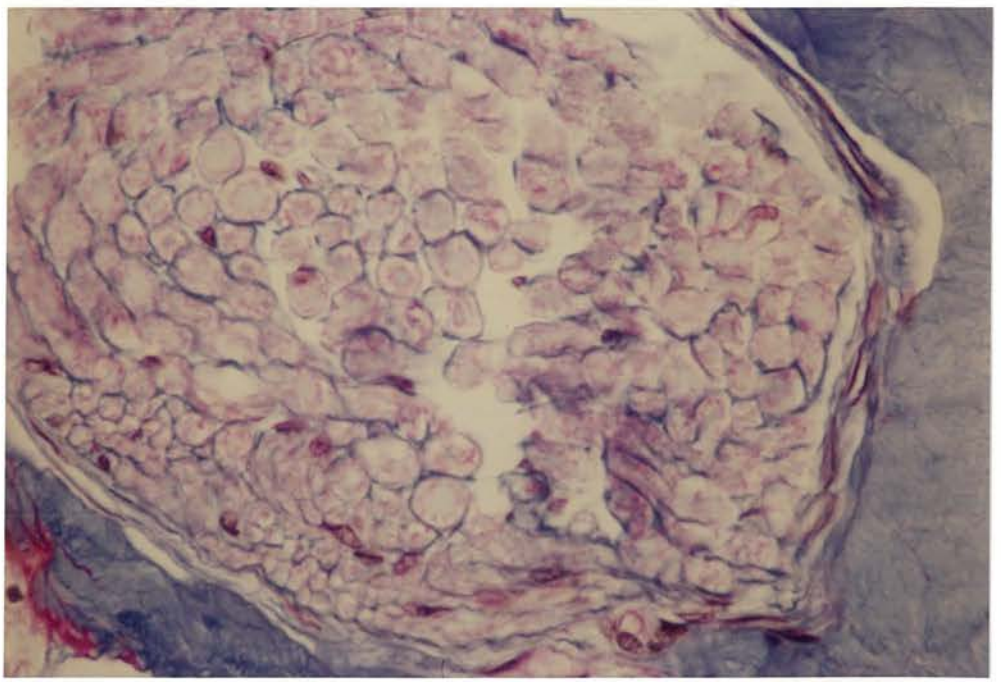


Plate 4 A transverse section of the left recurrent laryngeal nerve near its entry into the larynx. The sections were stained with Masson's blue trichrome.

a. Showing the distribution of the large and small diameter, myelinated fibres in the fascicle (x250).

b. Showing a higher magnification of the fascicle which has a majority of the large diameter, myelinated nerve fibres (x620).





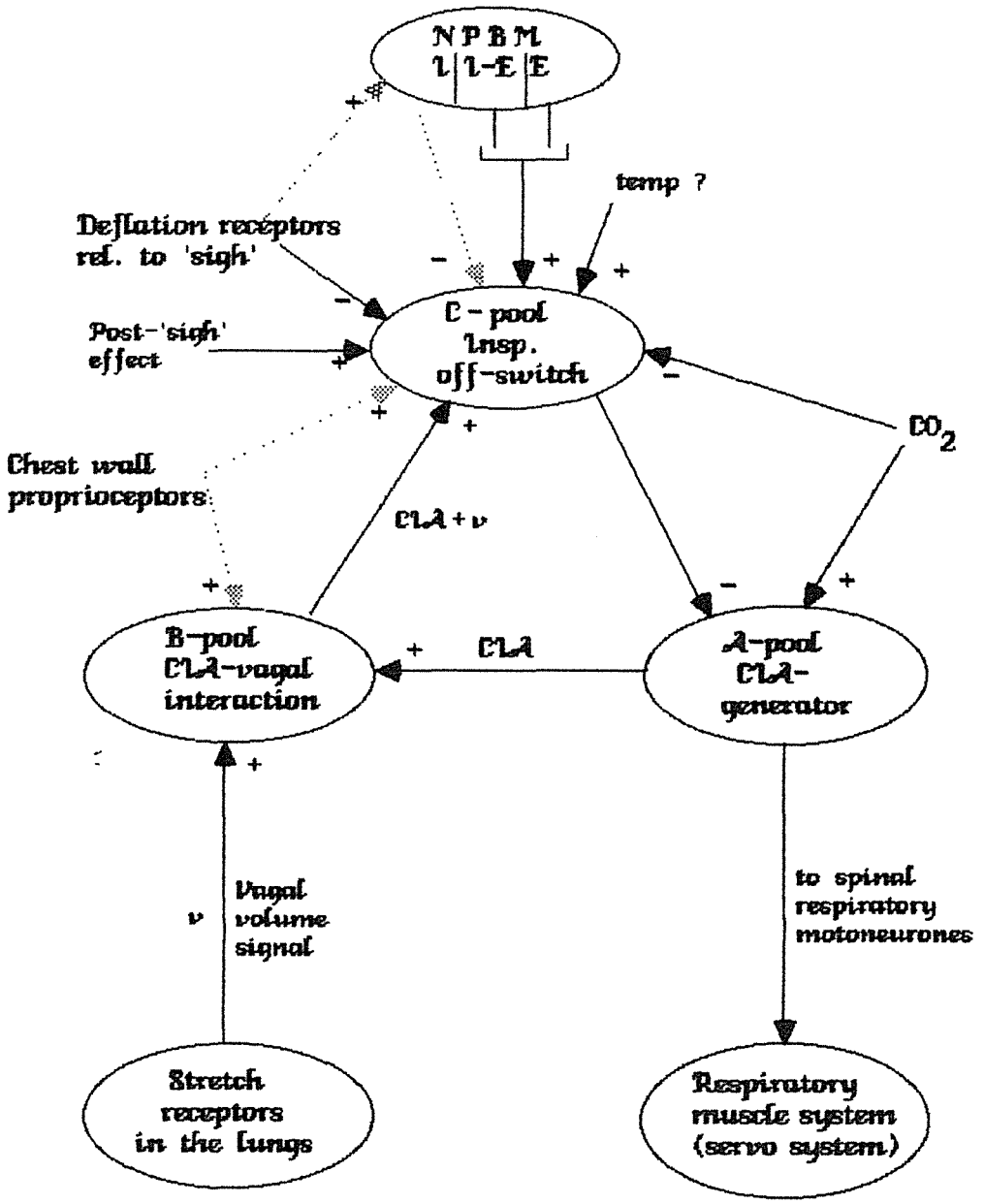


Fig. 80 Schematic representation of the functional organization of the basic inspiratory 'off-switch' mechanism. NPBM = medial parabrachial nucleus (pneumotaxic centre). + signs denote excitatory connections and − signs denote inhibitory connections. Hatched lines denote some alternative possibilities for connections. The 'pools' should be regarded as representing functions rather than the neural correlates to these functions. After von Euler and Trippenbach (1975).

Fig. 81 Mean percentage changes in frequency and duration of phasic-inspiratory laryngeal motoneurone discharge during the inspiratory phase caused by various interventions. Significance of change compared with zero effect : * $P < 0.05$, *** $P < 0.001$.

 Frequency of discharge

 Duration of discharge

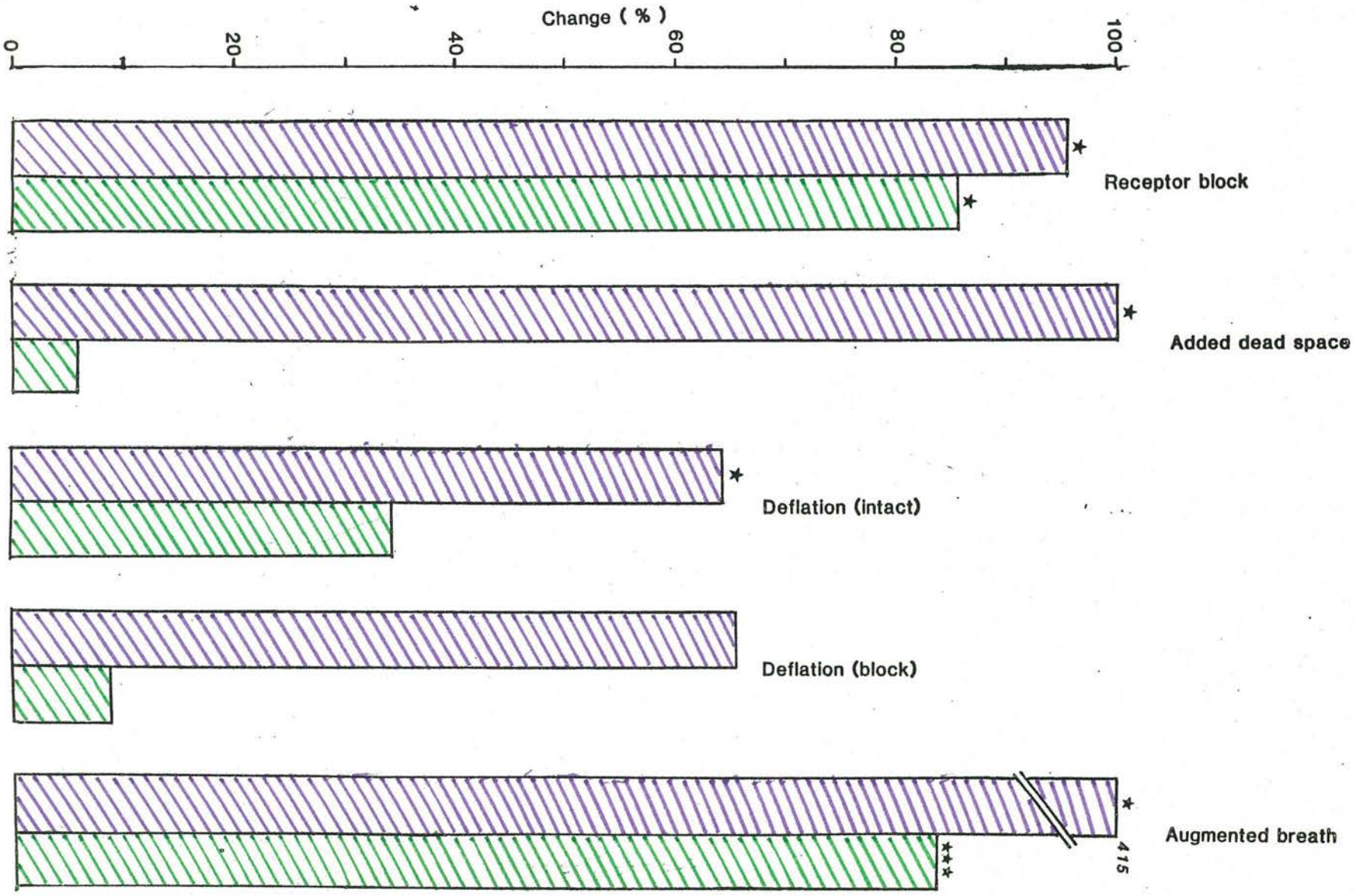

Change (%)
0
1
20
40
60
80
100
Receptor block
Added dead space
Deflation (intact)
Deflation (block)
Augmented breath
415

Fig. 82 Mean percentage changes in frequency and duration of phasic-inspiratory laryngeal motoneurone discharge during the expiratory phase caused by various interventions.


Frequency of discharge


Duration of discharge

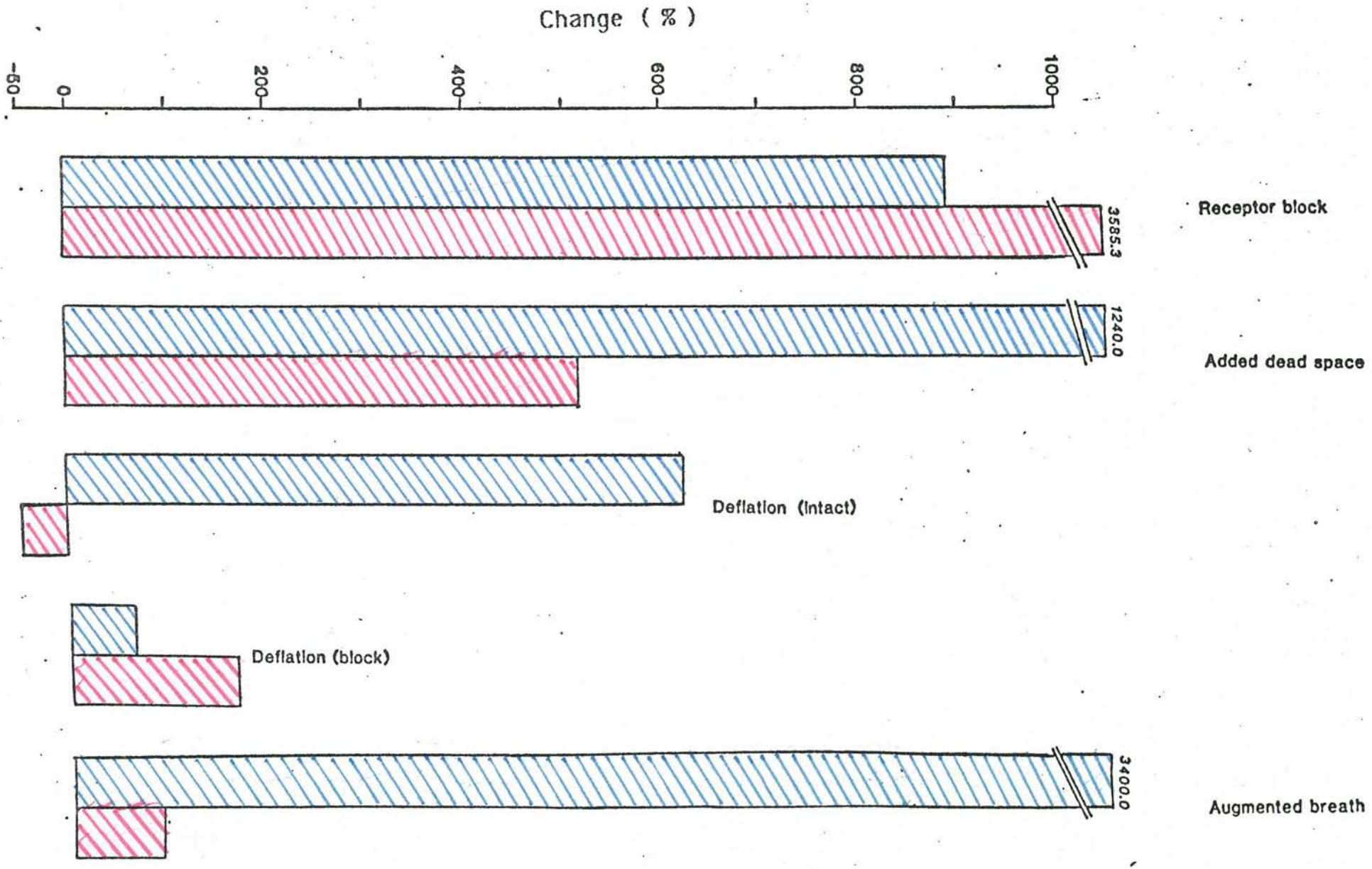

Change (%)
-50
0
200
400
600
800
1000
3585.3
Receptor block
1240.0
Added dead space
Deflation (intact)
Deflation (block)
3400.0
Augmented breath

Fig. 83 Mean percentage changes in frequency and duration of tonic-expiratory laryngeal motoneurone discharge during the inspiratory phase caused by various interventions (N = 2 fibres). Significance of change compared with zero effect : * $P < 0.05$. Significance of change in blocked condition compared with that in intact state : # $P < 0.05$.

Frequency of discharge

Duration of discharge

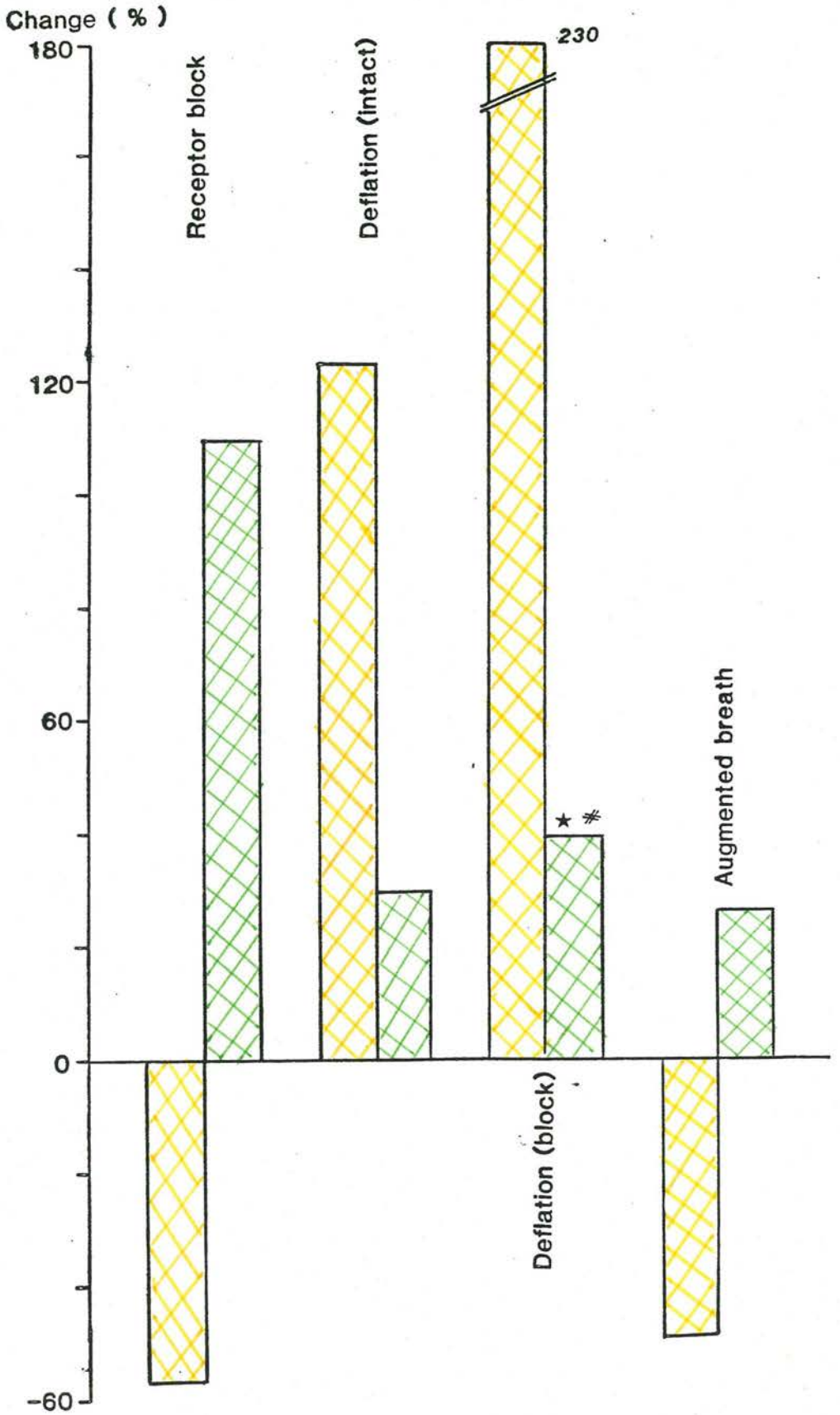

Change (%)
180
120
60
0
-60
Receptor block
Deflation (intact)
230
Deflation (block)
Augmented breath

Fig. 84 Mean percentage changes in frequency and duration of tonic-expiratory laryngeal motoneurone discharge during the expiratory phase caused by various interventions (N = 2 fibres). Significance of change compared with zero effect : * $P < 0.05$. Significance of change in blocked condition compared with that in intact state : # $P < 0.05$.

Frequency of discharge

Duration of discharge

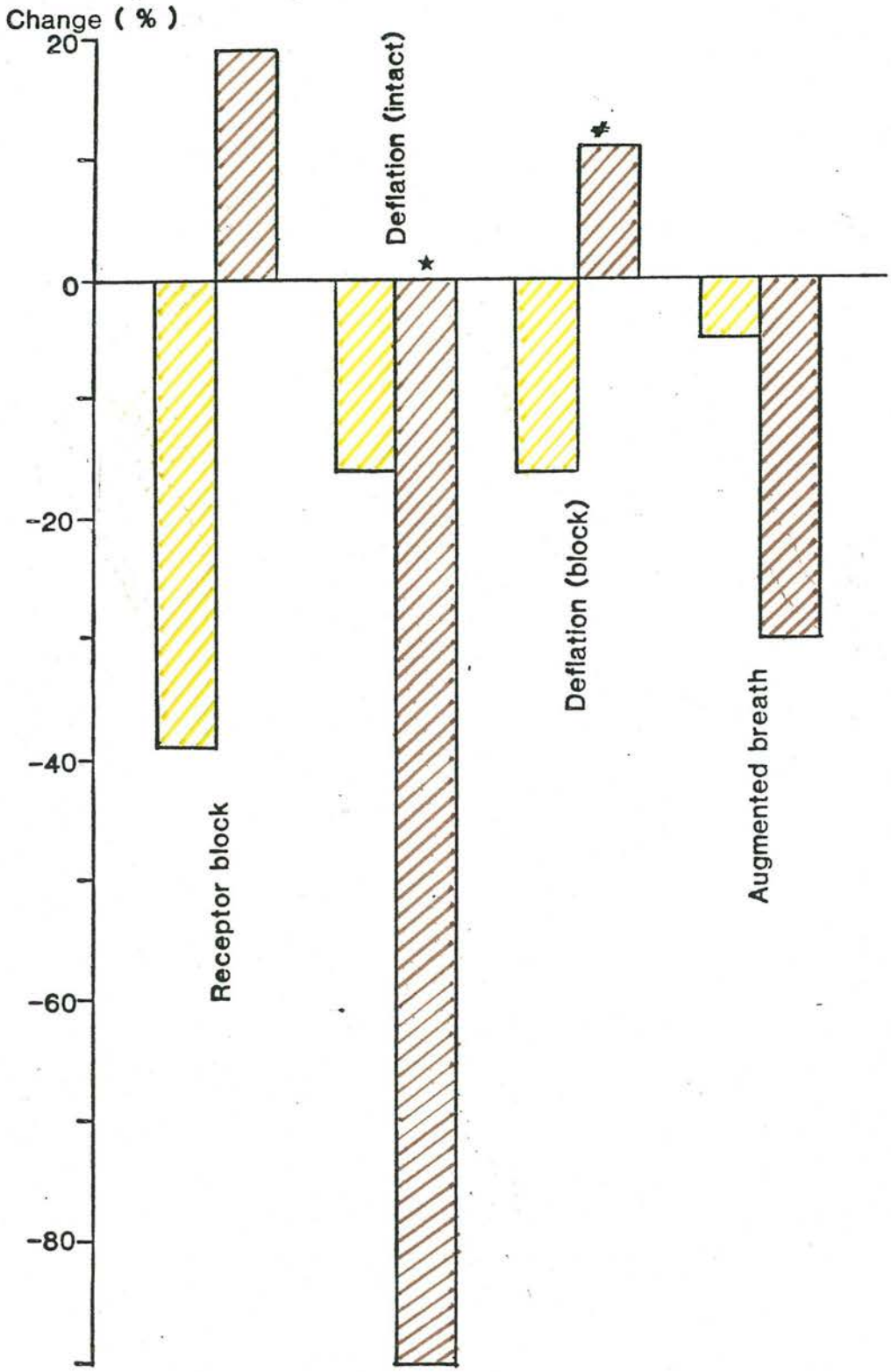
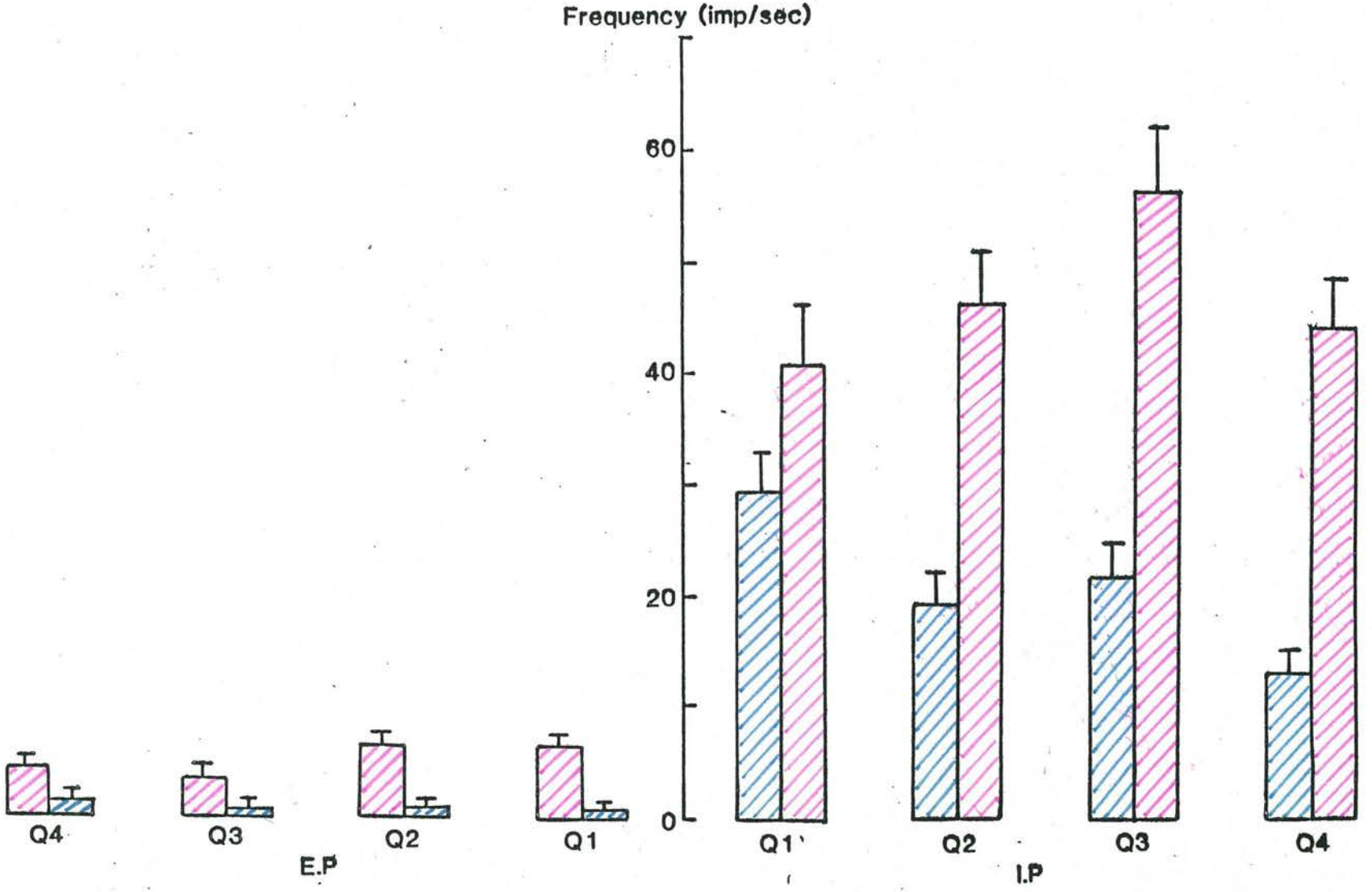

Change (%)
20
0
-20
-40
-60
-80
Receptor block
Deflation (intact)
Deflation (block)
Augmented breath

Fig. 85 Effect of pulmonary stretch receptor block by 20 minutes exposure to 200 ppm sulphur dioxide on the mean frequency of phasic-inspiratory laryngeal motoneurone discharge in different quartiles of the inspiratory (I.P) and expiratory (E.P) phases of the respiratory cycle. Vertical bars represent standard errors.

Stretch receptors intact

Stretch receptors blocked


Frequency (imp/sec)
60
40
20
0
Q4
Q3
Q2
Q1
E.P
Q1
Q2
Q3
Q4
I.P

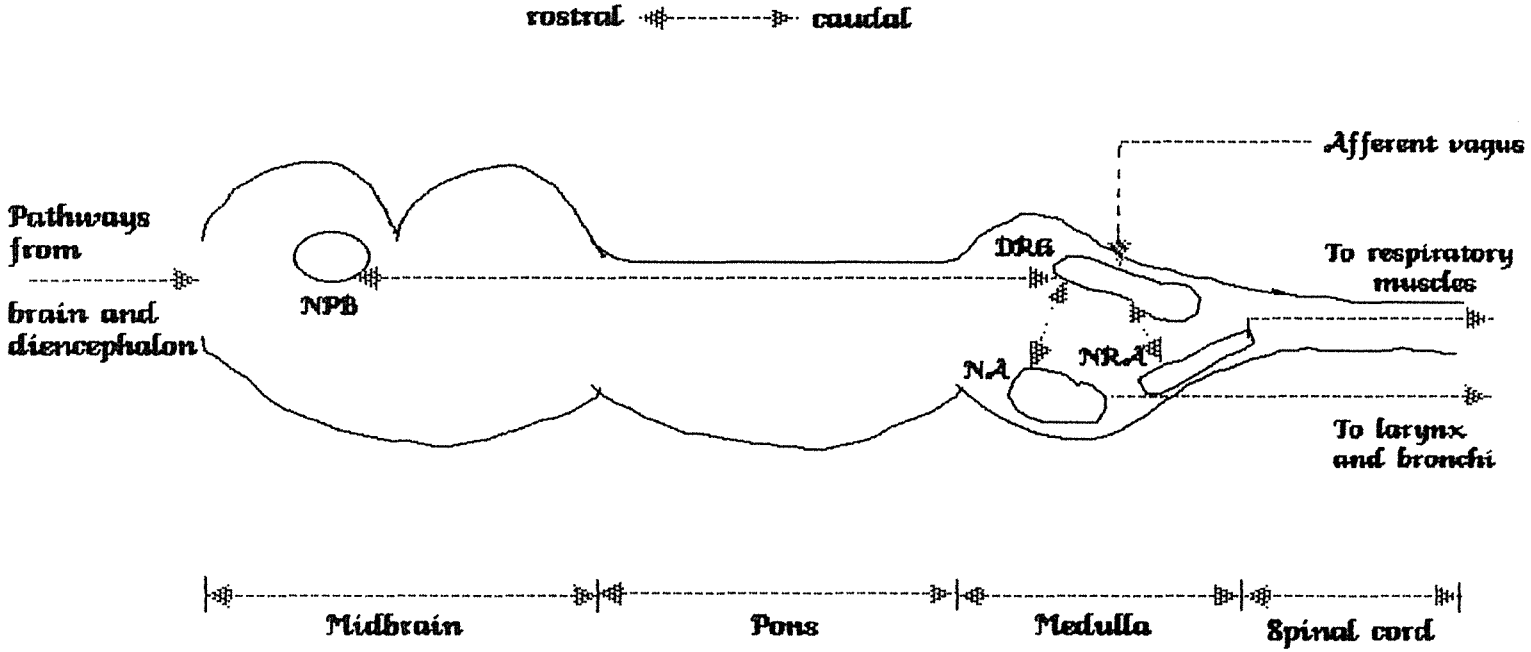


Fig. 86 Schematic diagram of brainstem structures involved in breathing. NPB = nucleus parabrachialis (= pneumotaxic centre). Respiratory neurones in medulla (after Mitchell and Berger, 1975): DRG = dorsal respiratory group, near nucleus tractus solitarius; VRG = ventral respiratory group, with neurones in nucleus ambiguus (NA) and in nucleus retroambigualis (NRA).

Appendix

Data obtained from recordings of 24 recurrent laryngeal motoneurones from 9 rabbits were tabulated as follows:

Rabbit Number: _____			Fibre Number: _____					PSR: Intact/Block		
Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1 Q2 Q3 Q4 Tot.				Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq. Range (Hz)

Abbreviations:

PSR:	Pulmonary Stretch Receptor
Br. No:	Breath Number
Ph:	Respiratory Phase; Inspiration (I) / Expiration (E)
Ph. Dr:	Inspiratory (tI) / Expiratory Duration (tE)
Q1:	First Quartile (0-25% of tI/tE)
Q2:	Second Quartile (25-50% of tI/tE)
Q3:	Third Quartile (50-75% of tI/tE)
Q4:	Fourth Quartile (75-100% of tI/tE)
Tot:	Total Number of Spikes
Bg. F:	Onset of Firing
End. F:	End of Firing
Dr. F:	Duration of Firing
Fmean:	Mean Frequency
Freq. Range:	Frequency Range

Rabbit Number: 1 Fibre Number: 1 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.range (Hz)
Pre-Infln	1	I	0.68	3	3	2	0	8	0.03	0.50	0.47	11.8	7.7- 50.0
		E	1.80	4	3	1	5	13	0.06	1.78	1.72	7.2	2.4-200.0
	2	I	0.67	4	3	3	1	11	0.04	0.56	0.52	16.4	14.3-100.0
		E	1.71	4	2	1	2	9	0.11	1.65	1.54	5.3	2.0- 40.0
	3	I	0.69	3	4	1	1	9	0.04	0.57	0.53	13.0	7.7- 66.7
		E	1.71	5	1	2	3	11	0.08	1.52	1.44	6.4	2.1- 50.0
Inflation	(Position: 83.3% tE; Duration of apnoea: 5.65 sec.)												
	1st 0.1 sec:							9					
	2nd 0.1 sec:							9					
	1st 1.0 sec:							94					
	Mid 1.0 sec:							96					
	End 1.0 sec:							95					
Post-Infln	1st 0.1 sec:							1					
	2nd 0.1 sec:							1					
	1	I	1.10	4	2	1	3	10	0.05	1.08	1.03	9.1	3.7- 50.0
		E	0.87	1	1	2	1	5	0.18	0.81	0.63	5.7	5.0- 11.1
	2	I	0.82	5	3	2	3	13	0	0.78	0.78	15.9	5.6-100.0
		E	0.88	1	1	1	1	4	0.18	0.85	0.67	4.5	
	3	I	0.82	2	3	5	0	10	0.05	0.59	0.54	12.2	5.6-200.0
		E	1.08	0	3	2	2	7	0.32	1.04	0.72	6.5	5.9- 14.3
Pre-Defln	1	I	0.66	6	0	2	1	9	0	0.58	0.58	13.6	4.3-100.0
		E	1.74	4	1	1	5	11	0.07	1.65	1.58	6.3	2.1- 40.0
	2	I	0.66	4	4	2	1	11	0.01	0.54	0.53	16.7	7.7-200.0
		E	1.73	2	3	4	6	15	0.32	1.72	1.40	8.7	4.2-200.0
	3	I	0.67	3	4	1	1	9	0.02	0.53	0.51	13.4	5.6-200.0
		E	1.79	4	4	1	4	13	0.10	1.66	1.56	7.3	2.1-200.0
Deflation:	(Position: 12.6% tE)												
	1st 0.1 sec:							8					
	2nd 0.1 sec:							13					
	1st	I	0.89	23	29	25	24	101	0	0.88	0.88	113.5	50.0-200.0
		E	0.29	6	7	9	4	26	0	0.28	0.28	89.7	33.3-200.0
	Mid	I	0.59	14	15	14	12	55	0	0.58	0.58	93.2	33.3-200.0
		E	0.28	8	5	6	7	26	0	0.27	0.27	92.9	50.0-200.0
	End	I	0.57	18	18	14	13	63	0	0.56	0.56	110.5	50.0-200.0
		E	0.31	5	7	7	7	26	0	0.30	0.30	83.9	66.7-200.0
Post-Defln	1st 0.1 sec:							0					
	2nd 0.1 sec:							1					
	1	I	0.57	7	2	3	3	15	0.02	0.51	0.49	26.3	8.3-200.0
		E	1.69	4	3	2	5	14	0.06	1.68	1.62	8.3	3.4- 28.6

Rabbit Number: 1 Fibre Number: 2 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.range (Hz)
Pre-Infln	1	I	0.70	16	9	6	6	37	0	0.62	0.62	52.9	25.0-200.0
		E	1.45	7	10	11	12	40	0.09	1.44	1.35	27.6	11.1-200.0
	2	I	0.69	15	8	8	5	36	0.01	0.66	0.65	52.2	16.7-200.0
		E	1.57	8	11	13	16	48	0	1.56	1.56	30.6	16.7-100.0
	3	I	0.67	15	9	9	5	38	0.01	0.58	0.57	56.7	28.6-200.0
		E	1.58	6	13	14	14	47	0.14	1.56	1.42	29.7	10.0-200.0
Inflation	(Position: 30.1% tE: Duration of apnoea: 10.29 sec.)												
	1st 0.1 sec:							5					
	2nd 0.1 sec:							4					
	1st 1.0 sec:							10					
	Mid 1.0 sec:							44					
	End 1.0 sec:							59					
Post-Infln	1st 0.1 sec:							5					
	2nd 0.1 sec:							6					
	1	I	1.01	28	11	12	9	60	0.01	1.00	0.99	59.4	28.6-200.0
		E	0.67	2	4	5	6	17	0.06	0.66	0.60	25.4	12.5-200.0
	2	I	0.79	11	5	5	2	23	0.01	0.63	0.62	29.1	20.0-200.0
		E	0.77	0	0	0	1	1	0	0.75	0.75		
	3	I	0.76	10	4	4	1	19	0.01	0.63	0.62	25.0	11.1-200.0
		E	0.85	0	0	0	1	1	0	0.84	0.84		

Pre-Defln	1	I	0.65	9	4	3	1	17	0.01	0.53	0.52	26.2	14.3-200.0
		E	1.43	2	4	6	10	22	0.08	1.42	1.34	15.4	4.8-200.0
	2	I	0.66	10	4	4	2	20	0.02	0.57	0.55	30.3	16.7-200.0
		E	1.49	3	7	6	13	29	0.01	1.48	1.47	19.5	2.8-200.0
	3	I	0.64	8	7	5	3	23	0	0.55	0.55	35.9	20.0-200.0
		E	1.55	1	8	10	11	30	0.31	1.54	1.23	19.4	11.1-100.0
Deflation	(Position: 4.6% tI)												
	1st 0.1 sec:							10					
	2nd 0.1 sec:							7					
	1st	I	0.90	16	14	12	8	50	0.02	0.87	0.85	55.6	28.6-200.0
		E	0.28	2	0	1	6	9	0.02	0.27	0.25	32.1	6.7-200.0
	Mid	I	0.40	5	4	4	2	15	0.01	0.37	0.36	37.5	16.7-100.0
		E	0.34	0	0	3	3	6	0.18	0.33	0.15	17.6	25.0-200.0
	End	I	0.51	5	6	4	2	17	0	0.44	0.44	33.3	25.0-200.0
		E	0.34	0	0	0	4	4	0.27	0.32	0.05	11.8	33.3-100.0
Post-Defln	1st 0.1 sec:							4					
	2nd 0.1 sec:							2					
	1	I	0.58	11	5	7	4	27	0	0.54	0.54	46.6	20.0-200.0
		E	1.60	1	9	8	14	32	0.33	1.59	1.26	20.0	12.5-200.0
	2	I	0.54	12	5	5	2	24	0	0.46	0.46	44.4	20.0-200.0
		E	1.16	7	12	12	13	44	0.18	1.60	1.42	27.3	14.3-200.0

Rabbit Number: 1 Fibre Number: 3 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.range (Hz)
Pre-Infln	1	I	0.64	2	1	3	0	6	0.03	0.46	0.43	9.4	6.7- 28.6
		E	1.50	1	0	0	0	1					
	2	I	0.65	1	2	2	0	5	0.01	0.43	0.42	7.7	5.3- 20.0
		E	1.45	0	0	0	0						
	3	I	0.65	4	2	0	1	7	0.03	0.57	0.54	10.8	3.2-200.0
		E	1.41	0	0	0	1	1					
Inflation	(Position: 44.1% tE; Duration of apnoea: 10.54 sec.)												
	1st 1.0 sec:							0					
	Mid 1.0 sec:							25					
	End 1.0 sec:							31					
Post-Infln	1	I	1.08	10	2	2	2	16	0.01	0.94	0.93	14.8	5.6-200.0
		E	0.58	0	0	0	0	0					
	2	I	0.78	3	1	0	0	4	0.03	0.36	0.33	5.1	4.8- 25.0
		E	0.64	0	0	0	0	0					
	3	I	0.71	2	1	1	0	4	0.04	0.41	0.37	5.6	4.8- 14.3
		E	0.72	0	0	0	0	0					
Pre-Defln	1	I	0.65	1	2	3	0	6	0.01	0.43	0.42	9.2	6.5-200.0
		E	1.32	0	0	0	0	0					
	2	I	0.64	1	2	1	0	4	0.07	0.34	0.27	6.3	7.1- 20.0
		E	1.48	0	0	0	0	0					
	3	I	0.65	4	1	2	0	7	0.02	0.42	0.40	10.8	8.3- 33.3
		E	1.51	0	0	0	0	0					
Deflation:	(Position: 51.4%; tE)												
	1st 0.1 sec:							4					
	2nd 0.1 sec:							2					
	1st	I	0.88	1	1	4	3	9	0.11	0.83	0.72	10.2	4.0- 25.0
		E	0.24	0	0	2	1	3	0.14	0.22	0.08	12.5	14.3-200.0
	Mid	I	0.46	0	4	1	2	7	0.11	0.45	0.23	15.2	7.7-100.0
		E	0.28	1	0	4	0	5	0.02	0.21	0.19	17.9	5.9-200.0
	End	I	0.48	3	3	0	0	6	0	0.16	0.16	12.5	12.5-200.0
		E	0.26	1	0	2	1	4	0.03	0.21	0.18	15.4	6.7-100.0
Post-Defln	1st 0.1 sec:							0					
	2nd 0.1 sec:							2					
	1	I	0.47	5	1	2	0	8	0	0.33	0.33	17.0	10.0-200.0
		E	1.48	0	0	0	0	0					
	2	I	0.50	8	0	1	0	9	0	0.27	0.27	18.0	6.3-200.0
		E	1.55	0	0	0	0	0					
	3	I	0.51	3	2	4	1	10	0	0.44	0.44	19.9	11.1-100.0
		E	1.57	0	0	0	0	0					
Augmented		I	0.79	7	1	7	10	25	0.04	0.72	0.68	31.6	4.4-200.0

Breath		E	1.26	0	0	0	0	0					

Rabbit Number: 1 Fibre Number: 4 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.range (Hz)
Pre-Infln	1	I	0.68	6	3	2	1	12	0	0.57	0.57	17.6	9.1-200.0
		E	1.15	0	0	1	0	1	0.67			0.9	
	2	I	0.66	4	1	2	0	7	0.01	0.46	0.45	10.6	4.8- 33.3
		E	1.28	0	0	0	0	0					
	3	I	0.64	8	4	3	0	15	0.03	0.48	0.45	23.4	14.3-200.0
		E	1.34	0	0	0	0	0					
Inflation	(Position: 8.73% tE; Duration of apnoea: 7.80 sec.)												
	1st 0.1 sec:							0					
	2nd 0.1 sec:							0					
	1st 1.0 sec:							1					
	Mid 1.0 sec:							2					
	End 1.0 sec:							8					
Post-Infln	1st 0.1 sec:							17					100.0-200.0
	2nd 0.1 sec:							13					66.7-200.0
	1	I	0.86	1	1	0	0	2	0.19	0.31	0.12	2.3	9.1
		E	0.53	0	0	0	0	0					
	2	I	0.66	1	1	1	0	3	0.03	0.35	0.32	4.5	5.7- 7.1
		E	0.57	0	0	0	0	0					
	3	I	0.65	2	2	1	0	5	0.07	0.41	0.34	7.7	5.9- 20.0
		E	0.60	0	0	0	0	0					
Augmented Breath	1	I	1.18	7	3	2	1	13	0.04	0.99	0.95	11.0	4.8-100.0
		E	0.43	0	0	0	0	0					
Pre-Defln	1	I	0.63	3	1	0	0	4	0.03	0.21	0.18	6.3	11.1- 33.3
		E	1.09	0	0	0	0	0					
	2	I	0.62	1	1	2	0	4	0.05	0.41	0.36	6.5	5.9- 12.5
		E	1.21	0	0	0	0	0					
	3	I	0.65	2	3	1	0	6	0.02	0.38	0.36	9.2	8.3- 20.0
		E	1.31	0	0	0	0	0					
Deflation	(Position: 36.7% tE)												
	1st 0.1 sec:							3					33.3-200.0
	2nd 0.1 sec:							1					
	1st	I	0.84	2	1	3	2	8	0.05	0.77	0.72	9.5	5.0- 28.6
		E	0.26	0	0	2	1	3	0.17	0.23	0.05	11.5	25.0-100.0
	Mid	I	0.50	3	1	1	0	5	0.03	0.30	0.27	10.0	11.1- 25.0
		E	0.26	0	0	2	0	2	0.18			7.7	
	End	I	0.45	2	1	1	1	5	0.01	0.37	0.36	11.1	7.1- 25.0
		E	0.29	0	0	1	2	3	0.21	0.24	0.03	10.3	50.0-200.0
Post-Defln (Augmented Breath)	1st 0.1 sec:							1					
	2nd 0.1 sec:							1					
	1	I	0.86	7	3	6	11	27	0	0.84	0.84	12.8	7.7-200.0
		E	1.03	0	0	0	0	0					

Rabbit Number: 2 Fibre Number: 5 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.Range (Hz)
Pre-Infln	1	I	0.61	29	21	10	1	61	0.01	0.57	0.56	100.0	33.3-200.0
		E	2.11	1	5	9	15	30	0.32	2.10	1.78	14.2	4.0-200.0
	2	I	0.54	23	17	17	8	65	0	0.53	0.53	120.4	33.3-200.0
		E	2.17	4	8	7	12	31	0.02	2.16	2.14	14.3	2.9-200.0
	3	I	0.57	26	16	12	7	61	0	0.55	0.55	107.0	33.3-200.0
		E	2.12	1	6	7	22	36	0.47	2.11	1.64	17.0	7.1-200.0
Inflation	(Position: 30.9% tE; Duration of apnoea: 10.26 sec)												
	1st 0.1 sec:							1					
	2nd 0.1 sec:							0					
	1st 1.0 sec:							13					6.3- 66.7
	Mid 1.0 sec:							94					33.3-200.0
	End 1.0 sec:							141					40.0-200.0
Post-Infln	1st 0.1 sec:							12					50.0-200.0
	2nd 0.1 sec:							12					50.0-200.0

	1	I	1.48	31	22	21	10	84	0.03	1.42	1.39	56.8	14.3-200.0
		E	1.52	2	11	16	11	40	0.08	1.51	1.43	26.3	3.2-200.0
	2	I	0.91	7	5	8	4	24	0.04	0.90	0.86	26.4	8.3-200.0
		E	1.60	0	2	1	8	11	0.73	1.59	0.86	6.9	1.5-200.0
	3	I	0.79	10	5	7	3	25	0.01	0.71	0.70	31.6	12.5-200.0
		E	1.93	0	0	0	11	11	1.84	1.92	0.08	5.7	0.1-200.0
Pre-Defln	1	I	0.52	15	16	9	2	42	0	0.51	0.51	80.8	12.5-200.0
		E	1.91	0	0	0	12	12	1.86	1.90	0.04	66.7	6.3-200.0
	2	I	0.53	25	17	21	7	70	0.01	0.52	0.51	132.1	0.5-200.0
		E	2.00	0	0	0	10	10	1.85	1.99	0.14	5.0	0.1-200.0
	3	I	0.53	24	27	19	5	75	0	0.47	0.47	141.5	40.0-200.0
		E	1.86	0	5	7	20	32	0.80	1.85	1.05	17.2	6.3-200.0
Deflation: (Position: 11.9% tE)													
	1st 0.1 sec:							17					66.7-200.0
	1	I	0.81	38	50	42	17	147	0	0.78	0.78	181.5	28.6-200.0
		E	0.21	0	0	10	8	18	0.12	0.20	0.08	85.7	50.0-200.0
	2	I	0.53	28	24	12	12	91	0.01	0.50	0.49	171.7	66.7-200.0
		E	0.26	0	0	1	0	2	0.18				
	3	I	0.55	31	27	24	17	95	0	0.51	0.51	172.7	66.7-200.0
		E	0.48	1	2	0	0	3	0.01	0.21	0.20	6.3	5.3-200.0
Post-Defln	1st 0.1 sec:							22					100.0-200.0
	2nd 0.1 sec:							8					25.0-200.0
	1	I	0.47	27	24	21	13	85	0	0.45	0.45	180.9	66.7-200.0
		E	1.87	3	15	19	28	65	0.28	1.86	1.58	34.8	12.5-200.0

Rabbit Number: 2 Fibre Number: 6 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.range (Hz)
Pre-Infln	1	I	0.58	24	21	15	12	72	0	0.57	0.57	124.1	20.0-200.0
		E	2.38	4	7	12	18	41	0.40	2.37	1.97	17.2	5.0-200.0
	2	I	0.55	25	19	17	10	71	0	0.51	0.51	129.1	33.3-200.0
		E	2.23	5	12	20	24	61	0.29	2.22	1.93	27.4	8.3-200.0
	3	I	0.55	24	21	21	6	72	0	0.51	0.51	130.9	16.7-200.0
		E	2.26	3	9	12	21	45	0.26	2.25	1.99	19.9	4.4-200.0
Inflation	(Position: 58.93% tE)												
	1st 0.1 sec:							7					33.3-200.0
	2nd 0.1 sec:							16					66.7-200.0
	1st 1.0 sec:							28					9.1-200.0
	Mid 1.0 sec:							88					25.0-200.0
	End 1.0 sec:							137					33.3-200.0
Post-Infln	1st 0.1 sec:							13					50.0-200.0
	2nd 0.1 sec:							7					50.0-200.0
	1	I	1.04	20	15	10	9	54	0	0.98	0.98	51.9	25.0-200.0
		E	1.05	2	0	6	7	15	0.02	1.01	0.99	14.3	1.7-200.0
	2	I	0.91	5	6	5	5	21	0.08	0.89	0.81	23.1	8.3-200.0
		E	1.18	0	0	1	6	7	0.86	1.16	0.30	5.9	7.7-100.0
	3	I	0.82	3	4	2	1	10	0.01	0.79	0.78	12.2	4.8- 20.0
		E	1.33	0	0	1	1	2	0.71	1.26	0.55	1.5	
Pre-Defln	1	I	0.54	12	16	8	3	39	0	0.44	0.44	72.2	25.0-200.0
		E	1.92	0	2	4	9	15	0.76	1.91	1.15	7.8	3.1-200.0
	2	I	0.51	21	15	12	3	51	0	0.44	0.44	100.0	25.0-200.0
		E	2.15	0	10	7	13	30	0.56	2.14	1.58	14.0	5.6-200.0
	3	I	0.50	14	18	15	5	52	0	0.44	0.44	104.0	40.0-200.0
		E	1.96	3	3	6	7	19	0.33	1.92	1.59	9.7	4.5- 50.0
Deflation: (Position 100% tE)													
	1st 0.1 sec:							16					100.0-200.0
	1st	I	0.84	39	36	39	26	140	0	0.81	0.81	166.7	28.6-200.0
		E	0.23	0	0	1	10	11	0.16	0.22	0.06	47.8	40.0-200.0
	Mid	I	0.58	33	23	22	8	86	0	0.56	0.56	148.3	20.0-200.0
		E	0.27	0	0	2	3	5	0.17	0.26	0.09	18.5	25.0-200.0
	End	I	0.58	30	27	29	12	98	0	0.52	0,52	169.0	66.7-200.0
		E	0.45	1	0	0	1	2	0.01	0.38	0.37	4.4	
Post-Defln	1st 0.1 sec:							0					
	2nd 0.1 sec:							1				134.8	
	1	I	0.44	22	27	14	4	67	0	0.38	0.38	152.3	40.0-200.0
		E	1.94	7	10	10	10	37	0	1.93	1.93	19.1	7.1-200.0

2	I	0.46	20	18	19	5	62	0	0.41	0.41	134.8	50.0-200.0
	E	2.03	3	9	12	13	37	0.30	2.02	1.72	18.2	7.7-200.0
3	I	0.45	21	17	13	4	55	0	0.38	0.38	122.2	28.6-200.0
	E	2.21	2	4	11	10	27	0.25	2.20	1.95	12.2	1.8-200.0

Rabbit Number: 2 Fibre Number: 7 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.range (Hz)
Pre-Infln	1	I	0.53	32	27	27	14	100	0	0.52	0.52	188.7	50.0-200.0
		E	1.82	4	13	18	23	58	0.01	1.81	1.80	31.9	3.7-200.0
	2	I	0.52	20	20	26	21	87	0	0.51	0.51	167.3	66.7-200.0
		E	1.99	10	10	12	22	54	0.02	1.98	1.96	27.1	5.3-200.0
	3	I	0.50	19	21	22	12	74	0	0.47	0.47	148.0	40.0-200.0
		E	2.32	0	0	0	5	5	2.30	2.32	0.02	2.2	50.0-200.0
Inflation	(Position: 57.69% tE; Duration of apnoea: 7.56 sec.)												
	1st 0.1 sec:							17					
	2nd 0.1 sec:							19					
	1st 1.0 sec:							8					
	Mid 1.0 sec:							25					
	End 1.0 sec:							77					
Post-Infln	1st 0.1 sec:							12					
	2nd 0.1 sec:							7					50.0-200.0
	1	I	0.80	26	25	31	21	103	0.01	0.79	0.78	128.8	25.0-200.0
		E	0.64	1	1	1	13	16	0	0.63	0.63	25.0	3.2-200.0
	2	I	0.99	24	29	28	23	104	0	0.93	0.93	105.1	28.6-200.0
		E	0.64	0	0	1	2	3	0.47	0.58	0.11	4.7	12.5- 33.3
	3	I	0.77	10	23	19	6	58	0	0.72	0.72	75.3	20.0-200.0
		E	0.49	0	0	1	3	4	0.36	0.41	0.05	8.2	33.3-200.0
Pre-Defln	1	I	0.46	16	17	27	9	69	0	0.44	0.44	150.0	25.0-200.0
		E	2.29	0	0	0	2	2	2.27			0.9	
	2	I	0.44	13	21	21	10	65	0	0.43	0.43	147.7	33.3-200.0
		E	2.02	0	0	0	4	4	2.00	2.01	0.01	1.9	200.0
	3	I	0.50	21	18	15	12	66	0	0.45	0.45	132.0	50.0-200.0
		E	2.26	2	0	0	5	7	0.34	2.20	1.91	3.1	0.6-200.0
Deflation	(Position: 13.24% tE)												
	1st 0.1 sec:							5					
	2nd 0.1 sec:							13					
	1st	I	0.73	36	38	35	19	1	0	0.72	0.72	175.3	50.0-200.0
		E	0.22	0	0	1	6	7	0.16	0.21	0.05	31.8	33.3-200.0
	Mid	I	0.52	19	21	23	10	73	0	0.47	0.47	140.4	100.0-200.0
		E	0.29	0	1	1	0	2	0.14	0.17	0.03	6.9	
	End	I	0.51	26	24	21	20	91	0.01	0.47	0.46	178.4	40.0-200.0
		E	0.30	0	0	3	3	6	0.15	0.26	0.11	20.0	28.6-200.0
Post-Defln	1st 0.1 sec:							22					
	2nd 0.1 sec:							7					
	1	I	0.43	21	24	17	14	76	0	0.41	0.41	176.7	50.0-200.0
		E	1.93	6	10	10	16	42	0.04	1.92	1.88	21.8	7.1-200.0
	2	I	0.44	24	26	16	12	78	0	0.43	0.43	177.3	33.3-200.0
		E	1.91	8	9	4	10	31	0.18	1.90	1.72	16.2	3.9-200.0

Rabbit Number: 2 Fibre Number: 8 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.range (Hz)
Pre-Infln	1	I	0.53	11	7	6	5	29	0	0.50	0.50	54.7	16.7-200.0
		E	2.30	5	3	3	3	14	0.03	2.29	2.26	6.1	1.2-200.0
	2	I	0.51	11	10	8	3	32	0.01	0.46	0.43	62.7	16.7-200.0
		E	2.00	2	2	4	5	13	0.30	1.71	1.41	6.5	1.8-200.0
	3	I	0.56	9	12	5	4	30	0	0.51	0.51	53.6	25.0-200.0
		E	1.96	2	3	6	4	15	0.13	1.89	1.76	7.7	1.9- 25.0
Inflation	(Position 78.0% tE; Duration of apnoea: 6.1 sec.)												
	1st 0.1 sec:							2					
	2nd 0.1 sec:							2					
	1st 1.0 sec:							14					
	Mid 1.0 sec:							11					
	End 1.0 sec:							7					

Post-Infln	1st 0.1 sec:						0						
	2nd 0.1 sec:						0						
	1	I	1.07	2	5	1	1	9	0.01	0.81	0.80	8.4	3.9- 66.7
		E	1.30	3	2	2	2	9	0.07	1.07	1.00	6.9	3.5- 50.0
	2	I	0.82	1	2	0	0	3	0.10	0.29	0.19	3.7	8.3- 14.3
		E	1.41	3	3	2	1	9	0.02	1.25	1.23	6.4	3.5- 25.0
	3	I	0.72	4	1	2	0	7	0.02	0.47	0.45	9.7	5.0-200.0
		E	1.36	3	1	0	3	7	0.01	1.34	1.33	5.1	1.6- 33.3
Pre-Defln	1	I	0.61	7	7	3	1	18	0.02	0.52	0.50	29.5	6.7-200.0
		E	1.39	1	4	2	2	9	0.07	1.28	1.21	6.5	2.2-200.0
	2	I	0.60	5	6	4	2	17	0.01	0.54	0.53	28.3	12.5-200.0
		E	1.89	4	1	3	5	13	0.01	1.88	1.87	6.9	2.2-200.0
Deflation	(Position: 100.0% tE)												
	1st 0.1 sec:						5						
	2nd 0.1 sec:						8						
	1st	I	0.91	16	20	17	15	68	0	0.90	0.90	74.7	25.0-200.0
		E	0.30	0	1	0	6	7	0.11	0.29	0.18	23.3	7.1-200.0
	Mid	I	0.57	13	14	10	6	43	0.02	0.56	0.54	75.4	25.0-200.0
		E	0.42	1	1	1	2	5	0.03	0.41	0.38	11.9	5.9-100.0
	End	I	0.54	11	7	10	7	35	0.01	0.51	0.50	64.8	25.0-200.0
		E	0.40	0	2	2	1	5	0.15	0.38	0.23	12.5	8.3- 40.0

Rabbit Number: 3 Fibre Number: 9 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.range (Hz)
Pre-Infln	1	I	0.47	20	3	14	4	41	0	0.41	0.41	87.2	1.1-200.0
		E	1.29	13	1	5	5	24	0.03	1.18	1.15	18.6	2.0-200.0
	2	I	0.48	17	2	4	3	26	0.04	0.47	0.43	54.2	6.7-200.0
		E	1.36	5	1	1	8	15	0.03	1.32	1.29	11.0	2.6- 50.0
	3	I	0.45	14	13	9	7	43	0	0.41	0.41	95.6	25.0-200.0
		E	1.31	11	0	9	11	31	0.01	1.30	1.29	23.7	2.5-200.0
Inflation	(Position 100% tI; Duration of apnoea: 11.11 sec.)												
	1st 0.1 sec:							1					
	2nd 0.1 sec:							2					
	1st 1.0 sec:							15					
	Mid 1.0 sec:							14					
	End 1.0 sec:							33					
Post-Infln	1st 0.1 sec:							1					
	2nd 0.1 sec:							0					
	1	I	1.27	14	13	15	15	57	0.04	1.24	1.18	44.9	11.1-200.0
		E	0.53	2	2	4	0	8	0.05	0.40	0.35	15.1	10.0- 66.7
	2	I	0.88	8	14	10	10	42	0.04	0.84	0.80	47.7	12.5-200.0
		E	0.54	0	3	1	6	10	0.20	0.50	0.30	18.5	9.1-200.0
	3	I	0.77	7	5	10	7	29	0.02	0.73	0.71	37.7	10.0-200.0
		E	0.51	4	0	0	0	4	0	0.11	0.11	7.8	11.8-100.0
Pre-Defln	1	I	0.47	27	13	5	9	54	0	0.46	0.46	114.9	20.0-200.0
		E	1.52	8	6	8	10	32	0.02	0.51	0.49	21.1	6.3-200.0
	2	I	0.46	15	8	7	6	36	0	0.44	0.44	8.3	25.0-200.0
		E	1.36	9	3	9	10	31	0	1.35	1.35	22.8	2.9-200.0
	3	I	0.50	21	7	16	5	49	0.01	0.47	0.46	98.0	16.7-200.0
		E	1.26	5	7	5	5	22	0.01	1.21	1.20	17.5	4.4-200.0
Deflation	(Position: 73.9% tE)												
	1st 0.1 sec:							12					
	2nd 0.1 sec:							9					
	1st	I	1.06	5	30	20	12	67	0.02	0.92	0.90	63.2	10.0-200.0
		E	0.16	3	1	9	8	21	0.01	0.15	0.14	131.3	20.0-200.0
	Mid	I	0.53	26	20	25	16	87	0	0.52	0.52	164.2	25.0-200.0
		E	0.25	4	6	19	6	35	0.01	0.24	0.23	140.0	33.3-200.0
	End	I	0.47	16	26	21	9	72	0	0.46	0.46	153.2	33.3-200.0
		E	0.26	2	0	8	19	29	0.01	0.25	0.24	111.5	7.7-200.0
Post-Defln	1st 0.1 sec:							5					
	2nd 0.1 sec:							3					
	1	I	0.41	29	19	12	9	69	0	0.39	0.39	168.3	33.3-200.0
		E	1.24	12	1	5	6	24	0.05	1.20	1.15	19.4	3.6-200.0
	2	I	0.42	9	4	4	0	17	0	0.26	0.26	40.5	12.5-200.0
		E	1.10	5	12	12	14	43	0.01	1.09	1.08	39.1	9.1-200.0
	3	I	0.41	22	11	9	2	44	0	0.36	0.36	107.3	14.3-200.0

	E	1.22	13	1	8	10	32	0	1.18	1.18	26.2	2.5-200.0

Rabbit Number: 3 Fibre Number: 10 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr. (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq-range (Hz)
Pre-Infln	1	I	0.45	12	8	9	4	33	0.40	0.85	0.45	73.3	20.0-200.0
		E	1.32	0	0	0	3	3	1.28	1.30	0.02	2.3	
	2	I	0.45	8	3	7	2	20	0	0.42	0.42	44.4	14.3-200.0
		E	1.48	0	0	0	2	2	1.46	1.47	0.01	1.4	
	3	I	0.48	9	10	8	4	31	0.01	0.46	0.45	64.6	25.0-200.0
		E	1.40	0	0	0	2	2	1.38	1.39	0.01	1.4	
Inflation (Position 43.5% tI; Duration of apnoea: 10.3 sec.)													
	1st 0.1 sec:							4					
	2nd 0.1 sec:							4					
	1st 1.0 sec:							0					
	2nd 1.0 sec:							0					
	3rd 1.0 sec:							0					
Post-Infln	1st 0.1 sec:							0					
	2nd 0.1 sec:							0					
	1	I	0.98	6	6	11	9	32	0.03	0.75	0.72	32.7	20.0-200.0
		E	0.65	0	0	0	0	0					
	2	I	0.57	5	4	3	6	18	0.04	0.74	0.70	31.6	16.7-200.0
		E	0.69	0	0	0	1	1	0.68			1.4	
	3	I	0.67	8	6	5	5	24	0.03	0.64	0.61	35.8	14.3-200.0
		E	0.64	0	0	0	0	0					
Pre-Defln	1	I	0.45	7	6	8	6	27	0.02	0.43	0.41	60.0	25.0-200.0
		E	1.26	0	0	0	5	5	1.00	1.26	0.26	4.0	4.2-200.0
	2	I	0.46	9	11	5	4	29	0.04	0.42	0.38	63.0	33.3-200.0
		E	1.11	1	1	0	1	3	0.01	1.09	1.08	2.7	1.5- 2.3
	3	I	0.46	9	10	9	9	37	0.01	0.45	0.44	80.8	25.0-200.0
		E	1.09	0	0	0	3	3	1.05	1.08	0.03	2.8	50.0-100.0
Deflation (Position: 33.0% tE)													
	1st 0.1 sec:							8					
	1st	I	0.64	14	17	12	11	54	0.01	0.64	0.63	84.4	40.0-200.0
		E	0.23	1	0	6	10	17	0.02	0.22	0.20	73.9	7.7-200.0
	Mid	I	0.45	10	5	8	5	28	0	0.41	0.41	62.2	25.0-200.0
		E	0.23	0	0	4	11	15	0.14	0.22	0.08	65.2	66.7-200.0
	End	I	0.43	8	14	7	7	36	0	0.40	0.40	83.7	25.0-200.0
		E	0.23	0	0	5	10	15	0.14	1.04	0.90	65.2	
Post-Defln	1st 0.1 sec:							2					
	2nd 0.1 sec:							0					
	1	I	0.42	9	9	9	2	29	0	0.38	0.38	69.0	16.7-200.0
		E	1.13	0	0	0	1	1	1.11			0.9	
	2	I	0.41	11	6	9	4	30	0	0.38	0.38	73.2	33.3-200.0
		E	1.12	0	0	0	3	3	1.09	1.11	0.02	2.7	50.0-200.0
	3	I	0.43	8	6	9	4	27	0	0.36	0.36	62.8	33.3-200.0
		E	1.08	0	0	0	2	2	1.06	1.07	1.01	1.9	

Rabbit Number: 3 Fibre Number: 10 PSR: Block

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq-range (Hz)
Pre-Infln	1	I	0.91	4	5	5	4	18	0	0.87	0.87	19.8	10.0-100.0
		E	1.48	10	10	13	6	39	0.01	1.36	1.35	26.3	12.5-200.0
	2	I	0.91	4	4	3	3	14	0.10	0.89	0.79	15.4	7.1- 50.0
		E	1.36	8	7	10	6	31	0.03	1.35	1.32	22.8	10.0-200.0
	3	I	0.92	3	5	7	0	15	0	0.61	0.61	16.3	5.9-200.0
		E	1.31	9	8	6	6	29	0.01	1.22	1.21	22.1	12.5- 66.7
Inflation (Position 33.3% tE; Duration of apnoea: 1.28 sec.)													
	1st 0.1 sec:							1					
	2nd 0.1 sec:							0					
Augmented Breath	1	I	0.67	0	1	8	24	33	0.27	0.66	0.39	49.3	16.7-200.0
		E	0.19	6	7	2	0	15	0	0.12	0.12	78.9	20.0-200.0
Post-Infln	1st 0.1 sec:							0					
	2nd 0.1 sec:							2					

	1	I	1.23	4	8	7	8	27	0.10	1.21	1.11	22.0	11.1-200.0
		E	0.80	8	9	11	6	34	0.06	0.77	0.71	42.5	20.0-200.0
	2	I	1.04	2	5	3	4	14	0.01	1.03	1.02	13.5	4.8- 28.6
		E	1.05	13	11	17	11	52	0	1.04	1.04	49.5	18.2-200.0
	3	I	0.99	4	9	7	5	25	0.04	0.96	0.92	25.3	12.5-200.0
		E	0.99	8	9	12	8	37	0.02	0.93	0.91	37.4	16.7-200.0
Pre-Defln	1	I	0.80	3	3	2	4	12	0.02	0.79	0.77	15.0	11.1- 50.0
		E	1.50	12	7	8	5	32	0.03	1.34	1.31	21.3	12.5- 66.7
	2	I	0.83	3	2	3	2	10	0.01	0.72	0.71	12.1	8.3- 50.0
		E	1.62	10	15	15	9	49	0.01	1.60	1.59	30.3	12.5-200.0
	3	I	0.85	3	4	2	3	12	0.12	0.83	0.71	14.1	8.3- 40.0
		E	1.61	9	10	10	8	37	0.04	1.54	1.50	22.0	12.5-200.0
Deflation	(Position: 27.4% tE)												
	1st 0.1 sec:							4					
	2nd 0.1 sec:							0					
	1st	I	0.66	4	6	4	3	17	0.02	0.63	0.61	25.8	14.3-100.0
		E	1.53	17	20	13	5	55	0.01	1.48	1.47	35.9	6.3-200.0
	Mid	I	0.75	5	6	7	5	23	0.06	0.64	0.58	30.7	20.0-200.0
		E	1.46	11	12	10	4	37	0.04	1.44	1.40	25.3	4.3-200.0
	End	I	0.88	8	13	14	11	46	0.01	0.87	0.86	52.3	25.0-200.0
		E	1.34	2	0	0	3	5	0.01	1.30	1.29	3.7	0.9-200.0
Post-Defln	1st 0.1 sec:							0					
	2nd 0.1 sec:							1					
	1	I	0.67	10	12	13	9	44	0.02	0.63	0.61	65.7	28.6-200.0
		E	0.95	1	0	1	1	3	0.12	0.94	0.82	3.2	1.8- 3.7
	2	I	0.67	7	12	11	7	37	0.05	0.65	0.60	55.2	33.3-200.0
		E	1.11	0	1	4	3	8	0.52	0.89	0.37	7.2	9.1- 66.7

Rabbit Number: 3 Fibre Number: 11 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr (sec)	No of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.range (Hz)
Pre-Infln	1	I	0.51	3	4	2	2	11	0.05	0.39	0.33	21.6	16.7- 66.7
		E	1.59	0	0	0	0	0					
	2	I	0.52	2	1	3	1	7	0.08	0.43	0.35	13.5	11.1-200.0
		E	1.54	0	0	0	0	0					
	3	I	0.49	2	3	3	1	9	0.07	0.38	0.31	18.4	13.3-100.0
		E	1.60	0	0	0	0	0					
Inflation	(Position: 49.7% tE; Duration of apnoea: 7.46 sec.)												
	1st 0.1 sec:							0					
	2nd 0.1 sec:							0					
	1st 1.0 sec:							0					
	Mid 1.0 sec:							0					
	End 1.0 sec:							0					
Post-Infln	1st 0.1 sec:							0					
	2nd 0.1 sec:							0					
	1	I	1.16	0	0	0	0	0					
		E	0.91	0	0	0	0	0					
	2	I	0.62	0	0	0	0	0					
		E	0.96	0	0	0	0	0					
	3	I	0.63	0	0	0	0	0					
		E	0.95	0	0	0	0	0					
Pre-Defln	1	I	0.51	1	1	1	0	3	0.07	0.34	0.27	5.9	7.1
		E	1.57	0	0	0	0	0					
	2	I	0.50	2	1	1	0	4	0.06	0.30	0.24	8.0	8.3- 20.0
		E	1.30	0	0	0	0	0					
	3	I	0.50	2	1	2	0	5	0.10	0.36	0.26	10.0	8.3-200.0
		E	1.21	0	0	0	0	0					
Augmented Breath	1	I	1.11	6	14	26	35	81	0.07	1.10	1.03	73.0	11.1-200.0
		E	0.96	6	2	6	2	16	0.01	0.79	0.78	16.7	3.3-200.0
Deflation	(Position: 48.0% tI)												
	1st 0.1 sec:							0					
	2nd 0.1 sec:							0					
	1st	I	0.92	0	3	5	3	11	0.27	0.87	0.60	12.0	6.3-100.0
		E	0.63	0	1	0	0	1	0.30			1.6	
	Mid	I	0.61	4	5	5	2	16	0.04	0.48	0.44	26.2	16.7-200.0
		E	0.62	0	0	0	0	0					

	End	I	0.51	7	4	4	2	17	0.01	0.43	0.42	33.3	20.0-200.0
		E	0.79	0	0	0	2	2	0.76			2.5	
Post-Defln	1st 0.1 sec:							0					
	2nd 0.1 sec:							0					
	1	I	0.45	3	5	2	0	10	0.03	0.33	0.30	22.2	10.0-200.0
		E	0.91	0	0	0	0	0					
	2	I	0.41	3	4	2	0	9	0.03	0.28	0.25	22.0	18.2-200.0
		E	0.89	0	0	0	0	0					
	3	I	0.44	3	4	3	0	10	0.04	0.30	0.26	22.7	14.3-200.0
		E	0.95	0	0	0	0	0					

Rabbit Number: 3 Fibre Number: 12 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.Range (Hz)
Pre-Infln	1	I	0.58	0	0	0	0	0					
		E	1.52	4	0	0	1	5	0.00	1.42	1.42	3.3	0.8- 25.0
	2	I	0.61	0	0	1	1	2	0.46	0.56	0.10	3.3	10.0
		E	1.43	6	0	0	2	8	0.00	1.26	1.26	5.6	1.1- 28.6
	3	I	0.59	0	0	0	2	2	0.47	0.55	0.08	3.4	14.3
		E	1.53	5	0	0	1	6	0.02	1.44	1.42	3.9	0.8- 33.3
Inflation	(Position: 53.7% tE; Duration of apnoea: 10.02 sec.)												
	1st 0.1 sec:							0					
	2nd 0.1 sec:							0					
	1st 1.0 sec:							0					
	Mid 1.0 sec:							0					
	End 1.0 sec:							0					
Post-Infln	1st 0.1 sec:							0					
	2nd 0.1 sec:							0					
	1	I	0.85	4	4	4	4	16	0.02	0.83	0.81	18.8	14.3- 25.0
		E	1.32	9	10	9	10	38	0.03	1.31	1.28	28.8	16.7- 50.0
	2	I	0.52	4	3	3	4	14	0.02	0.49	0.47	26.9	18.1- 33.3
		E	1.08	8	8	7	8	31	0.01	1.06	1.05	28.7	16.7- 40.0
	3	I	0.62	4	3	3	3	13	0.01	0.60	0.59	21.0	16.7- 28.6
		E	0.95	6	6	6	7	25	0.02	0.94	0.97	26.3	14.3- 29.5
Pre-Defln	1	I	0.55	1	0	0	1	2	0.05	0.48	0.43	3.6	2.3
		E	1.44	5	0	1	4	10	0.00	1.41	1.41	6.9	1.2- 25.0
	2	I	0.61	0	0	0	1	1	0.56			1.6	
		E	1.57	3	0	0	1	4	0.02	1.56	1.54	2.5	0.7- 20.0
	3	I	0.57	0	0	0	1	1	0.56				
		E	1.68	3	0	0	0	3	0.07	0.15	0.08	1.8	16.7- 40.0
Deflation	(Position: 53.2% tE)												
	1st 0.1 sec:							0					
	2nd 0.1 sec:							0					
	1st	I	0.59	0	0	0	1	1	0.58			1.7	
		E	1.49	2	0	0	0	2	0.05	0.11	0.06	1.3	16.7
	Mid	I	0.60	0	0	0	0	0					
		E	1.43	2	0	0	0	2	0.03	0.12	0.09	1.4	11.1
	End	I	0.60	0	0	0	0	0					
		E	1.37	2	0	0	0	2	0.03	0.11	0.08	1.5	12.5
Post-Defln	1st 0.1 sec:							0					
	2nd 0.1 sec:							0					
	1	I	0.60	0	1	0	0	1	0.28				
		E	1.30	1	0	0	0	1	0.05				

Rabbit Number: 3 Fibre Number: 12 PSR: Block

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.Range (Hz)
Pre-Infln	1	I	0.72	4	7	8	6	25	0.04	0.70	0.66	34.7	25.0-200.0
		E	1.67	3	0	0	1	4	0.02	1.65	1.63	2.4	0.8- 25.0
	2	I	0.76	7	7	10	7	31	0.03	0.75	0.72	40.8	25.0-200.0
		E	1.68	0	0	0	0	0					
	3	I	0.88	6	9	9	8	32	0.00	0.84	0.84	36.4	16.7-200.0
		E	1.56	1	0	0	1	2	9.55			1.3	
Inflation	(Position: 6.3% tI; Duration of apnoea: 9.60 sec.)												
	1st 0.1 sec:							3					

	2nd 0.1 sec:					5					
	1st 1.0 sec:					1					
	Mid 1.0 sec:					3					
	End 1.0 sec:					2					
Post-Infln	1st 0.1 sec:					1					
	2nd 0.1 sec:					0					
	1	I	1.11	6	8	8	8	30	0.06	1.10	1.04	27.0	16.7-200.0
		E	1.13	7	3	16	14	40	0.01	1.11	1.10	35.4	4.6-200.0
	2	I	0.90	7	7	7	11	32	0.02	0.89	0.87	35.6	18.2-200.0
		E	1.14	8	0	6	10	24	0.02	1.12	1.10	21.1	2.2-200.0
Pre-Defln	1	I	0.94	5	7	8	10	30	0.03	0.93	0.90	31.9	20.0-200.0
		E	1.56	2	3	1	1	7	0.01	1.55	1.54	4.5	1.4-200.0
	2	I	0.69	6	8	8	9	31	0.03	0.68	0.65	44.9	28.6-200.0
		E	1.54	3	0	0	1	4	0.02	1.53	1.51	2.6	1.0-100.0
Deflation	(Position: 70.7% tI)												
	1st 0.1 sec:					7					
	2nd 0.1 sec:					8					
	1st	I	0.41	5	8	5	4	22	0.02	0.37	0.35	53.7	33.3-200.0
		E	1.96	2	4	37	37	80	0.01	1.94	1.93	40.8	1.2-200.0
	Mid	I	0.63	12	10	9	8	39	0.02	0.62	0.60	61.9	22.2-200.0
		E	1.06	5	0	0	13	18	0.00	1.05	1.05	17.0	1.2-200.0
	End	I	0.61	11	11	16	6	44	0.00	0.60	0.60	72.1	28.6-200.0
		E	1.18	0	2	0	9	11	0.41	1.16	0.75	9.3	2.5-200.0
Post-Defln	1st 0.1 sec:					1					
	2nd 0.1 sec:					0					
	1	I	0.60	15	12	11	6	44	0.00	0.54	0.54	73.3	40.0-200.0
		E	0.93	4	0	2	0	6	0.02	0.57	0.55	7.2	2.6-100.0
	2	I	0.56	9	10	8	7	34	0.01	0.54	0.53	60.7	22.2-200.0
		E	0.77	2	1	0	2	5	0.06	0.75	0.69	6.5	2.6- 13.3
	3	I	0.55	8	8	10	5	31	0.02	0.51	0.49	56.4	20.0-200.0
		E	0.93	0	0	0	0	0					

Rabbit Number: 4 Fibre Number : 13 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr. (sec)	No of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.range (Hz)
Pre-Infln	1	I	0.87	4	3	3	2	12	0.02	0.69	0.60	13.8	12.5- 66.7
		E	0.97	0	1	0	0	1	0.32			1.0	
	2	I	0.66	3	4	2	1	10	0.01	0.50	0.49	15.2	12.5- 50.0
		E	0.96	0	0	0	0	0					
	3	I	0.73	10	7	5	1	23	0.03	0.57	0.54	31.5	20.0-200.0
		E	1.00	0	0	0	0	0					
Inflation	(Position: 28.0% tI; Duration of apnoea: 9.03sec.)												
	1st 0.1 sec:					2							
	2nd 0.1 sec:					2							
	1st 1.0 sec:					0							
	Mid 1.0 sec:					0							
	End 1.0 sec:					1							
Post-Infln	1st 0.1 sec:					0							
	2nd 0.1 sec:					1							
	1	I	1.46	4	4	5	1	14	0.01	1.16	1.15	9.6	1.4- 12.5
		E	0.60	0	0	0	0	0					
	2	I	0.91	2	1	3	1	7	0.03	0.70	0.67	7.7	3.9- 20.0
		E	0.70	0	0	0	0	0					
	3	I	0.77	1	1	0	0	2	0.11	0.35	0.24	2.6	4.0
Pre-Defln	1	I	0.62	3	3	1	0	7	0.02	0.35	0.33	11.3	12.5- 22.2
		E	1.16	1	0	0	0	1	0.01			0.9	
	2	I	0.62	4	2	1	2	9	0.01	0.58	0.57	14.5	8.3- 66.7
		E	1.20	4	0	0	0	4	0.00	0.16	0.16	3.3	10.0- 33.3
	3	I	0.64	2	2	2	2	8	0.03	0.62	0.59	12.5	6.3- 20.0
		E	1.20	3	0	0	0	3	0.00	0.06	0.06	2.5	28.6- 40.0
Deflation	(Position: 10.2% tE)												
	1st 0.1 sec:					1							
	2nd 0.1 sec:					2							
	1st	I	1.15	5	5	5	3	18	0.03	0.99	0.96	15.7	11.1- 50.0
		E	0.28	0	1	2	0	3	0.08	0.20	0.12	10.7	10.0- 66.7
	Mid	I	0.73	3	4	5	1	13	0.07	0.59	0.52	17.8	16.7-200.0

	E	0.33	0	0	0	2	2	0.25	0.31	0.06	6.1	16.7
End	I	0.69	3	4	4	2	13	0.05	0.59	0.54	18.8	14.3- 33.3
	E	0.29	0	0	0	1	1	0.23			3.5	

Post-Defln	1st 0.1 sec:						0					
	2nd 0.1 sec:						1					
	1 (Augmented Breath)											
	I	0.94	11	8	18	53	90	0.01	0.93	0.92	95.7	16.7-200.0
	E	1.37	0	0	0	7	7	0.00	0.07	0.07	5.1	40.0-200.0
2	I	0.51	2	1	1	2	6	0.04	0.49	0.45	11.8	5.3- 33.3
	E	1.23	3	0	0	0	3	0.00	0.07	0.07	2.4	25.0- 33.3
3	I	0.53	2	1	1	2	6	0.03	0.52	0.49	11.3	5.0- 40.0
	E	1.78	0	0	1	0	1	1.17			0.6	
4	I	0.52	2	1	2	0	5	0.00	0.36	0.36	9.6	
	E	1.70	0	1	0	0	1	0.62				

Rabbit Number: 4 Fibre Number: 14 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.range (Hz)
Pre-Infln	1	I	0.66	8	2	4	0	14	0.01	0.43	0.42	21.2	10.0-100.0
		E	1.42	0	0	0	0	0					
	2	I	0.67	6	6	3	0	15	0.02	0.48	0.46	22.4	9.1-100.0
		E	1.50	0	0	0	0	0					
	3	I	0.60	4	1	1	0	6	0.04	0.38	0.34	10.0	7.7- 50.0
		E	1.47	0	0	0	0	0					
Augmented Breath	1	I	1.15	10	8	44	66	128	0.02	1.14	1.12	111.3	12.5-200.0
		E	1.43	17	0	0	0	17	0.00	0.26	0.26	11.9	11.1-200.0
Inflation	(Position 12.3% tE; Duration of apnoea: 8.40 sec.)												
	1st 0.1 sec:							0					
	2nd 0.1 sec:							0					
	1st 1.0 sec:							0					
	Mid 1.0 sec:							0					
	End 1.0 sec:							0					
Post-Infln	1st 0.1 sec:							1					
	2nd 0.1 sec:							0					
1	I	1.80	1	0	0	0	1	0.17			0.6		
	E	0.54	0	0	0	0	0						
2	I	0.76	0	0	0	0	0						
	E	0.64	0	0	0	0	0						
3	I	0.66	0	0	0	0	0						
	E	0.71	0	0	0	0	0						
Pre-Defln	1	I	0.58	5	2	1	2	10	0.01	0.50	0.49	17.2	7.7- 66.7
		E	1.61	0	0	0	0	0					
	2	I	0.60	3	1	1	1	6	0.01	0.48	0.47	10.0	7.7-200.0
		E	1.58	0	0	0	0	0					
Deflation	(Position: 81.1% tE)												
	1st 0.1 sec:							3					
	2nd 0.1 sec:							1					
1st	I	1.55	5	12	15	7	39	0.14	1.48	1.37	25.2	11.1-200.0
	E	0.34	0	0	2	1	3	0.20	0.30	0.10	8.8	14.3- 50.0
Mid	I	0.84	9	10	12	4	35	0.01	0.73	0.72	41.7	16.7-200.0
	E	0.35	0	0	3	2	5	0.21	0.32	0.11	14.3	14.3-100.0
End	I	0.72	11	13	8	5	37	0.00	0.65	0.65	51.4	16.7-200.0
	E	0.31	0	0	0	7	7	0.23	0.29	0.06	22.6	33.3-200.0
Post-Defln	1st 0.1 sec:							7					
	2nd 0.1 sec:							6					
1	I	0.53	8	5	2	0	15	0.01	0.37	0.36	28.3	12.5-200.0
	E	1.32	0	0	0	0	0					
2	I	0.54	7	7	2	1	17	0.02	0.50	0.48	31.5	7.1-200.0
	E	1.34	0	0	0	0	0					
3	I	0.56	5	3	1	1	10	0.02	0.42	0.40	17.9	12.5-100.0
	E	1.38	0	0	0	1	1	1.37			0.7	

Rabbit Number: 4 Fibre Number: 14 PSR: Block

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.range (Hz)

Pre-Infln	1	I	1.02	3	3	7	4	17	0.16	0.92	0.76	16.7	6.7-200.0
		E	1.24	0	0	0	0	0					
	2	I	1.01	3	4	5	5	17	0.02	0.93	0.91	16.8	7.7- 66.7
		E	1.27	0	0	0	0	0					
Inflation	(Position 80.4% tI; Duration of apnoea: 8.65 sec.)												
	1st 0.1 sec:							1					
	2nd 0.1 sec:							1					
	1st 1.0 sec:							0					
	Mid 1.0 sec:							0					
	End 1.0 sec:							1					
Post-Infln	1st 0.1 sec:							0					
	2nd 0.1 sec:							0					
	1	I	2.01	0	1	1	10	12	0.55	1.94	1.39	6.0	1.2-200.0
		E	0.39	0	0	0	0	0					
	2	I	1.16	0	0	0	0	0					
		E	0.61	0	1	0	0	1	0.18			1.6	
	3	I	1.05	0	0	0	0	0					
		E	0.62	0	0	0	0	0					
Pre-Defln	1	I	0.99	6	3	4	4	17	0.04	0.92	0.88	17.1	6.7-100.0
		E	1.17	0	0	0	0	0					
	2	I	0.96	4	6	4	3	17	0.02	0.69	0.67	17.7	10.0-200.0
		E	1.13	0	0	0	0	0					
	3	I	1.10	9	8	4	1	22	0.04	0.89	0.85	20.0	7.1-200.0
		E	1.05	0	0	0	0	0					
Deflation	(Position: 66.1% tE)												
	1st 0.1 sec:							0					
	2nd 0.1 sec:							0					
	1st	I	2.39	7	7	11	4	29	0.16	2.05	1.89	12.1	5.0-100.0
		E	0.34	0	0	0	0	0					
	Mid	I	1.29	4	2	5	4	15	0.20	1.16	0.88	11.6	5.3- 66.7
		E	0.40	0	0	0	1	1	0.31			2.5	
	End	I	0.99	1	6	9	7	23	0.12	0.94	0.82	23.2	6.7-200.0
		E	0.52	0	0	0	0	0					
Post-Defln	1st 0.1 sec:							1					
	2nd 0.1 sec:							1					
	1	I	0.65	3	7	4	3	17	0.00	0.57	0.57	26.1	7.7-200.0
		E	0.71	0	0	0	0	0					
	2	I	0.65	4	4	5	0	13	0.03	0.46	0.43	20.0	9.1-200.0
		E	0.71	1	0	0	0	1	0.15			1.4	
	3	I	0.69	0	4	4	0	8	0.18	0.47	0.29	11.6	12.5-200.0
		E	0.70	0	0	0	0	0					
Augmented Breath	1	I	1.36	6	7	32	67	112	0.06	1.35	1.29	82.3	6.3-200.0
		E	0.62	10	0	2	6	18	0.01	0.61	0.60	29.0	4.4-200.0

Rabbit Number: 5 Fibre Number: 15 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr (sec)	No of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.range (Hz)
Pre-Infln (Run 1)	1	I	0.68	3	1	2	3	9	0.05	0.66	0.61	13.2	7.1- 33.3
		E	1.81	7	15	15	17	54	0.12	1.79	1.67	29.8	14.3- 50.0
	2	I	0.64	2	2	2	3	9	0.04	0.62	0.58	14.1	5.9- 40.0
		E	1.99	10	17	20	17	64	0.03	1.98	1.36	32.2	8.3- 66.7
	3	I	0.67	2	3	4	4	13	0.01	0.66	0.65	19.4	9.1- 50.0
		E	1.89	16	21	26	24	87	0.02	1.88	1.86	46.0	16.7-100.0
Inflation (Run 1)	(Position: 27.3% tI; Duration of apnoea: 8.71 sec.)												
	1st 0.1 sec:							2					
	2nd 0.1 sec:							0					
	1st 1.0 sec:							7					
	Mid 1.0 sec:							13					
	End 1.0 sec:							17					
Post-Infln (Run 1)	1st 0.1 sec:							2					
	2nd 0.1 sec:							5					
	1	I	1.34	14	16	15	16	61	0.02	1.33	1.31	45.5	33.3- 66.7
		E	0.56	6	7	8	8	29	0.01	0.55	0.54	51.8	28.6-100.0
	2	I	0.96	10	10	9	10	39	0.01	0.94	0.93	40.6	20.0- 66.7
		E	0.65	6	9	7	8	30	0.01	0.63	0.62	46.2	25.0- 66.7
	3	I	0.61	4	7	4	6	21	0.03	0.60	0.57	34.4	16.7- 50.0

		E	0.90	6	9	9	9	33	0.01	0.89	0.88	36.7	16.7- 66.7
Pre-Defln	1	I	0.63	3	3	1	4	11	0.05	0.61	0.56	17.5	7.1- 33.3
(Run 1)		E	2.04	13	17	18	19	67	0.08	2.01	1.93	32.8	14.3- 50.0
	2	I	0.61	4	2	2	2	10	0.00	0.54	0.54	16.4	7.1- 40.0
		E	2.16	14	19	20	21	74	0.07	2.15	2.08	34.3	16.7- 66.7
	3	I	0.64	5	1	2	3	11	0.01	0.61	0.60	17.2	7.1- 50.0
		E	1.79	14	17	23	15	69	0.01	1.77	1.76	38.5	9.1-100.0
Deflation	(Position: 41.5% tE)												
(Run 1)	1st 0.1 sec:							4					
	2nd 0.1 sec:							2					
	1st	I	1.35	6	2	13	18	39	0.05	1.34	1.29	28.9	2.9-100.0
		E	0.20	2	3	3	2	10	0.01	0.19	0.18	50.0	40.0-100.0
	Mid	I	0.77	6	11	8	9	34	0.02	0.75	0.73	44.2	14.3-100.0
		E	0.26	3	5	3	1	12	0.01	0.22	0.21	46.2	25.0-100.0
	End	I	0.69	8	6	12	8	34	0.05	0.68	0.63	49.3	16.7-100.0
		E	0.20	4	3	1	1	9	0.00	0.18	0.18	45.0	14.3-100.0
Post-Defln	1st 0.1 sec:							3					
(Run 1)	2nd 0.1 sec:							0					
	1	I	0.54	1	0	1	0	2	0.10	0.40	0.30	3.7	
		E	1.95	6	18	21	21	66	0.19	1.94	1.75	33.8	8.3-100.0
	2	I	0.57	2	0	2	0	4	0.05	0.34	0.29	7.0	4.8- 25.0
		E	2.03	12	21	21	22	76	0.03	2.01	1.98	37.4	7.7- 66.7
Pre-Infln	1	I	0.59	2	0	2	0	4	0.01	0.34	0.33	6.8	3.9- 40.0
(Run 2)		E	1.66	10	14	15	19	58	0.01	1.63	1.62	34.9	9.1-100.0
	2	I	0.65	1	0	1	1	3	0.09	0.60	0.51	4.6	3.2- 5.0
		E	1.58	7	14	18	20	59	0.04	1.57	1.53	101.7	6.7-100.0
Inflation	(Position: 71.0% tI; Duration of apnoea: 7.39 sec.)												
(Run 2)	1st 0.1 sec:							3					
	2nd 0.1 sec:							0					
	1st 1.0 sec:							6					
	Mid 1.0 sec:							13					
	End 1.0 sec:							18					
Post-Infln	1st 0.1 sec:							4					
(Run 2)	2nd 0.1 sec:							4					
	1	I	1.41	17	18	19	20	74	0.00	1.40	1.40	52.5	28.6-100.0
		E	0.43	6	9	8	5	28	0.01	0.42	0.41	65.1	33.3-100.0
	2	I	1.11	18	16	15	17	66	0.01	1.10	1.09	59.5	25.0-100.0
		E	0.44	7	7	8	7	29	0.00	0.43	0.43	65.9	50.0-100.0
	3	I	0.95	13	12	11	12	48	0.01	0.94	0.93	50.5	28.6-100.0
		E	0.43	6	7	6	7	26	0.00	0.42	0.42	60.5	40.0-100.0
Pre-Defln	1	I	0.64	6	3	1	5	15	0.01	0.61	0.60	23.4	7.1- 50.0
(Run 2)		E	2.09	22	27	30	23	102	0.01	2.08	2.07	48.8	16.6-100.0
	2	I	0.61	1	2	2	2	7	0.06	0.54	0.48	11.5	5.3- 40.0
		E	1.89	16	13	14	14	57	0.03	1.88	1.85	30.2	5.6-100.0
Deflation	(Position: 32.7% tE)												
(Run 2)	1st 0.1 sec:							2					
	2nd 0.1 sec:							2					
	1st	I	1.25	4	11	12	14	41	0.17	1.21	0.04	32.8	14.3-100.0
		E	0.24	4	4	3	0	11	0.00	0.15	0.15	45.8	50.0-100.0
	Mid	I	0.69	8	6	11	7	32	0.01	0.68	0.67	46.4	16.7-100.0
		E	0.24	5	4	1	1	11	0.00	0.19	0.19	45.8	16.7-100.0
	End	I	0.58	8	5	10	6	29	0.02	0.57	0.55	50.0	20.0-100.0
		E	0.26	4	4	3	0	11	0.01	0.19	0.18	42.3	25.0-100.0
Post-Defln	1st 0.1 sec:							3					
(Run 2)	2nd 0.1 sec:							0					
	1	I	0.53	3	1	1	1	6	0.01	0.46	0.45	11.3	4.8- 40.0
		E	1.60	7	16	20	21	64	0.18	1.58	1.40	40.0	16.7-100.0
	2	I	0.51	3	0	2	0	5	0.01	0.34	0.33	9.8	4.8- 33.3
		E	2.03	12	23	26	28	89	0.01	2.02	2.01	43.8	7.7-100.0

Rabbit Number: 5 Fibre Number: 15 PSR: Block

Condition	Br. No.	Ph.	Ph.Dr (sec)	No of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.Range (Hz)
Pre-Infln	1	I	1.46	1	1	2	1	5	0.15	1.38	1.23	3.4	3.2
(Run 1)		E	1.97	8	11	8	4	31	0.08	1.89	1.81	15.7	5.9- 33.3

	2	I	1.69	2	0	1	2	5	0.09	1.64	1.55	3.0	1.6- 3.5
		E	2.24	11	13	11	7	42	0.03	2.22	2.19	18.8	7.1- 40.0
Inflation (Run 1)	(Position 7.6% tE; Duration of apnoea: 7.53sec.)												
	1st 0.1 sec:							1					
	2nd 0.1 sec:							1					
	1st 1.0 sec:							10					
	Mid 1.0 sec:							6					
	End 1.0 sec:							11					
Post-Infln (Run 1)	1st 0.1 sec:							1					
	2nd 0.1 sec:							2					
	1	I	2.62	13	11	10	7	41	0.01	2.50	2.49	15.6	4.8- 40.0
		E	1.70	11	13	11	9	44	0.06	1.63	1.57	25.9	16.7- 40.0
Pre-Defln (Run 1)	1	I	1.50	2	1	0	3	6	0.02	1.48	1.46	4.0	1.6- 22.2
		E	2.60	12	18	14	10	54	0.07	2.52	2.45	20.8	9.1- 50.0
Deflation (Run 1)	(Position: 15.0% tI)												
	1st 0.1 sec:							1					
	2nd 0.1 sec:							0					
	1st	I	2.71	9	12	16	17	54	0.01	2.68	2.67	19.9	4.2- 50.0
		E	2.25	9	9	5	3	26	0.01	2.06	2.05	11.6	3.2- 50.0
	End	I	1.69	11	15	12	15	53	0.02	1.67	1.65	31.4	16.7- 66.7
		E	2.17	4	5	4	3	16	0.02	2.10	2.08	7.4	3.2- 50.0
Pre-Infln (Run 2)	1	I	1.32	0	2	1	1	4	0.33	1.26	0.93	3.0	3.2
		E	1.60	6	11	10	8	35	0.10	1.57	1.47	21.9	14.3- 40.0
	2	I	1.42	1	1	1	2	5	0.20	1.34	1.14	3.5	3.2- 5.0
		E	1.53	5	11	12	11	39	0.02	1.52	1.50	25.5	4.4- 50.0
Inflation (Run 2)	(Position 50.0% tI; Duration of apnoea: 12.19 sec.)												
	1st 0.1 sec:							1					
	2nd 0.1 sec:							1					
	1st 1.0 sec:							4					
	Mid 1.0 sec:							8					
	End 1.0 sec:							9					
Post-Infln (Run 2)	1st 0.1 sec:							1					
	2nd 0.1 sec:							3					
	1	I	2.41	11	8	7	5	31	0.05	2.32	2.27	12.9	4.8- 33.3
		E	1.36	10	12	12	10	44	0.00	1.30	1.30	32.4	20.0- 50.0
	2	I	1.49	2	1	2	1	6	0.10	1.42	1.32	4.0	2.9- 9.1
		E	1.50	6	12	12	12	42	0.13	1.49	1.36	28.0	11.1- 40.0
	3	I	1.39	2	1	1	1	5	0.02	1.30	1.28	3.6	3.1
		E	1.60	7	13	13	13	46	0.11	1.59	1.48	28.8	9.1- 50.0
Pre-Defln (Run 2)	1	I	1.37	1	1	1	1	4	0.17	1.15	0.98	2.9	2.9- 3.1
		E	1.62	7	13	13	12	45	0.09	1.60	1.51	27.8	11.1- 50.0
	2	I	1.39	2	1	1	1	5	0.01	1.28	1.27	3.6	2.9- 3.2
		E	1.87	7	16	12	14	49	0.05	1.85	1.80	26.2	6.7- 50.0
	3	I	1.30	2	1	1	1	5	0.01	1.23	1.22	3.8	3.2
		E	1.19	4	10	10	11	35	0.09	1.18	1.09	29.4	11.1- 50.0
Deflation (Run 2)	(Position: 76.3% tE)												
	1st 0.1 sec:						1						
	2nd 0.1 sec:						2						
	1st	I	2.49	8	14	16	18	56	0.01	2.47	2.46	22.5	5.3- 50.0
		E	2.18	12	14	11	6	43	0.00	2.15	2.15	19.7	3.5- 50.0
	Mid	I	1.62	10	12	14	17	53	0.00	1.61	1.61	32.7	10.0- 66.7
		E	2.05	11	7	8	3	29	0.01	1.77	1.76	14.2	4.8- 50.0
	End	I	1.30	10	12	13	13	48	0.01	1.29	1.28	36.9	16.7- 66.7
		E	0.65	6	2	0	2	10	0.00	0.62	0.62	15.4	3.9 100.0
Post-Defln (Run 2)	1st 0.1 sec:							2					
	2nd 0.1 sec:							1					
	1	I	1.02	0	1	0	1	2	0.00	0.81	0.81	2.0	2.0- 3.2
		E	1.54	6	11	6	8	31	0.00	1.52	1.52	20.1	5.9- 40.0

Rabbit Number:5 Fibre Number:16 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr (sec)	No of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.range (Hz)
Pre-Infln	1	I	0.88	2	4	2	5	13	0.09	0.81	0.72	14.8	5.9- 50.0
		E	1.78	4	13	11	13	41	0.10	1.77	1.67	23.0	3.7- 50.0

	2	I	0.93	5	5	3	3	16	0.08	0.86	0.78	17.2	6.3- 66.7
		E	2.37	13	17	16	24	70	0.03	2.36	2.33	29.5	6.7- 66.7
	3	I	0.88	10	12	7	10	39	0.00	0.87	0.87	44.3	16.7- 66.7
		E	1.42	16	15	19	15	65	0.01	1.41	1.40	45.8	16.7- 66.7
Inflation	(Position: 76.8% tE; Duration of apnoea: 9.55 sec.)												
	1st 0.1 sec:							6					
	2nd 0.1 sec:							1					
	1st 1.0 sec:							27					
	Mid 1.0 sec:							15					
	End 1.0 sec:							16					
Post-Infln	1st 0.1 sec:							3					
	2nd 0.1 sec:							2					
	1	I	1.40	16	16	17	17	66	0.01	1.38	1.37	47.1	25.0-100.0
		E	0.74	7	7	8	5	27	0.00	0.71	0.71	36.5	16.7-100.0
	2	I	1.02	9	8	11	10	38	0.01	1.00	0.99	37.3	12.5- 66.7
		E	0.83	8	7	4	1	20	0.02	0.79	0.77	24.1	9.1- 50.0
	3	I	0.94	7	8	8	10	33	0.00	0.92	0.92	35.1	16.7- 66.7
		E	0.93	6	9	9	7	31	0.01	0.92	0.91	33.3	16.7- 66.7
Pre-Defln	1	I	0.83	5	5	6	9	25	0.04	0.82	0.78	30.1	9.1-200.0
		E	2.95	22	26	25	27	100	0.01	2.94	2.93	33.9	11.1-100.0
	2	I	0.82	5	6	6	4	21	0.05	0.81	0.76	25.6	10.0- 50.0
		E	3.80	27	31	32	22	112	0.03	3.79	3.76	29.5	4.6- 66.7
	3	I	0.94	2	0	3	0	5	0.06	0.70	0.64	5.3	2.4- 33.3
		E	1.97	10	15	17	20	62	0.02	1.96	1.94	31.5	10.0- 66.7
Deflation	(Position: 74.4% tI)												
	1st 0.1 sec:							5					
	2nd 0.1 sec:							5					
	1st	I	1.26	12	17	17	15	61	0.01	1.25	1.24	48.4	20.0-100.0
		E	0.25	2	1	0	0	3	0.01	0.07	0.06	12.0	25.0- 50.0
	Mid	I	1.09	6	10	12	10	38	0.06	1.08	1.02	34.9	16.7- 66.7
		E	0.23	1	1	0	1	3	0.02	0.20	0.18	13.0	11.1- 12.5
	End	I	1.05	7	11	14	17	49	0.02	1.04	1.02	46.7	7.7- 66.7
		E	0.16	1	0	0	0	1	0.01			6.3	
Post-Defln	1st 0.1 sec:							1					
	2nd 0.1 sec:							2					
	1	I	0.69	1	1	0	2	4	0.06	0.66	0.60	5.8	3.5- 7.7
		E	2.78	12	18	21	23	74	0.27	2.74	2.47	26.6	12.5- 50.0
Pre-Infln (Run 2)	1 (Augmented Breath)												
		I	1.30	7	5	3	5	20	0.02	1.25	1.23	15.4	8.3- 66.7
		E	1.80	7	19	17	15	58	0.01	1.79	1.78	32.2	4.6-200.0
	2	I	0.67	4	4	2	3	13	0.01	0.61	0.60	19.4	10.0- 33.3
		E	2.33	14	19	24	24	81	0.09	2.32	2.23	34.8	10.0-200.0
Inflation (Run 2)	(Position: 35.8% tI)												
	1st 0.1 sec:							5					
	2nd 0.1 sec:							1					
	1st 1.0 sec:							3					
	Mid 1.0 sec:							4					
	End 1.0 sec:							8					
Post-Infln (Run 2)	1st 0.1 sec:							2					
	2nd 0.1 sec:							2					
	1	I	1.32	9	11	11	12	43	0.00	1.29	1.29	35.6	14.3-100.0
		E	0.72	3	8	10	4	25	0.00	0.66	0.66	34.7	6.7-100.0
	2	I	0.91	5	7	7	4	23	0.09	0.87	0.78	25.3	9.1- 50.0
		E	0.88	4	5	6	7	22	0.01	0.87	0.86	25.0	11.1- 50.0
	3	I	0.88	4	4	4	5	17	0.07	0.87	0.80	19.3	5.6- 66.7
		E	1.04	3	7	8	10	28	0.02	1.03	1.01	26.9	8.3- 50.0
Pre-Defln	1	I	0.88	5	3	4	5	17	0.06	0.86	0.80	19.3	10.0- 66.7
		E	1.33	6	11	11	11	39	0.08	1.32	1.24	29.3	12.5- 50.0
	2	I	0.84	2	5	4	6	17	0.05	0.82	0.77	20.2	8.3- 50.0
		E	1.60	8	13	13	8	42	0.05	1.58	1.53	26.3	6.3- 50.0
	3	I	0.91	1	1	1	1	4	0.05	0.77	0.72	4.4	3.2- 5.9
		E	1.92	7	12	18	17	54	0.04	1.90	1.86	28.1	7.7- 66.7
Deflation:	(Position: 13.7% tE)												
	1st 0.1 sec:							0					
	2nd 0.1 sec:							1					
	1st	I	1.67	12	9	14	15	50	0.01	1.66	1.65	29.9	14.3- 66.7

		E	0.39	3	1	0	0	4	0.02	0.14	0.12	10.3	25.0- 66.7
	Mid	I	0.76	4	5	7	5	21	0.01	0.71	0.70	27.6	14.3- 66.7
		E	0.47	4	0	0	2	6	0.00	0.45	0.45	12.8	2.9- 50.0
	End	I	0.66	2	5	5	6	18	0.07	0.65	0.58	27.3	16.7- 50.0
		E	0.53	3	0	0	1	4	0.07	0.47	0.40	7.5	2.9-100.0
Post-Defln	1st 0.1 sec:							1					
	2nd 0.1 sec:							0					
	1	I	0.66	0	0	1	0	1	0.04	0.37	0.33	1.5	
		E	2.18	6	15	15	11	47	0.28	2.17	1.89	21.6	7.1- 40.0

Rabbit Number: 5 Fibre Number: 16 PSR: Block

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.range (Hz)
Pre-Infln	1	I	1.56	7	5	1	2	15	0.00	1.34	1.34	9.6	3.6-200.0
		E	2.47	14	17	20	15	66	0.07	2.45	2.38	26.7	8.3- 66.7
	2	I	1.56	5	3	4	3	15	0.01	1.47	1.46	9.6	5.9- 25.0
		E	2.97	22	26	22	19	89	0.00	2.93	2.93	30.0	11.1- 50.0
Inflation	(Position: 19.2% tI; Duration of apnoea: 14.52 sec.)												
	1st 0.1 sec:							1					
	2nd 0.1 sec:							1					
	1st 1.0 sec:							7					
	Mid 1.0 sec:							10					
	End 1.0 sec:							12					
Post-Infln	1st 0.1 sec:							2					
	2nd 0.1 sec:							3					
	1	I	3.54	23	26	24	21	94	0.06	3.53	3.47	26.6	8.3- 50.0
		E	1.86	15	15	15	16	61	0.04	1.85	1.81	32.8	11.1-200.0
Pre-Defln	1	I	1.56	4	4	4	3	15	0.07	1.51	1.44	9.6	5.3- 33.3
		E	2.76	20	13	19	20	72	0.11	2.75	2.64	26.1	7.7-200.0
	2	I	1.55	7	5	6	3	21	0.01	1.53	1.52	13.5	4.6- 33.3
		E	2.46	17	16	20	20	73	0.01	2.45	2.44	29.7	6.7- 66.7
	3	I	1.57	4	4	4	4	16	0.01	1.46	1.45	10.2	5.6- 25.0
		E	2.74	17	13	10	13	53	0.01	2.72	2.71	19.3	3.7- 50.0
Deflation	(Position: 53.8% tI)												
	1st 0.1 sec:							2					
	2nd 0.1 sec:							0					
	1st	I	1.64	4	4	5	5	18	0.02	1.61	1.59	11.0	4.5- 28.6
		E	3.09	18	21	15	20	74	0.01	3.05	3.04	23.9	7.7- 50.0
	End	I	2.46	3	6	12	15	36	0.32	2.43	2.11	14.6	4.2- 50.0
		E	3.14	16	28	18	21	83	0.00	3.13	3.13	26.4	5.6-200.0
Post-Defln	1st 0.1 sec:							5					
	2nd 0.1 sec:							4					
	1	I	1.35	1	2	2	3	8	0.27	1.28	1.01	5.9	3.2- 20.0
		E	1.93	9	15	16	19	59	0.03	1.92	1.89	30.6	6.7- 66.7
Pre-Infln	1	I	1.38	2	2	3	5	12	0.13	1.37	1.24	8.7	5.9- 22.2
		E	2.24	16	16	20	20	72	0.03	2.23	2.20	32.1	6.7- 20.0
	2	I	1.50	1	2	2	5	10	0.21	1.48	1.27	6.7	3.3- 25.0
		E	2.52	20	25	23	19	87	0.01	2.47	2.46	34.5	7.1- 66.7
	3	I	1.51	3	3	6	14	26	0.06	1.50	1.44	17.2	5.6-100.0
		E	2.22	26	19	20	14	79	0.03	2.18	2.15	35.6	7.7-100.0
Inflation	(Position: 8.0% tI; Duration of apnoea: 20.15 sec.)												
	1st 0.1 sec:							3					
	2nd 0.1 sec:							2					
	1st 1.0 sec:							13					
	Mid 1.0 sec:							13					
	End 1.0 sec:							23					
Post-Infln	1st 0.1 sec:							4					
	2nd 0.1 sec:							3					
	1	I	2.29	21	17	15	9	62	0.01	2.25	2.24	27.1	12.5-100.0
		E	1.50	9	16	15	13	53	0.02	2.25	2.23	35.3	7.7-100.0
	2	I	1.74	12	8	3	6	29	0.01	1.72	1.71	16.7	3.7- 40.0
		E	1.53	7	13	14	14	48	0.00	1.52	1.52	31.4	7.1- 50.0
	3	I	1.69	9	4	5	5	23	0.06	1.63	1.57	13.6	4.6- 40.0
		E	1.72	9	16	15	16	56	0.04	1.69	1.65	32.4	10.0-100.0

Deflation	(Position: 22.8% tE)												
	1st 0.1 sec:					3							
	2nd 0.1 sec:					3							
	1st	I	1.93	6	11	11	11	39	0.13	1.92	1.79	20.2	8.3- 50.0
		E	4.40	34	31	27	31	123	0.03	4.32	4.29	28.0	7.1- 66.7
	End	I	2.14	19	18	20	20	77	0.01	2.11	2.10	36.0	11.1-100.0
		E	2.68	23	29	24	19	95	0.02	2.65	2.63	35.4	9.1- 83.3
Post-Defln	1st 0.1 sec:					3							
	2nd 0.1 sec:					1							
	1	I	1.25	5	3	3	7	18	0.02	1.24	1.22	14.4	3.6- 40.0
		E	1.95	11	16	5	3	35	0.01	1.86	1.85	17.9	4.0- 66.7

Rabbit Number: 6 Fibre Number: 17 PSR: Intact

Condtion	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.Range (Hz)
Pre-Infl	1	I	0.56	0	1	4	2	7	0.15	0.51	0.36	12.5	7.7-200.0
(Run 1)		E	1.35	0	0	0	0	0					
	2	I	0.57	6	4	3	0	13	0.00	0.54	0.54	22.8	11.8-100.0
		E	1.34	0	0	0	0	0					
Inflation	(Position 52.6% tE; Duration of apnoea: 10.15sec.)												
(Run 1)	1st 0.1 sec:					0							
	2nd 0.1 sec:					0							
	1st 1.0 sec;					0							
	Mid 1.0 sec:					0							
	End 1.0 sec:					6							
Post-Infl	1st 0.1 sec:					1							
(Run 1)	2nd 0.1 sec:					0							
	1	I	0.82	6	5	3	3	17	0.04	0.77	0.73	20.7	5.9-200.0
		E	0.41	0	1	0	0	1	0.20				
	2	I	0.64	5	2	8	2	17	0.00	0.52	0.52	26.6	9.1-200.0
		E	0.43	0	0	0	0	0					
	3	I	0.62	3	3	6	4	16	0.02	0.54	0.52	25.8	10.0-200.0
		E	0.46	0	0	0	0	0					
Pre-Defln	1	I	0.59	3	1	0	2	6	0.03	0.48	0.45	10.2	4.4- 28.6
(Run 1)		E	1.60	0	0	0	0	0					
	2	I	0.57	5	3	4	1	13	0.07	0.54	0.47	22.8	6.3-200.0
		E	1.56	0	0	0	0	0					
	3	I	0.57	2	4	3	1	10	0.02	0.53	0.51	17.5	7.7-200.0
		E	1.52	0	0	0	0	0					
Deflation	(Position : 26.3% tI)												
(Run 1)	1st 0.1 sec:					0							
	1st	I	0.74	6	6	3	9	24	0.03	0.69	0.66	32.4	12.5-200.0
		E	0.23	0	0	1	0	1	0.16				
	Mid	I	0.60	4	3	6	1	14	0.00	0.46	0.46	23.3	9.1-200.0
		E	0.24	0	0	0	0	0					
	End	I	0.51	6	3	2	5	16	0.02	0.46	0.44	31.4	12.5-200.0
		E	0.25	0	0	0	2	2	0.20	0.24	0.04		
Post-Defln	1st 0.1 sec:					4							33.3- 50.0
(Run 1)	1	I	0.49	3	4	6	2	15	0.02	0.42	0.40	30.6	13.3-100.0
		E	0.86	0	0	0	0	0					
	2	I	0.48	1	4	4	2	11	0.06	0.45	0.39	22.9	8.3-200.0
		E	0.89	0	0	0	0	0					
	3	I	0.48	2	4	5	2	13	0.02	0.44	0.42	27.1	11.1-200.0
		E	0.87	0	0	0	0	0					
Added Dead Space													
	1	I	0.55	2	3	4	1	10	0.04	0.46	0.42	18.2	9.5- 40.0
		E	0.78	1	1	1	1	4	0.19	0.63	0.44	5.1	4.8- 8.3
	2	I	0.56	4	4	7	3	18	0.00	0.50	0.50	32.1	7.7-200.0
		E	0.70	0	0	3	0	3	0.21	0.29	0.08		
	3	I	0.55	3	4	4	0	11	0.10	0.41	0.31	20.0	14.3-100.0
		E	0.69	1	3	0	1	5	0.09	0.66	0.57		

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.Range (Hz)
Pre-Infln	1	I	0.54	4	1	4	3	12	0	0.47	0.47	22.2	7.1-200.0
(Run 2)		E	1.36	0	0	0	0	0					
	2	I	0.53	3	3	2	0	8	0.02	0.40	0.38	15.1	7.7-200.0

		E	1.10	0	0	0	0	0					
	3	I	0.55	2	1	0	1	4	0.02	0.43	0.41	7.3	4.3- 22.2
		E	0.94	0	0	0	0	0					
Inflation	(Position: 55.5% tI; Duration of inflation apnoea: 10.65 sec.)												
	1st 0.1 sec:							0					
	1st 1.0 sec:							0					
	Mid 1.0 sec:							0					
	End 1.0 sec:							8					
Post-Infln	1st 0.1 sec:							1					
	2nd 0.1 sec:							1					
	1	I	0.77	5	5	4	5	19	0.06	0.75	0.69	24.7	9.1-200.0
		E	0.35	0	1	2	1	4	0.15	0.34	0.19	11.4	7.7- 40.0
	2	I	0.64	1	3	2	3	9	0.16	0.60	0.44	14.1	7.7- 33.3
		E	0.32	0	0	0	0	0					
	3	I	0.62	0	4	3	3	9	0.18	0.56	0.38	14.5	9.1-200.0
		E	0.37	0	0	1	0	1	0.22			2.7	
Pre-Defln	1	I	0.53	2	1	6	0	9	0.01	0.40	0.39	17.0	6.7-200.0
		E	1.18	0	0	0	0	0					
	2	I	0.57	5	2	2	0	9	0.01	0.41	0.40	15.8	10.0-200.0
		E	0.92	0	0	0	0	0					
	3	I	0.57	4	3	2	2	11	0.02	0.48	0.46	19.3	10.0-100.0
Deflation	(Position : 15.9% : tE)												
	1st 0.1 sec:							1					
	1st	I	0.95	5	5	9	6	25	0.07	0.92	0.85	26.3	10.0-200.0
		E	0.19	0	0	0	1	1	0.15			5.3	
	Mid	I	0.60	5	7	5	2	19	0.01	0.54	0.53	31.7	14.3-200.0
		E	0.21	0	0	0	0	0					
	End	I	0.59	3	2	3	4	12	0.01	0.58	0.57	20.3	7.7-200.0
		E	0.20	0	0	0	0	0					
Post-Defln	1st 0.1 sec:							0					
	2nd 0.1 sec:							0					
	1	I	0.51	5	1	1	1	8	0.04	0.40	0.36	15.7	7.7-200.0
		E	0.78	0	0	0	0	0					
	2	I	0.48	5	0	2	2	9	0.00	0.42	0.42	18.8	7.1-100.0
		E	0.89	0	0	0	1	1	0.87			1.1	
	3	I	0.49	3	3	3	2	11	0.03	0.42	0.39	22.4	16.7-200.0
		E	0.90	0	1	0	0	1	0.38			1.1	

Rabbit Number: 6 Fibre Number: 17 PSR: Block

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq. Range (Hz)
Pre-Infln	1	I	0.99	9	6	9	8	32	0.00	0.96	0.96	32.3	10.5-200.0
		E	1.86	2	1	0	0	3	0.19	0.28	0.09	1.6	
	2	I	1.03	7	9	11	7	34	0.00	0.96	0.96	33.0	11.1-200.0
		E	1.77	8	2	0	0	10	0.00	0.38	0.38	5.6	7.7-100.0
	3	I	0.99	6	8	12	7	33	0.03	0.89	0.86	33.3	11.1-200.0
		E	1.76	7	4	0	2	13	0.12	1.64	1.52	7.4	1.3-050.0
Inflation	(Position: 26.1% tE: Duration of apnoea: 7.0 sec.)												
	1st 0.1 sec:							4					
	2nd 0.1 sec:							3					
	1st 1.0 sec:							1					
	Mid 1.0 sec:							0					
	End 1.0 sec:							0					
Post-Infln	1st 0.1 sec:							1					
	1	I	1.38	5	8	7	13	33	0.02	1.35	1.33	23.9	10.0-200.0
		E	0.95	0	3	0	0	0	0.26	0.42	0.16	3.2	10.0- 16.7
	2	I	1.03	5	7	6	7	25	0.08	1.00	0.92	24.3	8.3-200.0
		E	1.03	0	3	0	0	3	0.20	0.86	0.66	8.7	2.8- 50.0
	3	I	0.99	9	7	9	7	32	0.02	0.92	0.90	32.3	12.5-200.0
		E	1.10	3	11	0	0	14	0.20	0.54	0.34	12.7	16.7-200.0
Pre-Defln	1	I	0.97	8	4	11	8	31	0.02	0.96	0.94	32.0	11.1-200.0
		E	1.65	4	3	0	0	7	0.02	0.25	0.23	4.2	15.4- 50.0
	2	I	0.96	12	6	8	7	33	0.00	0.88	0.88	34.4	16.7-200.0
		E	1.54	2	4	0	0	6	0.34	0.52	0.18	3.9	16.7- 50.0
	3	I	0.95	7	8	9	8	32	0.00	0.88	0.88	33.7	16.7-200.0
		E	1.61	1	4	0	1	6	0.28	1.24	0.96	3.7	1.6- 25.0

Deflation	(Position: 23.2% tI)												
	1st 0.1 sec:						2						
	2nd 0.1 sec:						3						
	1st	I	0.91	3	13	11	9	36	0.00	0.83	0.83	39.6	10.0-200.0
		E	1.49	4	1	0	6	11	0.24	1.42	1.18	7.4	1.1-200.0
	Mid	I	1.00	9	7	9	8	33	0.00	0.93	0.93	33.0	15.4-200.0
		E	1.60	2	0	0	11	13	0.20	1.52	1.32	8.1	0.9-200.0
	End	I	0.95	8	10	18	5	41	0.00	0.76	0.76	43.2	33.3-200.0
		E	1.50	2	0	0	7	9	0.24	1.46	1.22	6.0	1.0-200.0
Post-Defln	1st 0.1 sec:						3						
	2nd 0.1 sec:						5						
	1	I	0.86	11	14	12	14	51	0.00	0.82	0.82	59.3	20.0-200.0
		E	0.85	2	6	0	1	9	0.00	0.66	0.66	10.6	2.1-200.0
	2	I	0.76	12	8	15	12	47	0.00	0.73	0.73	61.8	18.2-200.0
		E	0.81	3	5	0	0	8	0.16	0.33	0.17	9.9	20.0-100.0
	3	I	0.79	12	9	8	13	42	0.00	0.73	0.73	53.2	16.7-200.0
		E	0.88	4	5	0	0	9	0.18	0.43	0.25	10.2	25.0-200.0
Pre-Infln	1	I	0.80	7	4	5	9	25	0.02	0.74	0.72	31.3	10.0-200.0
(Run 2)		E	1.08	4	4	0	3	11	0.12	1.06	0.94	10.2	1.9-100.0
	2	I	0.74	6	8	7	6	27	0.04	0.70	0.66	36.5	12.5-200.0
		E	1.06	3	4	0	1	8	0.22	1.00	0.78	7.5	14.3-200.0
	3	I	0.84	6	8	6	13	33	0.02	0.82	0.80	39.3	12.5-200.0
		E	1.12	5	5	0	1	11	0.18	1.04	0.86	9.8	1.7-200.0
Inflation	(Position: 77.8% tE; Duration of apnoea: 9.9 sec)												
	1st 0.1 sec:						0						
	2nd 0.1 sec:						0						
	1st 1 sec:						3						
	Mid 1 sec:						0						
	End 1 sec:						4						
Post-Infln	1st 0.1 sec:						0						
	2nd 0.1 sec:						0						
	1	I	1.08	8	11	16	16	51	0.04	1.06	1.02	47.2	10.0-200.0
		E	0.62	0	5	0	0	5	0.18	0.28	0.10	8.1	25.0-200.0
	2	I	0.84	8	5	10	7	30	0.00	0.80	0.80	35.7	6.3-100.0
		E	0.64	1	2	1	0	4	0.00	0.42	0.42	6.3	5.0- 25.0
	3	I	0.86	8	9	5	12	34	0.04	0.82	0.78	39.5	16.7-200.0
		E	0.74	0	7	0	0	7	0.20	0.30	0.10	9.5	25.0-200.0
Pre-Defln	1	I	0.82	7	9	7	6	29	0.00	0.76	0.76	35.4	16.7-200.0
		E	1.12	2	5	0	0	7	0.22	0.36	0.14	6.3	16.7-200.0
	2	I	0.80	9	7	7	10	33	0.00	0.78	0.78	41.3	16.7-200.0
		E	1.12	4	3	0	0	7	0.20	0.40	0.20	6.3	16.7-200.0
	3	I	0.84	5	4	12	5	26	0.00	0.80	0.80	31.0	10.0-200.0
		E	1.12	3	7	0	0	10	0.16	0.54	0.38	8.9	12.5-100.0
Deflation:	(Position: 35.7% tE)												
	1st 0.1 sec:						2						
	2nd 0.1 sec:						0						
	1st	I	1.02	6	10	11	8	35	0.00	0.98	0.98	34.3	11.1-200.0
		E	1.12	0	0	0	0	0					
	Mid	I	1.02	11	5	11	10	37	0.02	0.98	0.96	36.3	10.0-200.0
		E	1.16	2	4	0	0	6	0.24	0.44	0.20	5.2	8.3-100.0
	End	I	1.08	8	13	13	14	48	0.04	1.04	1.00	44.4	10.0-200.0
		E	1.26	1	7	2	0	10	0.22	0.80	0.58	7.9	6.3-200.0
Post-Defln	1st 0.1 sec:						0						
	2nd 0.1 sec:						2						
	1	I	0.76	12	13	11	11	47	0.00	0.74	0.74	61.8	14.3-200.0
		E	0.76	4	7	1	1	13	0.12	0.74	0.62	17.1	3.1-200.0
	2	I	0.70	8	8	7	11	34	0.04	0.66	0.62	48.6	16.7-200.0
		E	0.72	2	7	2	1	12	0.14	0.70	0.56	16.7	4.5-200.0
	3	I	0.70	10	8	7	10	35	0.00	0.64	0.64	50.0	16.7-200.0
		E	0.76	3	9	1	0	13	0.14	0.38	0.24	17.1	14.3-200.0

Rabbit Number: 6 Fibre Number: 18 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr. (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.Range (Hz)
Pre-Infl	1	I	0.72	0	0	3	4	7	0.38	0.63	0.25	9.7	9.1-200.0
		E	1.02	0	0	0	0	0					

	2	I	0.71	3	5	3	4	15	0.04	0.68	0.64	21.1	10.0-200.0
		E	1.07	2	0	0	0	2	0.06	0.21	0.15	1.9	
	3	I	0.72	2	3	4	1	10	0.10	0.57	0.47	13.9	12.5-200.0
		E	1.07	0	0	0	0	0					
Added Dead Space													
	1	I	0.63	5	4	5	2	16	0.00	0.59	0.59	25.4	8.3-200.0
		E	0.61	0	0	1	0	1	0.41				
	2	I	0.64	6	3	4	2	15	0.01	0.53	0.52	23.4	6.7-200.0
		E	0.63	1	0	0	0	1	0.04			1.6	
	3	I	0.66	3	5	3	4	15	0.03	0.64	0.61	22.7	10.0-200.0
		E	0.59	0	0	0	0	0					
Inflation (Position: 14.1% tI; Duration of apnoea: 8.00 sec.)													
	1st 0.1 sec:							2					
	2nd 0.1 sec:							2					
	1st 1.0 sec:							5					
	Mid 1.0 sec:							2					
	End 1.0 sec:							0					
Post-Infl	1st 0.1 sec:							0					
	2nd 0.1 sec:							0					
	1	I	0.70	4	2	5	2	13	0.10	0.61	0.51	18.6	7.7-200.0
		E	0.48	0	0	0	1	1	0.43			2.1	
	2	I	0.70	2	3	2	2	9	0.13	0.62	0.49	12.9	8.3- 33.3
		E	0.51	0	2	3	0	5	0.15	0.33	0.18	9.8	12.5- 40.0
	3	I	0.67	4	2	4	3	13	0.09	0.66	0.57	19.0	6.7-100.0
		E	0.49	0	0	0	0	0					
Pre-Defl	1	I	0.73	4	4	3	2	13	0.02	0.67	0.65	17.8	6.7-100.0
		E	0.84	1	0	1	0	2	0.01	0.52	0.51	2.4	
	2	I	0.70	1	4	3	3	11	0.00	0.53	0.53	15.7	5.6-200.0
		E	0.95	0	2	0	0	2	0.31	0.44	0.13	2.1	
	3	I	0.73	5	5	3	2	15	0.00	0.62	0.62	20.5	12.5- 50.0
		E	0.90	0	1	0	0	1	0.24				
Deflation (Position : 16.7% tE)													
(Run 1)	1st 0.1 sec:							0					
	1st	I	0.75	4	5	3	3	15	0.05	0.72	0.67	20.0	7.7- 40.0
		E	0.91	3	3	0	2	8	0.05	0.87	0.82	8.8	3.0- 50.0
	Mid	I	0.55	2	1	4	5	12	0.02	0.54	0.52	21.8	7.7-200.0
		E	0.94	1	1	1	6	9	0.02	0.92	0.90	9.6	3.9- 50.0
	End	I	0.49	3	1	5	3	12	0.02	0.45	0.43	24.5	10.0- 50.0
		E	1.06	0	0	1	4	5	0.72	0.96	0.24	4.7	10.0- 33.3
Post-Defl	1st 0.1 sec:							1					
	2nd 0.1 sec:							0					
	1	I	0.67	4	1	3	2	10	0.01	0.64	0.63	14.9	6.7- 40.0
		E	0.49	0	0	2	2	4	0.31	0.44	0.13	8.2	20.0- 33.3
	2	I	0.63	5	3	5	4	17	0.02	0.59	0.57	27.0	14.3-200.0
		E	0.46	0	0	1	1	2	0.32	0.43	0.11	4.3	
	3	I	0.61	5	1	4	2	12	0.01	0.54	0.53	19.7	5.6- 50.0
		E	0.45	1	0	2	1	4	0.08	0.36	0.28	8.9	5.0- 40.0
Pre-Defl	(Run 2)												
	1	I	0.67	2	3	4	2	11	0.12	0.57	0.45	16.4	12.5- 50.0
		E	0.80	0	0	0	0	0					
	2	I	0.68	6	3	2	2	13	0.02	0.66	0.64	19.1	5.9-200.0
		E	0.83	0	0	0	0	0					
	3	I	0.69	1	3	3	1	8	0.08	0.57	0.49	11.6	4.4-200.0
		E	0.86	0	0	1	0	1	0.50				
Deflation (Position : 84.1% tI)													
(Run 2)	1st 0.1 sec:							2					
	2nd 0.1 sec:							1					
	1st	I	0.62	3	2	0	1	6	0.02	0.56	0.54	9.7	3.6-28.6
		E	1.17	1	1	1	4	7	0.02	1.13	1.11	6.0	2.1-50.0
	Mid	I	0.50	2	2	0	2	6	0.01	0.04	0.03	12.0	5.9-200.0
		E	1.16	2	1	2	2	7	0.05	1.13	0.08	6.0	3.6-20.0
	End	I	0.51	2	2	1	1	6	0.00	0.39	0.39	11.8	7.7-40.0
		E	0.98	0	1	0	2	3	0.45	0.94	0.49	3.0	4.8-74.0
Post-Defl	1st 0.1 sec:							5					
	2nd 0.1 sec:							0					
	1	I	0.65	4	2	4	2	12	0.03	0.55	0.52	18.5	9.1-40.0
		E	0.53	0	0	1	0	1	0.37			1.9	

	2	I	0.62	2	3	4	5	14	0.10	0.59	0.58	22.6	13.3-200.0
		E	0.52	1	3	0	0	4	0.02	0.25	0.23	7.7	6.7-33.3
	3	I	0.59	2	3	4	1	10	0.07	0.53	0.46	16.9	7.7-100.0
		E	0.53	0	0	3	0	3	0.30	0.35	0.05	5.7	33.3-50.0
Pre-Infl	1	I	0.71	2	3	2	4	11	0.00	0.70	0.70	15.5	6.3-66.7
		E	0.94	0	0	0	0	0					
	2	I	0.70	3	2	3	3	11	0.06	0.67	0.61	15.7	10.0-40.0
		E	0.91	0	1	0	3	4	0.06	0.89	0.83	4.4	2.3-40.0
	3	I	0.71	0	1	6	2	9	0.32	0.69	0.37	12.7	8.3-200.0
		E	0.87	3	0	0	0	3	0.00	0.21	0.21	3.4	
Inflation	(Position: 66.7% tE; Duration of apnoea: 6.63 sec.)												
	1st 0.1 sec:							0					
	2nd 0.1 sec:							0					
	1st 1.0 sec:							1					
	2nd 1.0 sec:							5					
	3rd 1.0 sec:							2					
Post-Infl	1st 0.1 sec:							0					
	2nd 0.1 sec:							1					
	1	I	0.97	4	3	2	5	14	0.00	0.89	0.89	14.4	7.7-200.0
		E	0.40	0	0	0	0	0					
	2	I	0.65	2	4	1	3	10	0.11	0.63	0.52	15.4	6.7-100.0
		E	0.45	0	0	1	1	2	0.35	0.40	0.05	4.4	
	3	I	0.64	2	2	1	2	7	0.00	0.62	0.62	10.9	7.7-14.3
		E	0.51	1	0	0	0	1	0.06			2.0	

Rabbit Number: 7 Fibre Number: 19 PSR Intact

Condition	Br. No.	Ph.	Ph.Dr (sec)	No Q1	of Q2	Spikes Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.Range (Hz)
Pre-Infln	1	I	0.58	4	5	3	0	12	0.01	0.41	0.40	20.7	12.5-200.0
		E	3.57	0	0	0	0	0					
	2	I	0.62	8	4	4	0	16	0.01	0.41	0.40	25.8	15.4-200.0
		E	2.16	0	0	0	0	0					
	3	I	0.57	4	3	4	0	11	0.03	0.44	0.41	19.3	12.5- 66.7
		E	2.70	0	0	0	0	0					
Inflation	(Position: 73.3% tE; Duration of apnoea: 13.9 sec.)												
	1st 0.1 sec:							0					
	2nd 0.1 sec:							0					
	1st 1.0 sec:							0					
	Mid 1.0 sec:							13					
	End 1.0 sec:							22					
Post-Infln	1st 0.1 sec:							9					
	2nd 0.1 sec:							9					
	1	I	0.68	9	6	5	3	23	0.03	0.66	0.63	33.8	12.5-200.0
		E	0.93	0	2	1	1	4	0.28	0.91	0.63	4.3	3.6- 7.7
	2	I	0.63	6	6	6	0	18	0.02	0.42	0.40	28.6	14.3-200.0
		E	1.03	0	1	2	2	5	0.50	1.02	0.52	4.9	4.4- 50.0
	3	I	0.58	6	7	5	1	19	0.00	0.55	0.55	32.8	8.3-200.0
		E	0.97	1	3	4	6	14	0.14	0.96	0.82	14.4	5.6-200.0
Augmented Breath	1	I	0.95	8	12	8	20	48	0.00	0.92	0.92	50.5	14.3-200.0
		E	1.48	0	1	7	7	15	0.63	2.08	1.45	10.1	6.7- 25.0
Added Dead Space	1	I	0.55	7	6	7	2	22	0.00	0.43	0.43	40.0	20.0-200.0
		E	1.86	1	4	6	9	20	0.41	1.85	1.44	10.8	5.0- 40.0
	2	I	0.57	6	8	5	3	22	0.04	0.52	0.48	38.6	25.0-200.0
		E	1.79	1	5	6	7	19	0.37	1.78	1.41	10.6	5.6-200.0
	3	I	0.57	7	6	4	0	17	0.02	0.37	0.35	29.8	25.0-200.0
		E	1.93	1	5	5	8	19	0.42	1.93	1.51	9.8	5.0-200.0
Pre-Defln	1	I	0.60	5	3	2	0	10	0.01	0.37	0.36	16.7	14.3-200.0
		E	2.06	0	0	0	0	0					
	2	I	0.63	4	4	1	0	9	0.01	0.33	0.32	14.3	14.3- 50.0
		E	1.63	0	0	0	0	0					
	3	I	0.68	6	6	4	1	17	0.00	0.57	0.57	25.0	16.7-100.0
		E	1.36	0	0	2	5	7	0.77	1.34	0.57	5.2	5.3-100.0
Deflation	(Position : 60.3% tI)												
	1st 0.1 sec:							3					

	2nd 0.1 sec:						3						
	1st	I	0.60	6	5	2	1	14	0.00	0.45	0.45	23.3	12.5- 66.7
		E	0.34	0	0	1	0	1	0.25			2.9	
	Mid	I	0.61	4	3	5	2	14	0.02	0.57	0.55	23.0	
		E	0.41	0	0	0	1	1	0.36			2.4	
	End	I	0.60	4	3	4	2	13	0.04	0.51	0.47	21.7	14.3-100.0
		E	0.45	0	0	2	0	2	0.31	0.33	0.02	4.4	
Post-Defln	1st 0.1 sec:						2						
	2nd 0.1 sec:						3						
	1	I	0.54	7	7	4	3	21	0.01	0.48	0.47	38.9	20.0-200.0
		E	1.78	0	0	1	6	7	1.22	1.76	0.54	3.9	7.7- 16.7
	2	I	0.53	6	6	4	1	17	0.01	0.48	0.47	32.1	15.4-100.0
		E	1.96	0	1	5	6	12	0.87	1.90	1.03	6.1	3.5- 16.7
	3	I	0.52	7	6	4	3	20	0.00	0.46	0.46	38.5	22.2-200.0
		E	2.13	0	0	0	2	2	2.10	2.13	0.03	0.9	
Pre-Infln	1	I	0.59	4	3	2	0	9	0.02	0.37	0.35	15.3	14.3- 40.0
(Run 2)		E	2.48	0	0	0	0	0					
	2	I	0.66	5	4	1	0	10	0.02	0.35	0.33	15.2	16.7- 33.3
		E	1.29	0	0	0	0	0					
	3	I	0.69	7	4	5	0	16	0.01	0.50	0.49	23.2	16.7-100.0
		E	1.59	0	0	0	0	0					
Inflation:	(Position: 31.3% tI; Duration of apnoea: 10.65 sec)												
	1st 0.1 sec:						3						
	2nd 0.1 sec:						1						
	1st 1 sec:						0						
	Mid 1 sec:						0						
	End 1 sec:						18						
	1	I	0.69	6	9	5	2	22	0.00	0.58	0.58	31.9	14.3-200.0
		E	0.86	0	0	0	0	0					
	2	I	0.64	3	2	0	0	5	0.06	0.23	0.17	7.8	20.0- 28.6
		E	1.19	0	0	0	0	0					
	3	I	0.60	6	5	4	0	15	0.02	0.45	0.43	25.0	14.3-200.0
		E	1.34	0	0	0	0	0					
Pre-Defln	1	I	0.67	7	5	3	0	15	0.02	0.48	0.46	22.4	16.7-200.0
		E	1.78	0	0	0	0	0					
	2	I	0.69	6	5	2	0	13	0.01	0.42	0.41	18.8	20.0-100.0
		E	2.25	0	0	0	0	0					
Deflation:	(Position: 6.9% tE)												
(Run 2)	1st 0.1 sec:						0						
	1st	I	0.77	5	5	1	0	11	0.01	0.42	0.41	14.3	12.5-100.0
		E	0.34	0	0	0	0	0					
	Mid	I	0.70	4	4	3	1	12	0.05	0.56	0.51	17.1	11.1- 40.0
		E	0.35					1	0.33			2.9	
	End	I	0.71	2	3	4	4	13	0.13	0.67	0.54	18.3	12.5-200.0
		E	0.52	0	0	0	0	0					
Post-Defln	1st 0.1 sec:						2						
	2nd 0.1 sec:						2						
	1	I	0.56	7	4	3	1	15	0.02	0.45	0.43	26.8	16.7-200.0
		E	2.07	0	0	0	0	0					
	2	I	0.57	5	5	3	0	13	0.01	0.40	0.39	22.8	25.0-200.0
		E	2.37	0	0	0	0	0					
	3	I	0.58	5	4	3	0	12	0.02	0.40	0.38	20.7	20.0-200.0
		E	2.17	0	0	0	0	0					

Rabbit Number: 8 Fibre Number: 20 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.Range (Hz)
Pre-Infln	1	I	0.60	5	0	1	0	6	0.07	0.34	0.27	10.0	4.8-100.0
		E	2.25	0	0	0	0	0					
	2	I	0.61	7	2	1	1	11	0.03	0.51	0.48	18.0	6.3-200.0
		E	1.95	0	0	0	0	0					
	3	I	0.63	6	2	3	1	12	0.02	0.57	0.55	19.0	6.3-200.0
		E	2.18	0	0	0	0	0					
Inflation (Run 1)	(Position: 74.1% tI; Duration 18.20 sec.)												
	1st 0.1 sec:							0					
	1st 1.0 sec:							0					
	Mid 1.0 sec:							2					

	End 1.0 sec:						8						
Post-Infln	1st 0.1 sec:						0						
	2nd 0.1 sec:						1						
	1	I	0.61	20	7	6	6	39	0.00	0.60	0.60	63.9	16.7-200.0
		E	0.62	0	0	1	0	1				1.6	
	2	I	0.52	5	3	2	0	10	0.03	0.38	0.35	19.3	9.1-200.0
		E	0.95	0	0	0	0	0					
	3	I	0.55	6	4	3	0	13	0.04	0.39	0.35	23.6	11.1-200.0
		E	1.19	0	0	0	0	0					
Added Dead Space	1	I	0.53	6	9	7	0	22	0.03	0.38	0.35	41.5	18.2-200.0
		E	1.60	0	0	0	0	0					
	2	I	0.52	7	5	11	4	27	0.03	0.50	0.47	51.9	16.7-200.0
		E	1.56	0	0	1	0	1	0.86			0.6	
	3	I	0.52	14	8	4	1	27	0.02	0.48	0.46	51.9	10.0-200.0
		E	1.80	0	0	0	0	0					
Pre-Defln (Run 1)	1	I	0.54	3	3	2	1	9	0.03	0.51	0.48	16.7	6.7-200.0
		E	2.53	0	0	0	0	0					
	2	I	0.56	7	2	1	0	10	0.01	0.29	0.28	17.9	9.1-200.0
		E	2.45	0	0	0	0	0					
	3	I	0.57	3	3	6	2	13	0.03	0.46	0.43	22.8	9.1-100.0
		E	2.12	0	0	0	0	0					
Deflation	(Position: 9.3% tE)												
	1st 0.1 sec:						0						
	1st	I	0.71	2	3	6	1	12	0.03	0.61	0.58	16.9	5.6-200.0
		E	0.49	0	0	0	0	0					
	Mid	I	0.67	2	6	2	2	12	0.08	0.60	0.52	17.9	6.7-200.0
		E	0.68	0	0	0	0	0					
	End	I	0.67	4	3	3	3	13	0.08	0.61	0.53	19.4	11.1-100.0
		E	0.74	0	0	0	0	0					
Post-Defln (Run 1)	1st 0.1 sec:						0						
	1	I	0.52	10	4	1	1	16	0.00	0.41	0.41	30.8	7.7-200.0
		E	1.86	0	0	0	0	0					
	2	I	0.51	6	4	0	1	11	0.03	0.44	0.41	21.6	5.3-200.0
		E	2.00	0	0	0	0	0					
	3	I	0.53	5	2	4	0	11	0.03	0.39	0.36	20.8	9.1- 50.0
		E	2.07	0	0	0	0	0					
Pre-Infln (Run 2)	1	I	0.56	0	0	0	1	1	0.52			1.8	
		E	1.53	0	0	0	0	0					
	2	I	0.59	5	3	4	0	12	0.02	0.37	0.35	20.3	20.0-100.0
		E	1.63	0	0	0	0	0					
Inflation (Run 2)	(Position : 100% tE; Duration of apnoea: 15.95 sec)												
	1st 0.1 sec:						5						
	2nd 0.1 sec:						3						
	1st 1.0 sec:						0						
	Mid 1.0 sec:						4						
	End 1.0 sec:						5						
Augmented Breath (During Inflation)													
	1	I	0.85	8	6	13	9	36	0.01	0.80	0.79	42.4	12.5-200.0
Post-Infln (Run 2)	1st 0.1 sec:						10						
	2nd 0.1 sec:						8						
	1	I	0.43	14	9	6	2	31	0.02	0.43	0.41	72.1	14.3-200.0
		E	0.64	1	1	0	0	2	0.00	0.23	0.23	3.1	
	2	I	0.54	5	1	1	1	8	0.02	0.42	0.40	14.8	4.8-200.0
		E	0.88	0	0	0	0	0					
	3	I	0.55	7	2	2	0	11	0.02	0.34	0.32	20.0	7.7-200.0
		E	1.10	0	0	0	0	0					

Rabbit Number: 8 Fibre Number : 20 PSR: Block

Condition	Br. No.	Ph.	Ph.Dr (sec)	No of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.range (Hz)
Pre-Infln	1	I	1.05	12	17	17	14	60	0.02	1.01	0.99	57.1	22.2-200.0
		E	1.86	1	0	0	0	1	0.28			0.5	
	2	I	1.03	10	18	19	14	61	0.03	1.01	0.98	59.2	25.0-200.0
		E	1.70	0	0	0	2	2	1.31	1.40			
	3	I	1.07	13	19	22	18	72	0.01	1.06	1.05	67.3	14.3-200.0

		E	1.77	0	0	0	0	0					
Inflation	(Position 78.5% tE; Duration of apnoea: 17.4 sec.)												
	1st 0.1 sec:						0						
	2nd 0.1 sec:						0						
	1st 1.0 sec:						1						
	Mid 1.0 sec:						3						
	End 1.0 sec:						3						
Augmented Breath (During Inflation)													
	1	I	1.70	16	22	120	140	298	0.03	1.69	1.66	175.3	25.0-200.0
Post-Infln	1st 0.1 sec:						15						
	2nd 0.1 sec:						11						
	1	I	1.50	18	24	29	19	90	0.04	1.44	1.40	60.0	20.0-200.0
		E	1.50	0	0	0	1	1					
Pre-Defln	1	I	0.94	24	33	35	18	110	0.01	0.89	0.88	117.0	33.3-200.0
		E	1.81	0	1	0	0	1	0.71			0.6	
	2	I	0.95	25	26	36	30	117	0.03	0.91	0.88	123.2	40.0-200.0
		E	1.77	0	2	0	1	3	0.53	1.58	1.05	1.7	1.4- 2.9
	3	I	0.90	30	33	41	22	126	0.00	0.90	0.90	140.0	20.0-200.0
		E	1.54	0	0	1	2	3	1.06	1.44	0.38	1.9	3.0- 16.7
Deflation	(Position: 16.5% tE)												
	1st 0.1 sec:						1						
	1st	I	1.01	54	55	53	37	199	0.00	0.98	0.98	197.0	50.0-200.0
		E	0.86	0	1	0	0	1	0.35			1.2	
	Mid	I	0.89	30	48	47	36	161	0.03	0.88	0.85	180.9	50.0-200.0
		E	1.01	0	0	1	0	1	0.67			1.0	
	End	I	0.86	30	43	51	43	167	0.01	0.85	0.84	194.2	66.7-200.0
		E	1.01	0	0	0	0	1	0.96			1.0	
Post-Defln	1st 0.1 sec:						18						
	2nd 0.1 sec:						16						
	1	I	0.76	30	40	36	35	141	0.02	0.78	0.76	185.5	50.0-200.0
		E	0.91	0	0	0	0	0					
	2	I	0.75	38	37	42	32	149	0.02	0.72	0.70	198.7	66.7-200.0
		E	0.93	1	0	0	0	1	0.13			1.1	
	3	I	0.77	36	42	44	31	153	0.01	0.76	0.75	198.7	100.0-200.0
		E	0.85	1	0	0	0	1	0.01			1.2	
Inflation	(Position: 24.1% tI)												
(Run 2)	1st 0.1 sec:						14						
	1st	I	0.63	26	27	18	16	87	0.00	0.58	0.58	138.1	33.3-200.0
		E	1.80	0	1	0	0	1	0.46			0.6	
	End	I	0.63	19	25	24	23	91	0.00	0.61	0.61	144.4	33.3-200.0
		E	1.50	1	1	0	2	4	0.00	0.98	0.98	2.7	1.6- 66.7
Post-Infln	1st 0.1 sec:						17						
(Run 2)	2nd 0.1 sec:						16						
	1	I	0.86	26	36	42	32	136	0.03	0.84	0.81	158.1	50.0-200.0
		E	1.01	0	0	0	0	0					
	2	I	0.81	35	38	40	29	142	0.00	0.81	0.81	175.3	33.3-200.0
		E	1.15	0	1	2	0	3	0.31	0.67	0.36	2.6	3.3- 16.7
	3	I	0.81	40	40	45	21	146	0.01	0.78	0.77	180.2	50.0-200.0
		E	1.23	0	1	0	0	1	0.53			0.8	
Pre-Defln	1	I	0.88	28	28	31	21	108	0.02	0.86	0.84	122.7	40.0-200.0
		E	1.50	1	0	0	0	1	0.18			0.7	
	2	I	0.87	22	22	32	25	101	0.03	0.85	0.82	116.1	50.0-200.0
		E	1.62	0	0	2	0	2	0.05	0.08	0.03	1.2	
	3	I	0.89	27	29	25	24	105	0.02	0.87	0.85	118.0	33.3-200.0
		E	1.57	0	0	0	0	0					
Deflation	(Position: 7.3% tI)												
(Run 2)	1st 0.1 sec:						16						
	1st	I	1.03	31	46	37	29	143	0.02	1.02	1.00	138.8	33.3-200.0
		E	0.93	1	0	1	0	2	0.22	0.57	0.35	2.2	
	Mid	I	0.86	35	41	39	31	146	0.00	0.84	0.84	169.8	50.0-200.0
		E	0.98	0	0	0	0	0					
	End	I	0.81	37	40	44	40	161	0.10	0.79	0.69	198.8	40.0-200.0
		E	0.94	0	0	0	0	0					
Post-Defln	1st 0.1 sec:						18						
	2nd 0.1 sec:						14						

1	I	0.73	35	38	46	26	145	0.02	0.72	0.70	198.6	66.7-200.0
	E	1.05	1	0	0	0	1	0.03			1.0	
2	I	0.72	38	42	35	31	146	0.01	0.71	0.70	202.8	66.7-200.0
	E	1.12	0	1	1	0	2	0.29	0.82	0.53	1.8	
3	I	0.78	23	21	38	23	105	0.03	0.76	0.73	134.6	40.0-200.0
	E	1.27	0	0	0	0	0					

Rabbit Number: 9 Fibre Number: 21 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.range (Hz)
Augmented Breaths	1	I	0.69	5	8	26	37	76	0.01	0.69	0.68	110.1	16.7-200.0
		E	0.28	8	7	3	2	20	0.00	0.23	0.23	71.4	33.3-200.0
Pre-Infln	1	I	0.73	7	5	2	3	17	0.00	0.67	0.67	7.1	0.7- 7.1
		E	0.91	0	0	0	0	0					
	2	I	0.65	6	4	5	1	16	0.00	0.53	0.53	16.7	0.5- 24.6
		E	1.11	0	1	0	1	2	0.38	1.09	0.71	1.8	
	3	I	0.68	5	4	7	6	22	0.01	0.65	0.64	32.4	12.5-200.0
		E	1.07	0	1	1	1	3	0.46	1.05	0.59	2.8	2.6- 4.5

Inflation (Position: 30.4% tI; Duration of apnoea: 2.36 sec.)

1st 0.1 sec:	4
2nd 0.1 sec:	1
1st 1.0 sec:	0
End 1.0 sec:	8

Augmented Breath (During lung inflation)

1	I	0.84	8	7	7	7	29	0.00	0.79	0.79	34.5	14.3-200.0
	E	2.20	5	4	5	5	19	0.03	1.93	1.90	8.6	3.0- 25.0

Post-Infln	1st 0.1 sec:						1					
	2nd 0.1 sec:						0					
1	I	0.77	5	5	4	3	17	0.00	0.67	0.67	12.5	0.7- 22.1
	E	0.47	1	0	1	1	3	0.01	0.37	0.36	5.0	0.4- 6.4
2	I	0.66	7	5	9	4	25	0.02	0.55	0.53	25.0	0.5- 37.9
	E	0.68	0	2	0	0	2	0.23	0.32	0.09	2.9	
3	I	0.62	6	6	6	2	20	0.01	0.52	0.51	32.3	16.7-200.0
	E	0.82	0	1	0	0	1	0.22			1.2	
Pre-Infln	1	I	0.80	2	1	4	1	8	0.06	0.73	0.67	10.0	5.3- 50.0
(Run 2)		E	1.35	0	0	0	0	0					
	2	I	0.77	2	3	1	2	8	0.02	0.59	0.57	10.4	4.3- 40.0
		E	1.32	0	0	0	0	0					

Inflation (Position 100% tI; Duration of apnoea: 2.25 sec.)

(Run 2) 1st 0.1 sec:	1
2nd 0.1 sec:	0
1st 1.0 sec:	10
End 1.0 sec:	25

Post-Infln	1st 0.1 sec:						7					
(Run 2)	2nd 0.1 sec:						5					
1	I	0.89	10	6	6	8	30	0.03	0.86	0.83	33.7	15.4-200.0
	E	0.58	0	0	0	0	0					
2	I	0.73	5	2	5	4	16	0.04	0.71	0.67	21.9	9.1-100.0
	E	0.68	1	0	0	0	1	0.06			1.5	
3	I	0.72	5	2	6	4	17	0.00	0.67	0.67	23.6	10.0-200.0
	E	0.71	0	0	0	0	0					

Rabbit Number: 9 Fibre Number:22 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.range (Hz)
Pre-Infln	1	I	0.69	3	3	3	2	11	0.01	0.60	0.59	15.9	12.5- 40.0
		E	1.58	0	0	0	0	0					
	2	I	0.71	1	0	2	3	6	0.02	0.63	0.61	8.5	3.0- 20.0
		E	1.15	0	2	0	1	3	0.30	0.91	0.61	2.6	2.2- 6.7
	3	I	0.71	0	2	2	1	5	0.28	0.53	0.25	7.0	10.0- 40.0
		E	1.45	0	0	0	0	0					

Inflation (Position: 28.6% tI; Duration of apnoea: 3.46 sec.)

1st 0.1 sec:	4
2nd 0.1 sec:	5

	1st 1.0 sec:					29					
	Mid 1.0 sec:					58					
	End 1.0 sec:					69					
Post-Infln	1st 0.1 sec:					8					
	2nd 0.1 sec:					6					
	1	I	0.64	7	6	6	6	25	0.01	0.61	0.60	39.1	33.3-100.0
		E	0.62	8	8	6	7	29	0.00	0.61	0.61	46.8	40.0-200.0
	2	I	0.61	7	6	6	5	24	0.02	0.59	0.57	39.3	33.3-200.0
		E	0.87	9	9	11	8	37	0.01	0.85	0.84	42.5	33.3-200.0
	3	I	0.65	7	7	8	5	27	0.01	0.63	0.62	41.4	33.3-200.0
		E	0.69	6	7	7	6	26	0.01	0.67	0.66	37.7	20.0- 50.0
Pre-Defln	1	I	0.65	3	3	2	1	6	0.06	0.60	0.54	9.2	6.3- 50.0
		E	1.55	0	0	3	0	3	1.07	1.08	0.01	1.9	100.0-200.0
	2	I	0.62	5	1	4	1	11	0.02	0.51	0.49	17.7	6.7-200.0
		E	1.37	0	0	1	1	2	1.36			0.7	
	3	I	0.66	3	5	2	4	14	0.00	0.65	0.65	21.2	8.3-200.0
		E	1.43	0	0	0	0	0					
Deflation	(Position: 15.9% tE)												
	1st 0.1 sec:					16					
Augmented Breath (immediately following deflation)													
	1	I	0.95	25	38	15	6	84	0.01	0.89	0.88	88.4	13.3-200.0
		E	0.67	5	0	1	1	7	0.04	0.65	0.61	10.4	3.7- 66.7
	1st	I	0.70	4	2	3	3	12	0.01	0.63	0.62	17.1	12.5- 50.0
		E	1.11	0	1	0	0	1	0.55			0.9	
	Mid	I	0.61	5	6	2	3	16	0.00	0.55	0.55	26.2	8.3-100.0
		E	1.11	0	0	0	0	0					
	End	I	0.65	5	5	3	2	15	0.01	0.59	0.58	23.1	12.5-200.0
		E	1.16	0	0	0	0	0					
Post-Defln	1st 0.1 sec:					1					
	2nd).1 sec:					2					
	1	I	0.54	4	3	3	1	11	0.00	0.41	0.41	20.4	11.1-100.0
		E	1.56	0	1	0	0	1	0.73			0.6	
	2	I	0.58	2	2	3	1	8	0.03	0.51	0.48	13.8	10.5- 50.0
		E	1.58	0	0	0	0	0					
	3	I	0.63	3	3	4	1	11	0.04	0.50	0.46	17.5	16.7- 50.0
Pre-Infln (Run 2)	1	I	0.78	2	1	1	1	5	0.05	0.66	0.61	6.4	3.2- 14.3
		E	1.52	0	0	0	0	0					
	2	I	0.79	2	1	2	1	6	0.01	0.63	0.62	7.6	5.6- 20.0
		E	1.65	0	0	0	0	0					
	3	I	0.75	3	1	2	0	6	0.06	0.55	0.49	8.0	5.0- 40.0
		E	1.59	0	0	1	1	2	0.91	1.40	0.49	1.3	
Inflation (Run 2)	(Position 26.0% tI, Duration of apnoea: 5.75 sec.)												
	1st 0.1 sec:					1					
	2nd 0.1 sec:					3					
	1st 1.0 sec:					3					
	Mid 1.0 sec:					1					
	End 1.0 sec:					3					
Post-Infln (Run 2)	1st 0.1 sec:					0					
	1	I	0.85	2	1	2	2	7	0.08	0.67	0.59	8.2	4.3- 50.0
		E	0.67	0	0	0	0	0					
	2	I	0.72	2	1	1	1	5	0.11	0.61	0.50	16.9	4.2- 20.0
		E	0.96	0	1	1	0	2	0.45	0.53	0.08	2.1	
	3	I	0.71	1	0	4	0	5	0.01	0.53	0.52	7.0	2.6- 40.0
		E	1.19	0	0	0	1	1	1.11			0.8	
Pre-Defln (Run 2)	1	I	0.77	1	1	3	1	6	0.00	0.73	0.73	7.8	2.8- 40.0
		E	1.47	0	0	1	0	1	1.02			0.7	
	2	I	0.77	0	1	1	1	3	0.37	0.72	0.35	3.9	5.6- 6.3
		E	1.45	0	0	0	0	0					
	3	I	0.75	1	1	2	0	4	0.01	0.50	0.49	5.3	3.8- 33.3
		E	1.58	0	0	0	0	0					
Deflation (Run 2)	(Position: 2% tE)												
	1st 0.1 sec:					0					
	1st	I	0.91	6	4	4	4	18	0.01	0.86	0.85	19.8	9.1-100.0
		E	0.32	0	0	0	1	1	0.29			3.1	
	Mid	I	0.68	3	3	1	1	8	0.02	0.61	0.59	11.8	6.3- 50.0
		E	0.39	0	0	0	0	0					
	End	I	0.73	1	0	4	1	6	0.45	0.66	0.21	8.2	2.3-100.0

		E	0.40	0	0	0	0	0					
Post Defln	1st 0.1 sec:							0					
	2nd 0.1 sec:							1					
	1	I	0.70	2	2	5	3	12	0.00	0.62	0.62	17.1	6.7-200.0
		E	1.39	0	0	1	0	1	0.94			0.7	
	2	I	0.70	3	2	3	1	9	0.06	0.58	0.52	12.9	10.0-100.0
		E	1.55	0	0	0	0	0					
	3	I	0.69	2	3	4	0	9	0.01	0.42	0.41	13.0	7.7-100.0
		E	1.62	0	1	0	0	1	0.72			0.6	

Rabbit Number: 9 Fibre Number: 23 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.Range (Hz)
Pre-Infln	1	I	0.82	0	3	3	3	9	0.25	0.81	0.56	11.0	5.0-200.0
		E	1.78	0	0	0	0	0					
	2	I	0.85	3	0	1	0	4	0.01	0.56	0.55	4.7	2.0-100.0
		E	1.70	0	0	0	0	0					
	3	I	0.82	0	3	0	0	3	0.23	0.39	0.16	3.7	12.5- 14.3
		E	1.70	0	1	0	0	1	0.75			0.6	
Inflation	(Position: 36.1% tI; Duration of apnoea: 4.18 sec.)												
	1st 0.1 sec:							1					
	2nd 0.1 sec:							0					
	1st 1.0 sec:							3					
	Mid 1.0 sec:							10					
	End 1.0 sec:							14					
Post-Infln	1st 0.1 sec:							1					
	2nd 0.1 sec:							1					
	1	I	0.90	7	6	4	4	21	0.01	0.82	0.81	23.3	11.1-200.0
		E	0.76	0	0	0	0	0					
	2	I	0.73	5	2	1	1	9	0.01	0.61	0.60	12.3	5.6-200.0
		E	1.06	0	1	0	0	1				0.9	
	3	I	0.70	4	2	3	1	10	0.01	0.62	0.61	14.3	5.6-200.0
		E	1.22	0	0	0	0	0					
Pre-Defln	1	I	0.85	4	4	4	3	15	0.00	0.76	0.76	17.6	10.0-100.0
		E	1.19	1	3	3	1	8	0.22	1.09	0.87	6.7	4.0- 20.0
	2	I	0.77	3	2	3	1	9	0.01	0.71	0.70	11.7	5.0- 50.0
		E	1.59	0	0	0	1	1	1.58			0.6	
	3	I	0.78	2	4	1	3	10	0.05	0.70	0.65	12.8	5.6-100.0
		E	1.58	2	2	2	1	7	0.32	1.56	1.24	4.4	1.6- 13.0
Deflation	(Position: 32.5% tE)												
	1st 0.1 sec:							0					
	1st	I	0.96	9	4	10	7	30	0.00	0.87	0.87	31.3	5.9-200.0
		E	0.31	0	0	0	1	1	0.27			3.2	
	Mid	I	0.69	8	12	7	3	30	0.03	0.64	0.61	43.5	14.3-200.0
		E	0.38	0	0	0	2	2	0.31	0.36	0.05	5.3	
	End	I	0.72	6	6	9	3	24	0.01	0.64	0.63	33.3	11.1-200.0
		E	0.50	1	0	0	0	1	0.12			2.0	
Post-Defln	1st 0.1 sec:							3					
	2nd 0.1 sec:							0					
	1	I	0.78	9	7	7	9	32	0.01	0.71	0.70	41.0	11.1-200.0
		E	1.47	2	8	3	3	16	0.21	1.37	1.16	10.9	3.6-200.0
	2	I	0.68	8	6	5	1	20	0.00	0.62	0.62	29.4	9.1-200.0
		E	1.60	5	5	2	4	16	0.27	1.59	1.32	10.0	3.1-100.0
	3	I	0.78	6	3	5	5	19	0.02	0.74	0.72	24.4	10.0- 66.7
		E	1.54	3	4	2	2	11	0.12	1.38	1.26	7.1	2.7- 50.0

Rabbit Number: 9 Fibre Number: 23 PSR: Block

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.range (Hz)
Pre-Infln	1	I	1.46	4	7	12	14	37	0.02	1.44	1.42	25.3	6.3-200.0
		E	1.62	0	1	0	0	1	0.52			0.6	
	2	I	1.55	6	9	13	15	43	0.11	1.47	0.03	27.7	9.1-200.0
		E	1.50	0	0	0	0	0					
	3	I	1.46	4	10	14	10	38	0.02	1.41	1.39	26.0	4.8-200.0
		E	1.76	0	0	0	0	0					

Inflation	(Position: 22.0% tI)												
	1st 0.1 sec:						6						
	2nd 0.1 sec:						3						
	1st	I	1.08	5	11	13	7	36	0.00	1.04	1.04	33.3	5.0-200.0
		E	1.87	0	1	0	0	1	0.70			1.1	
	Mid	I	1.08	10	7	15	9	41	0.03	0.98	0.96	38.0	20.0-200.0
		E	1.62	0	1	0	3	4	0.44	1.60	1.16	2.5	1.1- 20.0
	End	I	1.11	5	11	16	11	43	0.07	1.00	0.93	38.7	11.1-200.0
		E	1.66	0	3	2	2	7	0.53	1.64	1.11	4.2	2.9- 12.5
Post-Infln	1st 0.1 sec:						0						
	2nd 0.1 sec:						0						
	1	I	1.45	18	26	23	27	94	0.01	1.41	1.40	64.8	16.7-200.0
		E	1.33	5	1	0	1	7	0.13	1.32	1.19	5.3	1.4- 40.0
	2	I	1.33	21	25	27	23	96	0.00	1.32	1.32	72.2	20.0-200.0
		E	1.35	0	5	3	1	9	0.34	1.33	0.99	6.7	2.6-100.0
	3	I	1.29	17	14	20	14	65	0.01	1.24	1.23	50.4	14.3-200.0
		E	1.34	2	0	0	1	3	0.24	1.32	1.08	2.2	1.0- 33.3
Pre-Defln	1	I	1.41	6	10	13	12	41	0.00	1.35	1.35	29.1	6.7-200.0
		E	1.74	0	0	0	2	2	1.26	1.73	0.47	1.2	
	2	I	1.37	5	11	15	12	43	0.01	1.27	1.26	31.4	7.1-200.0
		E	1.81	0	0	0	1	1	1.78			0.6	
	3	I	1.44	12	11	22	17	62	0.00	1.35	1.35	43.1	8.3-200.0
		E	1.76	2	1	0	0	3	0.21	0.55	0.34	1.7	3.6- 20.0
Deflation	(Position : 9.1% tE)												
	1st 0.1 sec:						0						
	2nd 0.1 sec:						2						
	1st	I	1.60	26	24	30	29	109	0.01	1.62	1.61	68.1	16.7-200.0
		E	1.49	4	1	0	3	8	0.23	1.48	1.25	5.4	1.3-200.0
	Mid	I	1.54	25	40	50	35	150	0.00	1.53	1.53	97.4	20.0-200.0
		E	1.56	9	8	13	23	53	0.19	1.55	1.36	34.0	9.1-200.0
	End	I	1.63	42	40	41	57	180	0.01	1.62	1.61	110.4	33.3-200.0
		E	1.15	6	10	6	9	31	0.09	1.14	1.05	27.0	5.9-200.0
Post-Defln	1st 0.1 sec:						13						
	2nd 0.1 sec:						13						
	1	I	1.24	30	33	31	32	126	0.01	1.22	1.21	101.6	28.6-200.0
		E	1.20	4	11	3	9	27	0.11	1.19	1.08	22.5	6.7-200.0
	2	I	1.15	28	21	31	19	99	0.00	1.14	1.14	86.1	25.0-200.0
		E	1.29	4	4	3	8	19	0.22	1.28	1.06	14.7	4.2-200.0
	3	I	1.18	23	22	29	29	103	0.01	1.17	1.16	87.3	33.3-200.0
		E	1.28	3	3	5	5	16	0.25	1.26	1.01	12.5	6.3-400.0
Pre-Infln	1	I	1.37	21	19	32	33	105	0.00	1.35	1.35	76.6	14.3-200.0
		E	1.30	4	1	1	2	8	0.18	1.29	1.11	6.2	5.3- 40.0
	2	I	1.23	25	24	22	25	96	0.01	1.19	1.18	78.1	16.7-200.0
		E	1.39	0	2	2	5	9	0.38	1.32	0.94	6.5	2.0- 20.0
	3	I	1.33	21	35	30	36	122	0.01	1.30	1.29	91.7	20.0-200.0
		E	1.57	2	0	3	5	10	0.09	1.56	1.47	6.4	1.9- 20.0
Inflation	(Position: 23.7% tI)												
(Run 2)	1st 0.1 sec:						6						
	2nd 0.1 sec:						10						
	1st	I	0.95	22	22	84	18	86	0.00	0.90	0.90	90.5	20.0-200.0
		E	1.82	2	3	3	7	15	0.31	1.81	1.50	8.2	2.7-200.0
	Mid	I	0.94	23	19	24	15	81	0.01	0.89	0.88	86.2	25.0-200.0
		E	1.70	3	6	2	8	19	0.17	1.60	1.43	11.2	3.9-200.0
	End	I	0.95	20	19	20	15	74	0.00	0.89	0.89	77.9	33.3-200.0
		E	1.51	8	4	4	7	23	0.09	1.49	1.40	15.2	5.0-200.0
Post-Infln	1st 0.1 sec:						2						
	2nd 0.1 sec:						5						
	1	I	1.39	36	33	43	37	149	0.00	1.37	1.37	107.2	33.3-200.0
		E	1.44	14	8	8	9	39	0.00	1.43	1.43	27.1	6.3-200.0
	2	I	1.25	28	28	32	25	113	0.00	1.22	1.22	90.4	33.3-200.0
		E	1.34	9	12	5	4	30	0.16	1.32	1.16	22.4	6.3-200.0
	3	I	1.19	35	27	30	16	108	0.00	1.15	1.15	90.8	20.0-200.0
		E	1.46	5	3	0	3	11	0.12	1.44	1.32	9.5	1.3-200.0
Pre-Defln	1	I	1.25	6	13	8	16	43	0.03	1.20	1.17	34.4	9.1-200.0
		E	1.62	0	1	0	0	1	0.57			0.6	
	2	I	1.29	5	14	15	21	55	0.01	1.26	1.25	42.6	4.4-200.0
		E	1.64	0	1	0	0	1	0.81			0.6	
	3	I	1.38	9	11	15	13	48	0.02	1.32	1.30	34.8	11.1-200.0
		E	1.71	0	0	0	1	1	1.69			0.6	

Deflation: (Position: 8.9% tE)														
	1st 0.1 sec:						0							
	2nd 0.1 sec:						0							
	1st	I	1.73	22	28	38	23	111	0.05	1.68	1.63	4.2	20.0-200.0
		E	1.53	0	0	2	5	7	0.90	1.52	0.62	4.6	4.8- 50.0
	Mid	I	1.55	32	40	46	34	152	0.00	1.49	1.49	98.1	25.0-200.0
		E	1.35	5	5	3	7	20	0.13	1.33	1.20	14.8	4.8-200.0
	End	I	1.41	32	30	44	31	137	0.00	1.40	1.40	97.2	25.0-200.0
		E	1.38	6	9	0	11	26	0.14	1.37	1.23	18.8	2.8-200.0
Post-Defln	1st	0.1 sec:						10						
	2nd	0.1 sec:						13						
	1	I	1.33	33	32	42	33	140	0.00	1.27	1.27	105.3	40.0-200.0
		E	1.29	6	8	6	8	28	0.20	1.28	1.08	21.7	9.1-100.0
	2	I	1.13	23	28	32	21	104	0.01	1.10	1.09	92.0	20.0-200.0
		E	1.33	3	4	4	4	15	0.15	1.29	1.14	11.3	50.0-200.0
	3	I	1.24	23	26	24	20	93	0.00	1.17	1.17	75.0	20.0-200.0
		E	1.33	2	2	1	4	9	0.21	1.32	1.11	6.8	3.0-200.0
Added Dead Space	1	I	1.06	19	31	27	20	97	0.00	1.03	1.03	91.5	25.0-200.0
		E	1.41	8	4	5	7	24	0.00	1.40	1.40	17.0	6.7-200.0
	2	I	1.14	18	22	30	26	96	0.01	1.10	1.09	84.2	25.0-200.0
		E	1.39	12	12	8	16	48	0.14	1.38	1.24	34.5	6.7-200.0
	3	I	1.56	35	41	42	35	153	0.00	1.55	1.55	98.1	25.0-200.0
		E	1.05	45	34	14	15	108	0.00	1.03	1.03	102.9	20.0-200.0

Rabbit Number: 9 Fibre Number: 24 PSR: Intact

Condition	Br. No.	Ph.	Ph.Dr (sec)	No of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.range (Hz)
Pre-Infln	1	I	1.04	2	2	2	2	8	0.01	0.92	0.91	7.7	3.2- 50.0
		E	1.72	0	0	0	1	1	1.44			0.6	
	2	I	0.99	3	1	3	4	11	0.00	0.94	0.94	11.1	2.6-200.0
		E	1.75	0	0	0	0	0					
	3	I	1.07	2	1	9	1	13	0.02	1.04	1.02	12.1	3.6-100.0
		E	1.68	0	4	0	0	4	0.42	0.71	0.29	2.4	5.0- 25.0
Inflation	(Position: 32.0% tI; Duration of apnoea: 6.37 sec.)												
	1st 0.1 sec:						1						
	2nd 0.1 sec:						2						
	1st 1.0 sec:						6						
	Mid 1.0 sec:						3						
	End 1.0 sec:						5						
Post-Infln	1st 0.1 sec:						4						
	2nd 0,1 sec:						0						
	1	I	1.02	3	4	4	7	18	0.11	0.97	0.86	17.6	6.7-100.0
		E	1.22	0	3	0	0	3	0.34	0.60	0.26	2.5	
	2	I	0.98	7	7	8	7	29	0.01	0.93	0.92	29.6	9.1-200.0
		E	1.27	0	5	1	2	8	0.41	1.24	0.83	6.3	2.2-100.0
	3	I	0.96	8	3	6	7	24	0.00	0.90	0.90	25.0	8.3-200.0
		E	1.33	2	2	1	0	5	0.25	0.68	0.43	3.8	6.7- 20.0
Pre-Defln	1	I	1.02	2	3	3	6	14	0.07	0.96	0.89	13.7	5.3-200.0
		E	1.77	0	1	0	1	1	0.72	1.60	0.98	1.1	
	2	I	1.07	0	2	7	4	13	0.45	0.91	0.46	12.1	7.7-200.0
		E	1.74	2	0	1	0	3	0.21	0.90	0.69	1.7	
	3	I	1.01	2	1	5	6	14	0.01	0.99	0.98	13.9	2.9-200.0
		E	1.70	0	2	0	0	2	0.75	0.83	0.08	1.2	
Deflation	(Position: 10.4% tE)												
	1st 0.1 sec:						2						
	2nd 0.1 sec:						1						
	1st	I	1.22	6	9	15	12	42	0.04	1.19	1.15	34.4	6.3-200.0
		E	0.80	0	3	0	0	3	0.25	0.38	0.13	3.7	12.5- 20.0
	Mid	I	1.15	5	8	13	5	31	0.01	1.14	1.13	27.0	5.3-200.0
		E	0.87	0	1	0	0	1				1.2	
	End	I	0.99	5	9	12	13	39	0.02	0.97	0.95	39.4	6.3-200.0
		E	1.25	4	1	0	2	7	0.00	1.24	1.24	5.6	1.2-100.0
Post-Defln	1st 0.1 sec:						8						
	2nd 0.1 sec:						9						
	1	I	0.95	17	11	14	14	56	0.00	0.95	0.95	58.9	16.7-200.0

		E	1.26	0	0	2	3	5	0.68	1.24	0.56	4.0	3.2- 40.0
	2	I	0.94	6	7	11	16	40	0.01	0.92	0.91	42.6	12.5-200.0
		E	1.44	0	0	0	0	1	1.06			0.7	
	3	I	0.94	8	9	11	12	40	0.02	0.92	0.90	42.6	14.3-200.0
		E	1.47	1	0	0	0	1	0.31			0.7	

Inflation (Position: 20.2% tI; Duration of apnoea: 9.80 sec)

1st 0.1 sec:	4
2nd 0.1 sec:	1
1st 1.0 sec:	6
Mid 1.0 sec:	13
End 1.0 sec:	28

Post-Infln	1st 0.1 sec:							9					
(Run 2)	2nd 0.1 sec:							3					
	1	I	0.97	7	9	17	10	43	0.02	0.95	0.93	44.3	11.1-200.0
		E	1.02	6	5	1	2	14	0.06	1.00	0.94	13.7	4.4-100.0
	2	I	0.93	13	10	17	13	53	0.01	0.84	0.83	57.0	16.7-200.0
		E	1.11	2	6	2	1	11	0.11	0.94	0.83	9.9	3.2-200.0
	3	I	0.89	15	9	15	12	51	0.02	0.85	0.83	57.3	20.0-200.0
		E	1.18	2	1	1	0	4	0.18	0.62	0.44	3.4	3.2- 20.0
Added Dead	1	I	0.80	16	7	16	12	51	0.00	0.78	0.78	63.8	20.0-200.0
Space		E	1.24	1	4	3	3	11	0.27	1.23	0.96	8.9	11.5- 50.0
	2	I	0.90	11	13	11	11	46	0.01	0.85	0.84	51.1	14.3-200.0
		E	1.15	4	6	5	3	18	0.13	1.09	0.26	15.7	7.1-100.0
	3	I	0.96	15	10	21	13	67	0.00	0.93	0.93	69.8	20.0-200.0
		E	0.98	7	8	6	2	23	0.01	0.97	0.96	23.5	5.0-200.0

Rabbit Number: 9 Fibre Number: 24 PSR: Block

Condition	Br. No.	Ph.	Ph.Dr (sec)	No. of Spikes Q1	Q2	Q3	Q4	Tot.	Bg.F (sec)	End.F (sec)	Dr.F (sec)	Fmean (Hz)	Freq.range (Hz)
Pre-Infln	1	I	1.35	3	6	8	6	23	0.03	1.31	1.28	17.0	5.3-200.0
(Run 1)		E	2.06	1	1	0	0	2	0.01	0.80	0.79	1.0	
	2	I	1.40	4	6	8	11	29	0.01	1.35	1.34	20.7	3.3-200.0
		E	2.17	2	0	0	1	3	0.25	2.11	1.86	1.4	0.6- 12.5
	3	I	1.40	5	6	7	9	27	0.02	1.38	1.36	19.3	5.9-200.0
		E	2.25	0	1	0	0	1	0.80				

Inflation (Position: 18.8% tI)

1st 0.1 sec:	6
2nd 0.1 sec:	2

	1	I	1.08	7	7	10	7	31	0.02	0.97	0.95	28.7	7.7-100.0
		E	2.35	3	1	1	5	10	0.07	2.08	2.01	4.3	1.7-100.0
Post-Infln	1st 0.1 sec:							0					
	2nd 0.1 sec:							0					
	1	I	1.38	2	9	9	6	26	0.06	1.29	1.23	18.8	3.5-200.0
		E	1.62	2	1	0	0	3	0.33	0.48	0.15	1.9	8.3- 33.3
	2	I	1.26	5	9	8	11	33	0.02	1.21	1.19	26.2	5.6-200.0
		E	1.56	5	3	1	1	10	0.01	1.53	1.52	6.4	1.6-200.0
	3	I	1.24	7	6	9	8	30	0.02	1.22	1.20	24.2	5.3-200.0
		E	1.76	1	0	1	0	2	0.30	1.11	0.81	1.1	
Inflation	1	I	1.08	6	8	12	7	33	0.04	1.04	1.00	30.6	12.5-100.0
(Run 2)		E	2.00	4	3	2	0	9	1.13	1.38	0.25	4.5	3.6- 12.5
Post-Infln	1st 0.1 sec:							0					
(Run 2)	2nd 0.1 sec:							0					
	1	I	1.35	2	10	5	12	29	0.26	1.27	1.01	21.5	7.1-200.0
		E	1.81	1	0	0	1	2	0.23	1.76	0.53	1.1	
	2	I	1.27	6	4	10	6	26	0.00	1.16	1.16	20.5	6.7-200.0
		E	1.86	0	3	1	0	4	0.55	0.94	0.39	2.2	3.2- 40.0
	3	I	1.23	3	3	8	10	24	0.08	1.17	1.09	19.5	6.3-200.0
		E	1.96	0	1	0	1	2	0.51	1.95	1.44	1.0	
Pre-Defln	1	I	1.39	2	1	9	16	28	0.03	1.38	1.35	20.1	2.2-200.0
		E	1.97	0	0	0	0	0					
	2	I	1.25	4	8	11	3	26	0.02	1.19	1.17	20.8	3.7-200.0
		E	1.50	0	0	0	0	0					
	3	I	1.24	3	1	7	5	16	0.00	1.15	1.15	12.9	2.9-200.0
		E	1.78	1	0	9	9	1	0.29			0.6	

Deflation: (Position: 10.1% tE)													
	1st	I	1.35	9	15	22	22	68	0.00	1.31	1.31	50.4	8.3-200.0
		E	1.38	8	5	4	2	19	0.00	1.34	1.34	13.8	5.6-200.0
	Mid	I	1.40	15	14	18	18	65	0.01	1.39	1.38	46.4	10.0-200.0
		E	1.58	2	2	3	4	11	0.31	1.57	1.26	7.0	2.0-200.0
	End	I	1.35	9	16	14	13	52	0.00	1.31	1.31	38.5	6.3-200.0
		E	1.14	1	4	0	2	7	0.18	1.12	0.94	6.1	1.8-100.0
Post-Defln	1st 0.1 sec:							1					
	2nd 0.1 sec:							0					
	1	I	1.33	9	11	19	15	54	0.01	1.30	1.29	40.6	11.1-200.0
		E	1.30	11	23	5	4	43	0.01	1.27	1.26	33.1	4.8-200.0
	2	I	1.57	10	11	17	16	54	0.02	1.55	1.53	34.4	10.0-200.0
		E	1.16	1	1	0	1	3	0.14	1.00	0.86	2.6	1.4- 6.7
	3	I	1.16	7	8	6	13	34	0.02	1.15	1.13	29.3	6.7-200.0
		E	1.28	0	0	0	0	0					
Added Dead Space	1	I	1.24	5	3	9	12	29	0.01	1.21	1.20	23.4	4.8-200.0
		E	1.63	2	2	2	0	6	0.15	1.18	1.03	3.7	4.2- 5.6
	2	I	1.24	5	5	14	8	32	0.01	1.22	1.21	25.8	7.7-200.0
		E	1.46	1	4	3	4	12	0.31	1.44	1.13	8.2	4.4- 40.0
	3	I	1.21	7	11	8	8	34	0.00	1.19	1.19	28.1	7.7-200.0
		E	1.42	3	0	1	1	5	0.05	1.26	1.21	3.5	1.5- 20.0
Deflation: (Position: 10.4% tE)													
	1st 0.1 sec:							1					
	1	I	1.56	5	11	19	15	50	0.11	1.53	1.42	32.1	10.0-200.0
		E	1.77	4	1	1	0	6	0.06	1.02	0.96	3.4	2.0- 20.0
	2	I	1.52	11	16	14	13	54	0.01	1.51	1.50	35.5	10.0-200.0
		E	1.85	1	2	2	5	10	0.43	1.85	1.42	5.4	3.1-200.0
	3	I	1.57	10	19	17	19	65	0.06	1.55	1.49	41.4	6.7-200.0
		E	1.45	2	7	3	4	16	0.01	1.44	1.43	11.0	2.8-200.0
Post-Defln	1st 0.1 sec:							6					
	2nd 0.1 sec:							4					
	1	I	1.39	8	18	15	13	54	0.05	1.37	1.32	38.8	14.3-200.0
		E	1.20	3	6	3	0	12	0.15	0.84	0.69	10.0	7.7-100.0
	2	I	1.20	15	10	7	10	42	0.00	1.19	1.19	35.0	11.1-200.0
		E	1.32	2	1	1	0	4	0.21	0.76	0.55	3.0	4.4- 10.0
	3	I	1.24	8	7	7	9	31	0.06	1.23	1.17	25.0	6.7-200.0
		E	1.42	0	1	1	1	3	0.45	1.34	0.89	2.1	1.8- 2.9